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Understanding and Managing Dementia-Related Sleep Problems:

Community-Based Research with Older New Zealanders

A thesis presented in partial fulfilment of the requirements for the degree of Doctor of Philosophy

at Massey University, Sleep/Wake Research Centre
Wellington, New Zealand

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2014

Abstract

Sleep changes with ageing and people with dementia and their carers often have disturbed sleep, but information on the sleep of older New Zealanders is lacking. Four studies were conducted in order to address these factors. The first two used pre-existing survey data to understand the sleep health of older people, and to explore the relationship between caregiving and sleep. Sleep problems were reported by 20-32% of participants, prevalence decreased with increasing age. In those aged at least 79 years, sleep problems were associated more with health status rather than demographic factors. Older carers were more likely to report feeling tired than non-carers.

Dementia-related sleep problems are challenging for individuals and their carers, and poor sleep may exacerbate waking dementia symptoms. However, there is limited research with community-dwelling dyads of people with dementia (PWD) and their carers. Studies 3 and 4 were conducted to understand and treat dementia-related sleep problems. Focus groups with 12 dyads revealed the multifaceted nature of their sleep problems. Normalisation of sleep problems was common. In the final study, a five-week trial was piloted involving sleep education, light therapy and an exercise programme. Sleep of the dyads was monitored using actigraphy and standardised questionnaires. Questionnaires also measured cognitive functioning, quality of life, and dementia-related disruption, as well as carers' mental health and coping. Fifteen pairs participated, of whom nine completed the trial. Case studies revealed that five PWD had improvements to their subjective sleep ratings. These PWD also showed some improvements in wake time at night, cognitive functioning, and carer-rated quality of life. These changes did not always translate into improved sleep or mental health for carers. Many PWD's health deteriorated across the trial, masking the effects of the intervention.

Overall, these studies illustrate the importance and diverse nature of sleep with ageing, dementia, and caregiving. Non-pharmacological interventions can be used successfully by some community-dwelling dyads. It is recommended that these low-risk interventions are considered by healthcare professionals. Increased knowledge and options could empower individuals to manage their own symptoms, providing hope for improving the sleeping and waking experience of older people affected by dementia.

Dedication

During my time as an undergraduate student I worked as a care assistant for older people, the majority of whom had dementia. What I observed with regards to sleep and wake behaviours fascinated me. Some nursing home residents would behave in a polite, sociable manner whilst eating their breakfast; but by afternoon tea time would be paranoid, pacing, and requiring physical assistance. There were those who would be up in the night, walking the halls, seeking something out, or prematurely preparing for the day. In the nursing home environment there was a band of us care assistants and nurses who could take shifts in supporting our residents, guiding them back to bed at night, and taking the brunt of their sleep deprivation the following day.

Many people who have these symptoms live at home and are cared for by a family member, sometimes single-handedly. How these families managed with such disruptions to their sleep and wake intrigued me. For several months I worked as an assistant for Dorothy and Doreen, sisters aged over 80 years, one of whom had Alzheimers Disease. My job was to support them at the end of their day. To help Dorothy safely have her final cup of tea and cigarette, prepare for bed, read to her, listen to her, guide her back upstairs as many times as it would take, and to accompany Doreen while she waited for the sound of silence permitting her to go to bed. The tenacity of these sisters to remain together at home was overwhelming. They managed until Dorothy's dementia was such that she required overnight assistance to prevent accidents, and to allow Doreen sufficient rest. Ultimately both women had falls, leading to hospitalisation and institutionalised care. This experience stayed with me.

After graduating, I diverged into the fascinating world of sleep science, in a clinical and academic sense. Almost ten years on, it is a privilege to have the opportunity to present my research concerning the sleep of older people and people with dementia. I dedicate this thesis to all families affected by dementia, especially Dorothy and Doreen for inspiring me to take this journey.

Nights and Days[1]

Aching limbs, tired eyes,

Silently weeping, pitiful cries,

All these things happen at night,

Never in daylight and out of sight,

How I wish my night was day,

Sunshine chasing my nightmares away,

Until then I continue to fight,

All these demons every night,

Just one thing I want to say,

For my Elaine will you please pray?

Every night she puts up with this,

Yet with a warm embrace,

And a gentle kiss,

She brings me round,

And I awake,

Safe in her arms,

No more to take

[1] Poem by Norman McNamara, who has lewy body dementia and is the founder of the Torbay Dementia Action Alliance (http://tdaa.co.uk/poems/)

PREFACE

Overview

Sleep changes with ageing. As we grow older the likelihood of having sleep disturbances increases. These include changes to sleep timing, primary sleep disorders (e.g., sleep disordered breathing or insomnia), as well as daytime sleepiness or a general dissatisfaction with sleep. Previous international studies show that sleep disturbances are associated with the neurophysiological ageing process as well as with psycho-social factors, demographic status and comorbidities. However there is limited information concerning the sleep of older New Zealanders (aged ≥ 60 years) and the factors that affect it.

Dementia is a progressive brain disease which manifests as cognitive impairment, disruptive behaviours, as well as changes to mood and personality. Dementia is more common with ageing and involves accelerated neuropathological deterioration to the areas of the brain responsible for sleep timing and maintenance. This causes increased sleep disturbances for older people with dementia (PWD, used interchangeably to include "person with dementia"), which can be associated with exacerbated waking symptoms. Previous research has identified that sleep problems are considered by carers as among the most disruptive dementia-related symptoms.

Life expectancy is increasing and the prevalence of dementia is also rising. With the increased pressure on residential care facilitates, there is an increased need and desire for PWD to be cared for within their homes, resulting in more individuals in our communities affected by dementia and requiring support. People with dementia are often cared for informally by a family member. Due to the nature of the disease, carers for PWD are often older themselves and are already predisposed to age-related changes to sleep. Carers' sleep is further disrupted due to being woken by the person in their care, as well as by the physical and mental burden of the caregiver role. Previous research has shown that when the sleep of both PWD and their carer is disturbed, there is increased risk of accidents or injury. Such situations have also been identified as contributing to the decision to institutionalise PWD. Therefore understanding and treating the sleep problems of both PWD and their carers is of importance.

Rationale for Thesis

The sleep of older people affected by dementia is an area with many current research gaps and a growing research need. Through reviewing the literature, it became apparent that there were three key areas which required attention in order to address the sleep of older PWD living in Aotearoa, New Zealand (NZ). Firstly, better understanding is needed of the distribution and prevalence of reported sleep problems among older New Zealanders, as well as the factors associated with reporting a sleep problem, including mental health and caregiving status. Secondly, this research field is moving into a more *person-centred* approach. However there is still a lack of qualitative, first-person reports regarding the sleep experience of PWD, and what PWD and their carers do to try and improve their sleep. In this thesis, three studies were undertaken to address these areas, to provide a basis for designing and conducting a final pilot study to address the third issue, that there is a lack of community-based trials of non-pharmaceutical interventions to improve the sleep of PWD ("community" and "community-dwelling" are used to refer to people living in their own homes, as opposed to institutions). If successful, such interventions could have positive effects on carers' sleep and for the waking symptoms of dementia, thus potentially benefiting both the PWD and their family carers. The ultimate goals of the research are to raise awareness about the importance of sleep health for older people and those affected by dementia, leading to better options for improving the sleep and quality of life of PWD and their family carers.

Organisation of Thesis

This thesis includes four research studies. *Figure 0.1* gives an outline of the topics covered and the progression between the studies to reach the final research question and aims. Studies 1-3 are presented as research papers, two of which have been published in peer-reviewed journals and the other has been submitted for review (see Appendix 1 for statement of contribution forms). These have been reformatted to maintain the style of the thesis. The references are incorporated into the full reference list at the end of the thesis. Study 4 is presented in traditional thesis form.

Studies 1-2 analyse data from two pre-existing cohorts, one of New Zealanders of advanced age (≥ 79 years), the other of retirees. Both cohorts included large samples of Māori participants allowing

equal explanatory power, which made it possible to investigate the factors contributing to self-reported sleep problems of older Māori (the indigenous people of NZ, comprising 14% of the total population, Statistics New Zealand, 2006a). The third study contributed vital information for a person-centred approach for the intervention pilot study (Study 4), through the use of focus groups conducted with PWD and their carers. The focus groups sought to improve understanding of their experience of sleep problems, their beliefs and attitudes towards sleep, and the methods they had in place for managing sleep problems.

Study 4, the central research project for this thesis, was a pilot study trialling non-pharmaceutical interventions with a small sample of PWD and their carers. The methods and results presented in Chapters 4 and 5 relate to Study 4, which is presented as a series of case studies to illustrate the common and the couple-specific effects of the intervention. The final discussion chapter addresses the results, implications and limitations of the pilot study, drawing on the conclusions from the three introductory papers. Overarching the entire thesis are the ethical and methodological considerations involved in conducting research with PWD. The symptoms, stages, and nature of dementia can make this a sensitive and complicated field, and relevant literature is included and revisited throughout the thesis.

The scope of this work crosses several disciplines including psychology, public health, sleep science, and sociology. The Publication Manual of the American Psychological Association (American Psychological Association, 2010) was used to guide many aspects of the presentation of this thesis. However, the style used was also informed by the conventions of the other disciplines listed.

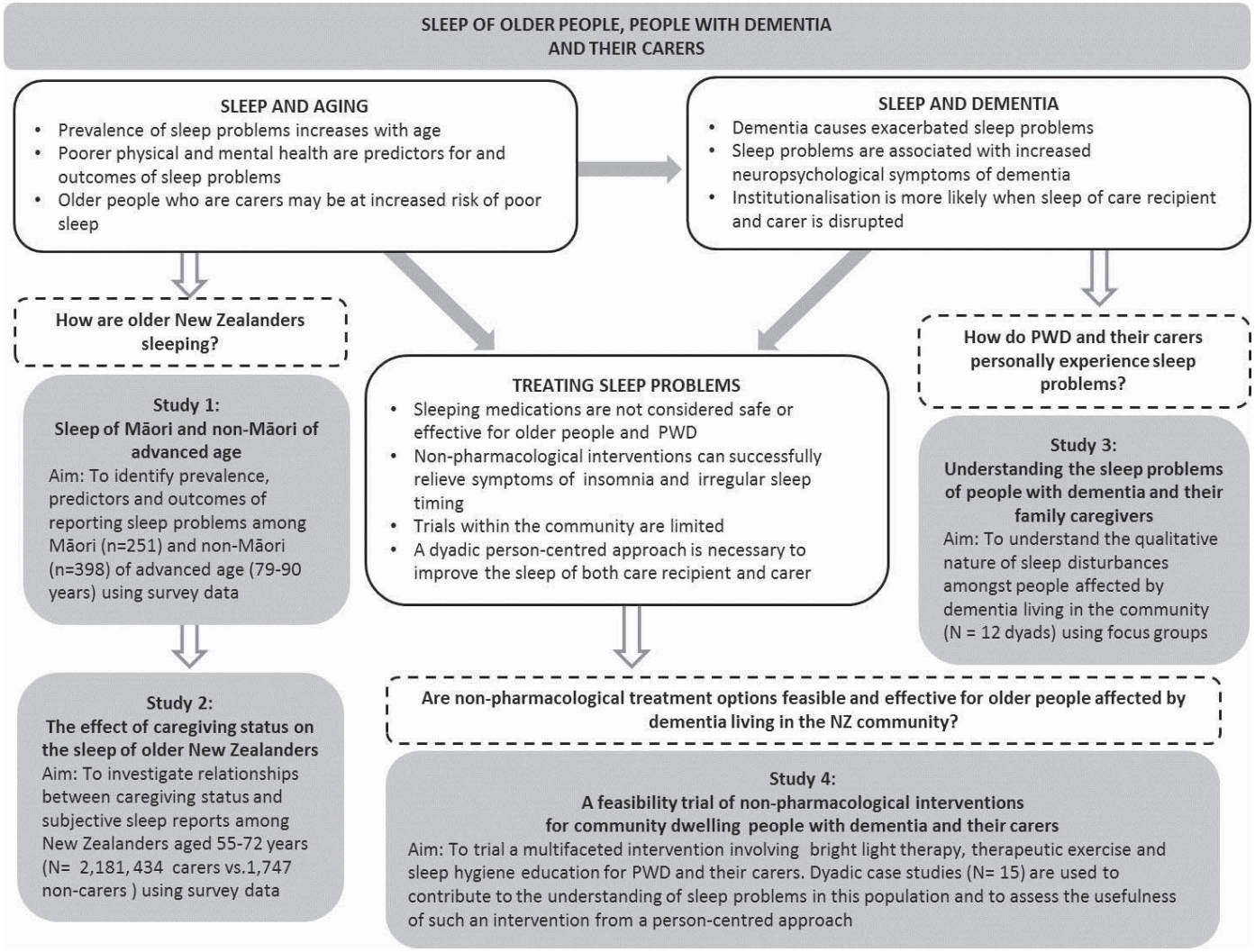


Figure 0.1. Outline of key thesis topics, questions and research studies.

Acknowledgements

I'd like to acknowledge all of the people who took part in the research presented here. I am particularly grateful for the participants who were affected by dementia. I'm aware that, for some of you, incorporating home visits, sleep monitoring, as well as interventions into the daily routine was a lot to take on. *Thank you* for being open to taking part, experiencing something new, and contributing to greater knowledge. I hope you feel that you benefited. Your contributions will help make a difference for other families affected by dementia.

I was only able to reach these participants through the generous support of those assisting me with recruitment. Invaluable support was provided by local support workers, healthcare professionals, and retirement village management who took the time to support this research regarding recruitment and advocacy. I'd especially like to acknowledge the team at Alzheimers Wellington. Thank you Liz O'Hare, Nigel Heard, and Verna Schofield for taking the time to help me design and conduct this research, and having faith in me to work closely with your members. Also to the wider team at Alzheimer's Wellington (Catherine Timms, Trish Howard, Annie Manning, and Leonie Crawshaw) for assisting with advertising, encouraging people to take part, inviting me to promote my study at your support groups, and assisting with conducting focus groups – *Thank you!*

When developing the documents for Study 4, Cass Alexander of Alzheimers New Zealand offered friendly and supportive advice regarding the appropriate use of language and style. Cass introduced me to Nigel and Tania Wynn, who also reviewed the materials for the intervention study. Having somebody with dementia and their partner give feedback on the study design and documents, particularly the sleep education booklet, was invaluable during the early stages of this study. Thank you all for your interest, time, and support.

During the initial stages of this thesis I co-wrote and applied for funding grants to make the dementia-related studies possible. Funding was secured from the Massey Doctorial Scholarships Committee, the Health Research Council (feasibility grant, 11/562), the Maurice and Phyllis Paykel Trust (project and equipment grant, 2010), and the Alzheimer's Charitable Trust (small project grant, 2010).

This funding helped to support me during this journey, and allowed for the purchase of equipment necessary for an intervention and the objective recording of sleep. It also allowed for additional time, and travel expenses deemed necessary for the recruitment and conduct of this community-based project. Without such support this project would have been limited and I am extremely grateful for the opportunity these agencies provided me.

The preliminary studies presented in this thesis were made possible via collaboration with two other research groups: the Life and Living in Advanced Age team at Auckland University, especially Professor Ngaire Kerse; and the Health Work and Retirement team at Massey University, especially Professor Fiona Alpass. Thank you for sharing and trusting me with your valuable data, and contributing to my production of papers regarding the sleep of older New Zealanders. I am grateful to all of the participants of these studies, and the funders of these projects (acknowledged within their relevant chapters). These analyses allowed for the sleep health of older New Zealanders to be described for the first time.

I have been extremely fortunate to have such a supportive group of supervisors during my PhD journey. Linda Jones – your knowledge in health psychology and working with people with dementia helped me to conduct and present my research in an appropriate manner, and your attention to detail is much appreciated. Tony Dowell – you always brought a considered view concerning the conduct and interpretation of my research, and with regards to the bigger picture and clinical framework. Your pragmatic advice and guidance helped to keep me and these projects grounded in what matters, the people. To my primary supervisor, Philippa Gander, thank you for supporting me in my second postgraduate thesis. You are a sucker for punishment! Thank you for providing me with the opportunity to undertake a piece of work in an area that I was, and still am, passionate about. Your enthusiastic, pragmatic, and respectful approach to me and my research facilitated an encouraging and supportive environment for my journey.

Philippa is not only an internationally acclaimed expert in sleep science, she is also the director of a wonderful and inspiring group of researchers at the Sleep/Wake Research Centre. Being a member of this group provided me with immeasurable support throughout my post graduate journey. Philippa and co-

director, Leigh Signal welcomed me as a Masters student in 2008. I was an international student from England on a one-year scholarship to conduct research regarding the factors affecting infant sleep. Six years have passed and somehow I have comfortably become a part of the furniture and written another thesis concerning the other end of the life-span. This is down to the incredibly open, friendly, and supportive environment and people that constitute Sleep/Wake. I'd especially like to acknowledge Bronwyn Sweeney, office buddy extraordinaire, who has been my thesis writing companion throughout. I cannot wait to graduate and celebrate with you! Sarah-Jane Paine, you have been a fantastic mentor and friend, always there for a coffee with fantastic statistical and writing advice. Kanch Pathirana, thank you for your assistance with data entry and double scoring, I miss your bubbly presence around the lab. Karyn O'Keefe, you are a fountain of knowledge, thank you for being there and sharing your ideas! And to my new PhD buddies: Dee Muller, Margo van den berg, and Jen Zaslona; thank you for your words of enthusiasm, coffee dates, and companionship. It makes such a difference having friends like you to share the journey with.

I'd also like to dedicate this work to my families, the Gibsons and my new in-laws, the Mouldeys. Mum and dad, thank you for believing in me and encouraging me, even if it did mean living on the other side of the world. And Noel and Beryl, thank you for taking me in and helping me to achieve my goals. I think I've almost made it, next stop a glass of wine and a game!

Finally, I am indebted to my husband Gavin, who has stood by me through three degrees, listening to my woes, reading my work, and having faith in me all of the way. You are my rock. Thank you for all of the sacrifices you have made for the sake of my career. I am so lucky to have you as my life partner, as Piglet said to Winnie the Pooh: "If you live to be 100, I hope I live to be 100 minus one day, so I never have to live without you".

My baby, Lyla, came and provided an intermission for me during this research project. Thank you, my love, for sleeping so beautifully and giving me the opportunity to write. Thank you for your warm cuddles and laughter that have motivated me to get this job done so I can spend more time with you. Gavin and Lyla, if I ever have the misfortune of losing you in my mind, you will always, *always* be in my heart.

Glossary and Abbreviations

Actigraphy	A method of assessing rest and activity, using a small wrist-worn device over a period of days or weeks
Advanced age	Individuals aged more than 79 years
AChEI	Acetylcholinesterase inhibitors
AD	Alzheimer's disease
ApoE	Apolipoprotein E gene
ARAS	Ascending reticular activating system
BLT	Bright light therapy
BZD	Benzodiazepine
Carer/caregiver	Used interchangeably to describe someone who provides care or support for someone with a disability
Circadian dysrhythmia	Changes in sleep timing and fragmentation of the sleep/wake pattern
CBT-I	Cognitive behaviour therapy for insomnia
CI	Confidence interval
CME	Continuing medical education
Community-dwelling	Used to refer to people living in their own homes (as opposed to institutions)
Constant routine	Protocol using a controlled environment with the absence of external time cues in order to accurately measure markers of circadian regulation
COPE index	Carers of Older People in Europe index
CSA	Central sleep apnoea
Declarative memory	Explicit knowledge, e.g., what something is
DMH	Dorsomedial nucleus of the hypothalamus
DRBs	Dementia-related behaviours
Dx	Diagnosis
Dyadic approach	Refer to the act of considering the experience of the carer and care recipient at the same time in order to more reliably and ethically inform understanding and treatment
EEG	Electroencephalography
FDA	Food and drug administration
GABA	Gamma-amminobutyric acid
GDS	Geriatric Depression Scale
GP	General Practitioner
HADS	Hospital Anxiety and Depression Scale
CHDEC	Central Health and Disability Ethics Committee
HWR	Health work and retirement
ID	Identification
Informal carer/family carer/whānau carer	Used interchangeably to refer to those providing unpaid care, usually to a family member or friend
Institution	Used to refer to residential or nursing homes or hospitals
Interdaily stability	A figure calculated from actigraphic data to represent the stability of day-to-day circadian timing
Intradaily variability	A figure calculated from actigraphic data to represent the variability in the amount of sleep and wake per hour across the 24-hour day
Koha	Māori term meaning donation/gift
LiLACSNZ	Life and living in advanced age: A cohort study in NZ
LBD	Lewy body dementia

LTD	Light therapy device
Lux	Units of measure for light intensity
Māori	The indigenous population of NZ
MCI	Mild cognitive impairment
MMSE	Mini Mental State Exam
NEAC	National ethics advisory committee
N	Sample size
NBZ	Non benzodiazepine
Non declarative memory	Implicit knowledge, e.g., knowing how to perform a task
NREM	Non-rapid eye movement
NZ	Aotearoa/New Zealand
NZDep	NZ Deprivation index
Older	Typically used to refer to individuals aged ≥60 years
OR	Odds ratio
OSA	Obstructive sleep apnoea
PASE	Physical Activity Scale for the Elderly
PLMS	Periodic limb movements of sleep
PMS	Pearlin Mastery Scale
Polypharmacy	Taking ≥ 5 medications
Polysomnography	A method of measuring sleep using direct physiological measures, usually in a laboratory setting
PSQI	Pittsburgh Sleep Quality Index
PWD	Used interchangeably for "people with dementia" or "person with dementia"
QOL-AD	Quality Of Life in Alzheimer's Disease
RCT	Randomised controlled trial
REM	Rapid eye movement
RHT	Retino-hypothalamic tract
RLS	Restless legs syndrome
RMBPC	Revised Memory and Behaviour Problem Checklist
SCN	Suprachiasmatic nuclei
SD	Standard deviation
SDI	Sleep Disorders Inventory
SF-12	Short Form 12 item survey
Sleep hygiene	An individual's behaviours and environment that can influence sleep
SPZ	Supraventricular zone of the hypothalamus
SWRC	Sleep/Wake Research Centre
SWS	Slow wave sleep
UK	United Kingdom
USA	United States of America
VaD	Vascular dementia
VLPO	Ventrolateral preoptic nucleus
Whānau	Māori term meaning family including extended family and community
Young old	Individuals aged between 60-79 years

Table of Contents

1 SLEEP AND AGEING 1

1.1 Defining Sleep 1

1.1.1 Sleep Architecture and Changes with Ageing 1

1.1.2 Basic Neurophysiology of Sleep and Changes with Ageing 4

1.2 The Regulation of Sleep Timing and Changes with Ageing 7

1.2.1 Circadian Rhythms 7

1.2.2 The Homeostatic Sleep Process 11

1.2.3 Sleep Regulation and Ageing 13

1.3 Sleep Disorders and Ageing 16

1.3.1 Sleep Apnoea 17

1.3.2 Restless Leg Syndrome and Periodic Limb Movements of Sleep 18

1.3.3 Insomnia/ "Poor Sleep" 19

1.3.4 Sleep of Older Carers 23

1.4 Rationale for Studies 1 and 2 27

1.5 Study 1: Sleep of Māori and non-Māori of Advanced Age.... 29

1.5.1 Abstract 29

1.5.2 Introduction 29

1.5.3 Methods 31

1.5.4 Results 34

1.5.5 Discussion 39

1.6 Study 2: The Effect of Caregiving Status on Sleep of Older New Zealanders 43

1.6.1 Abstract 43

1.6.2 Introduction 43

1.6.3 Methods 44

1.6.4 Results 46

1.6.5 Discussion 50

1.7 Overview of the Findings from Studies 1 and 2..... 53

2 SLEEP AND DEMENTIA 55

2.1 What is Dementia? 56

2.1.1 Alzheimer's Disease 59

2.1.2 Vascular Dementia 62

2.1.3 Lewy Body Dementia 63

2.2 Dementia-Related Sleep Disturbances 65

2.2.1 Changes to Sleep Architecture with Dementia 67

2.2.2 Changes to Sleep Regulation with Dementia 70

2.2.3 Primary Sleep Disorders and "Problem Sleep" in Dementia 77

2.2.4 Summary 83

2.2.5 Sleep of those Caring for a Family Member with Dementia 84

2.3 Philosophy of Conducting Research with PWD and their carers 89

2.3.1 Actively Including People with Dementia in Research 89

2.3.2 Research with PWD/Carer Dyads 91

2.3.3 A Qualitative, Person-Centred Approach 92

2.4 Rationale for Study 3 93

2.5 Study 3: Understanding the Sleep Problems of People with Dementia and their Family Caregivers 94

2.5.1 Abstract 94

2.5.2 Introduction 94

2.5.3 Design and Methods 96

2.5.4 Findings 99

2.5.5 Discussion 107

2.6 Personal Reflection 112

3 TREATING SLEEP DISTURBANCES OF OLDER PEOPLE AND PEOPLE WITH DEMENTIA 113

3.1 Pharmacological Treatments 114

3.1.1 Pharmacological Approaches for Improving Sleep Quality or Duration 114

3.1.2 Pharmacological Approach for Improving Circadian Dysrhythmia 118

3.2 Non-Pharmacological Therapies 121

3.2.1 Cognitive and Behavioural Strategies for Problem Sleep 121

3.2.2 Chronotherapeutics for Circadian Dysrhythmia 123

3.2.3 A Person-Centred, Dyadic Approach to Treating Dementia-Related Sleep Problems 138

3.3 Rationale for Study 4 140

3.4 Ethical & Methodological Considerations for Dementia-Related Research 142

3.4.1 Risks and Benefits for Participants 142

3.4.2 Recruitment 144

3.4.3 Informed Consent and Assent 145

3.4.4 Collecting Dementia-Related Data 149

3.4.5 Considerations for Study 4 150

4 STUDY 4 METHODS 153

4.1 Design 153

4.2 Ethics 153

4.3 Recruitment of Participants 154

4.3.1 Recruitment Phase One 155

4.3.2 Recruitment Phase Two 159

4.3.3 Recruitment Phase Three 160

4.4 Materials 162

4.4.1 Questionnaires for People with Dementia 162

4.4.2 Questionnaires for Carers, about Themselves 168

4.4.3 Questionnaires for Carers about the PWD 171

4.5 Materials: Sleep Monitoring 174

4.5.1 Actigraphy 174
4.5.2 Sleep Diaries 175
4.6 Interventions 176
4.6.1 Bright Light Therapy 177
4.6.2 Therapeutic Exercise 178
4.6.3 Sleep Hygiene Education 178
4.7 Materials: Feedback Questionnaire 179
4.8 Procedure 179
4.8.1 Screening 179
4.8.2 Consent 181
4.8.3 Week One (Time 1) 182
4.8.4 Weeks Two-Five 186
4.8.5 Week Six (Time 2) 188
4.8.6 Withdrawal 189
4.9 Data Analysis 190
4.9.1 Analysis and Presentation of Questionnaire Data 192
4.9.2 Analysis and Presentation of Diary data 193
4.9.3 Analysis and Presentation of Actigraphy Data 194
4.9.4 Analysis and Presentation of Field Notes and Feedback 200
4.10 Approach to the Presentation of Results 201
5 STUDY 4 RESULTS 203
5.1 Description of the Full Sample at Time 1 204
5.1.1 Demographic Details 204
5.1.2 Summary of Results at Time 1 206
5.1.3 Questionnaire Data by Completion Status 212
5.1.4 Actigraphy Data by Completion Status 217
5.1.5 Light and Activity, Time 1 222
5.1.6 Participant Feedback 224
5.2 Summary of Pairs who withdrew 225

5.3 Case Studies of Participants who Completed the Full Study 227

5.3.1 Pair 1 (Liam and Sophie) 228

5.3.2 Pair 2 (Jack and Ella) 236

5.3.3 Pair 3 (George and Amy) 246

5.3.4 Pair 4 (Thomas and Sarah) 255

5.3.5 Pair 5 (Adam and Claire) 263

5.3.6 Pair 6 (Anne, Matthew and Katie) 275

5.3.7 Pair 7 (Violet and Charlie) 283

5.3.8 Pair 8 (Leonard and Phoebe) 292

5.3.9 Pair 9 (Andrew and Emily) 300

5.3.10 Summary of Data Between Times 1 and 2 309

5.4 Summary of Findings: Study 4 318

6 DISCUSSION 321

6.1 Thesis Findings: Contributions to the Literature 323

6.1.1 How are Older New Zealanders Sleeping? 323

6.1.2 How do PWD and their Carers Personally Experience Sleep Problems? 324

6.1.3 Are Non-Pharmacological Treatment Options Feasible and Effective for Community-Dwelling PWD and their Carers? 325

6.2 Limitations and Recommendations 332

6.2.1 Recruitment Issues 332

6.2.2 Sample Biases 335

6.2.3 Ethical Considerations for Inclusion and Retention of Participants 339

6.2.4 Methods, Protocol, and Compliance 340

6.2.5 Recommendations 349

6.3 Implications 352

6.3.1 Research Implications 353

6.3.2 Clinical implications 354

6.3.3 Community implications 357

6.4 Conclusion 357

7 REFERENCES 361

8 APPENDICIES 389

1 Statement of Contributions to Doctoral Thesis Containing Publications 389

2 Study 3: Supporting Documents 392

3 Study 4: Ethics Approval 402

4 Study 4: Examples of Recruitment-Related Documents[1] 403

5 Questionnaire: PWD 410

6 Questionnaire: Carer 414

7 Sleep Diary Examples 421

8 Sleep Support Handbook 430

9 Feedback Questionnaire 443

10 Study 4: Information for Participants 444

11 Study 4: Consent Forms 449

12 Example Letters to GP 451

13 Participant Instructions: Sleep Monitoring 454

14 Participant Instructions: Sleep Intervention 455

15 Fridge Magnet 458

16 Example of Feedback Letter to Participants 459

17 Example of Feedback Letters to GP 460

18 Software Procedures used to Define Actigraphy Variables 462

19 Case Studies of Pairs who Withdrew 464

Pair 10 (Nicole and Claude) 464

Pair 11 (Henry and Beverly) 469

Pair 12 (Sidney and Fiona) 475

Pair 13 (Maria and Felix) 480

Pair 14 (Georgina and Lisa) 485

Pair 15 (Edward and Rose) 491

20 Medications Being Taken by all PWD and Carers at Time 1 497

21 Actograms Concerning Pairs who Completed the Trial 504

22 Additional Results Concerning Pairs who Completed the Trial 522

List of Tables

Table 1.1 *Description of EEG Activity of Young Adults During Wakefulness, NREM Stages 1-4, and REM sleep* 2

Table 1.2 *Predictors and Outcome Variables Considered for Logistic Regression Models* 33

Table 1.3 *Demographic Characteristics of Māori and Non-Māori Participants in the Sleep Sample* 35

Table 1.4 *Independent Associations Between Reporting a Current Sleep Problem and Demographic and Health-Related Factors* 37

Table 1.5 *Independent Associations between Reporting Current or Past Sleep Problems and Poorer Health* 38

Table 1.6 *Description of Carers* 47

Table 1.7 *Sleep Responses by Caregiving Status* 48

Table 1.8 *Associations Between Reporting Feeling Worn out, Tired, Dissatisfied With Sleep, or Having a Diagnosed Sleep Disorder, with Caregiving and Other Demographic Factors* 49

Table 4.1 *Inclusion/exclusion Criteria Used in Recruitment Phase One* 156

Table 4.2 *Recruitment Strategies Used in Phase One, Alzheimers NZ & Alzheimers Wellington* 157

Table 4.3 *Recruitment Strategies Used in Phase One, Other Senior Services* 158

Table 4.4 *Recruitment Strategies used in Phase Two, Healthcare Professionals* 161

Table 4.5 *Reasons for Potential Participants not Taking Part in the Intervention Study* 162

Table 4.6 *Overview of Materials Used in Study 4* 163

Table 4.7 *Reference for Interpreting Questionnaire, Diary and Actigraphy Variables* 191

Table 4.8 *Descriptions of the Variables Calculated from the Actigraphy Records* 199

Table 5.1 *Demographics of the Full Sample and Summary of Each Dyad's Outcome* 205

Table 5.2 *Summary of Questionnaire and Diary Data for PWD and Carers at Time 1 (N=15)* 208

Table 5.3 *Pittsburgh Sleep Quality Index: Item Level Descriptive Statistics* 209

Table 5.4 *Sleep Disorders Inventory: Item Level Descriptive Statistics* 210

Table 5.5 *Summary of Actigraphy Data for all PWD (n=15) and Carers (n=14) at Time 1* 211

Table 5.6 *Summary of Light Exposure (9-11am) and Physical Activity (11am-2pm) for all PWD at Time 1* 222

Table 5.7 *Frequencies and Percentages of How Participants Rated Data Collection (n=16)* 224

Table 5.8 *Frequencies and Percentages of How Participants Rated Aspects of the Intervention (n=9)* 224

Table 5.9 *Factors Affecting Compliance and Study Withdrawal: Case Studies 10-15* 226

Table 5.10 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 1* 229

Table 5.11 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 1* 231

Table 5.12 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 1* 233

Table 5.13 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 1* 234

Table 5.14 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 1* 235

Table 5.15 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 2* 238

Table 5.16 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 2* 239

Table 5.17 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 2* 241

Table 5.18 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 2* 243

Table 5.19 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 2* 245

Table 5.20 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 3* 248

Table 5.21 *Comparison of Questionnaire Data at Times 1 and 2 Concerning PWD 3* 249

Table 5.22 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 3* 251

Table 5.23 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 3* 252

Table 5.24 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 3* 253

Table 5.25 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 4* 256

Table 5.26 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 4* 257

Table 5.27 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 4* 259

Table 5.28 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 4* 260

Table 5.29 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 4* 262

Table 5.30 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 5* 265

Table 5.31 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 5* 267

Table 5.32 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 5* 269

Table 5.33 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 5* 271

Table 5.34 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 5* 273

Table 5.35 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 6* 277

Table 5.36 *Comparison of Questionnaire Data at Times 1 and 2 Concerning PWD 6* 278

Table 5.37 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 6* 280

Table 5.38 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 6* 281

Table 5.39 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 7* 284

Table 5.40 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 7* 286

Table 5.41 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 7*..... 288

Table 5.42 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 7* 289

Table 5.43 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 7*..... 290

Table 5.44 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 8* 293

Table 5.45 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 8* 295

Table 5.46 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 8* 297

Table 5.47 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 8* 298

Table 5.48 *Actigraphic Sleep Outcomes at Time 1 Concerning Carer 8* 298

Table 5.49 *Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 9* 302

Table 5.50 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 9* 303

Table 5.51 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 9*..... 305

Table 5.52 *Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 9* 306

Table 5.53 *Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 9*..... 308

Table 5.54 *Ranked Position of Change of PWD's PSQI Scores Between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables* 315

Table 5.55 *Ranked Position of Change of PWD's % Sleep at Night Between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables* 316

Table 5.56 *Ranked Position of Change of Carers PSQI scores between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables.* 317

Table 6.1 *Comparison of Non-Pharmacological Interventions used in Study 4 with Previous Community-Based Trials* 327

Table 6.2 *Comparison of Study 4 Sample and Actigraphic Sleep Data with Previous Community-Based Trials ^*..... 330

List of Figures

Figure 1.1. Hypnogram example of younger adults' NREM/REM cycle across the night. 3

Figure 1.2. Hypnogram example of older adults' NREM/REM cycle across the night. 4

Figure 1.3. The flip-flop switch model of sleep and wake. 5

Figure 1.4. The opponent processes model of sleep/wake timing. 13

Figure 1.5. The proportion of sleep symptoms endorsed by Māori (n=66) and non-Māori (n=126) participants who reported having a current sleep problem. 36

Figure 1.6. Zealand Deprivation Index 2006 (NZdep) profile for carers at home, other carers and non-carers (n=2,164). 47

Figure 2.1. The temporal patterning of sundowning behaviours. 66

Figure 2.2. Categorisation of learning processes and how sleep contributes to consolidated memory. 70

Figure 2.3. Average amount of time spent sleeping in each hour across a 24-hour period. 73

Figure 2.4. Factors associated with poor sleep regulation for people with dementia. 74

Figure 2.5. Conceptual framework showing the intrinsic and extrinsic factors and mediating context associated with the presentation and reporting of PWD's sleep problems. 86

Figure 2.6. Global and organising themes concerning the sleep of those with dementia. 100

Figure 2.7. Global and organising themes concerning the carers. 102

Figure 3.1. Phase response curve to 1 hour high intensity (stair climbing) exercise at different times in the SCN pacemaker cycle. 127

Figure 3.2. Phase response curve to bright light pulses (10,000 lux, for 6.7 hours) administered at different times in the SCN pacemaker cycle. 132

Figure 3.3. The sleep state ratio of a PWD trailing BLT. 137

Figure 3.4. Considerations for the appropriate and ethical conduct of dementia-related research, based on the reviewed literature. 151

Figure 4.1. The Mini Mitter Actiwatch-2 ™ 175

Figure 4.2. Timing of prescribed intervention plotted on the approximate circadian phase. 176

Figure 4.3. The Day-Light LTD (Uplift Technologies, 2007). 177

Figure 4.4. Overview of protocol for Study 4. 180

Figure 4.5. An example of 1 week of actigraphy recording from a PWD. 195

Figure 4.6. Actiware® software *algorithm for calculating the sum of activity counts for a 1 minute sampling epoch in order to score sleep or wake.* 196

Figure 4.7. An example of the sleep propensity curves of a participant with dementia, based on seven 24-hour periods at Time 1 and at Time 2. 200

Figure 5.1. Global MMSE scores of PWD and carers who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data). 213

Figure 5.2. RMBPC-disruptive behaviour scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15). 214

Figure 5.3. RMBPC-memory scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not IDs 10-15). 214

Figure 5.4. QOL-AD scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15)..... 215

Figure 5.5. HADS anxiety and depression scores of carers who completed the full trial (IDs 1-9) and those who did not (IDs 10-14, Time 1 data). 215

Figure 5.6. Positive and negative COPE scores of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 216

Figure 5.7. Global PSQI scores of PWD and carers who completed the full trial (IDs1-9) and those who did not (IDs 10-15, Time 1 data). 216

Figure 5.8. Global SDI scores of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data). 217

Figure 5.9. Total sleep time at night of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data). 218

Figure 5.10. Total sleep time at night of carers who completed the full trial (IDs 1-9) and those who did not (IDs10-15, Time 1 data). 218

Figure 5.11. Number of wake bouts at night of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 219

Figure 5.12. Number of wake bouts at night of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 219

Figure 5.13. Sleep efficiency of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 220

Figure 5.14. Sleep efficiency of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 220

Figure 5.15. Total sleep duration in the daytime of PWD who completed the full trial and those who did not (Time 1 data). 221

Figure 5.16. Total sleep duration in the daytime of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data)..... 221

Figure 5.17. Total light exposure recorded between 9-11am of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data). 223

Figure 5.18. Average activity counts per minute between 11am-2pm of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data). 223

Figure 5.19. Flow chart illustrating the presentation of case studies..... 227

Figure 5.20. Sleep timing of PWD 1 at Time 1 and Time 2 (7 days). 232

Figure 5.21. Sleep of carer 1 at Time 1 and Time 2 (7 days). 234

Figure 5.22. Sleep timing of PWD 2 at Time 1 and Time 2 (6 days). 240

Figure 5.23. Two days of actigraphy data extracted from Jack's (top) and Ella's (bottom) sleep studies at Times 1 and 2..... 242

Figure 5.24. Sleep of carer 2 at Times 1 and 2 (7 days)..... 244

Figure 5.25. Sleep timing of PWD 3 at Time 1 and Time 2 (6 days). 250

Figure 5.26. Sleep of carer 3 at Times 1 and 2 (6 days)..... 254

Figure 5.27. Sleep timing of PWD 4 at Time 1 and Time 2 (7 days). 258

Figure 5.28. Sleep of carer 4 at Times 1 and 2 (7 days)..... 261

Figure 5.29. Sleep timing of PWD 5 at Time 1 and Time 2 (6 days). 268

Figure 5.30. Two days of actigraphy data extracted from Adam (top) and Claire's (bottom) sleep studies at Times 1 and 2..... 270

Figure 5.31. Sleep of carer 5 at Times 1 and 2 (6 days)..... 272

Figure 5.32. Sleep timing of PWD 6 at Time 1 and Time 2 (8 days). 279

Figure 5.33. Sleep timing of PWD 7 at Time 1 and Time 2 (3 days). 286

Figure 5.34. Sleep of carer 7 at Times 1 and 2 (3 days)..... 289

Figure 5.35. Sleep timing of PWD 8 at Time 1 and Time 2 (4 days). 295

Figure 5.36 Sleep of PWD 9 at Times 1 and 2 (7 days)..... 304

Figure 5.37. Sleep of carer 9 at Times 1 and 2 (6 days)..... 307

Figure 5.38. Comparison of global PSQI scores of PWD between Times 1 and 2..... 311

Figure 5.39. Comparison of global PSQI scores of carers between Times 1 and 2. 311

Figure 5.40. Comparison of total sleep time at night of PWD between Times 1 and 2. 312

Figure 5.41. Comparison of total sleep time at night of carers between Times 1 and 2. 312

Figure 5.42. Comparison of sleep efficiency at night (sleep %) of PWD between Times 1 and 2.313

Figure 5.43. Comparison of sleep efficiency at night (sleep %) of carers between Times 1 and 2.313

Figure 5.44. Comparison of number of wake bouts at night of PWD between Times 1 and 2.. 314

Figure 5.45. Comparison of number of wake bouts at night of carers between Times 1 and 2... 314

Figure 6.1. Key findings, recommendations for future research, and implications from the thesis.322

Figure 6.2. Conceptual framework showing the distal and proximal factors and the mediating context associated with the successful recruitment, engagement, and retention of PWD and carers in research. 334

1 SLEEP AND AGEING

In order to understand how sleep changes with dementia it is important to be aware of the changes that occur in healthy ageing without dementia. Increased sleep disturbances with ageing have been associated with several factors including neurophysiological changes affecting the timing and quality of sleep, changes to the type and intensity of circadian time cues that individuals are exposed to, and the exacerbation of pre-existing sleep disorders. The likelihood of having disturbed sleep also varies with demographic factors, physical and mental comorbidities, as well as changes to expectations around sleeping, and caregiving status. This chapter gives an overview of sleep and its changes with age.

1.1 Defining Sleep

1.1.1 Sleep Architecture and Changes with Ageing

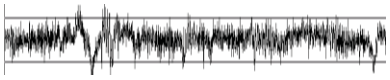
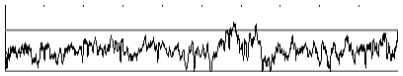
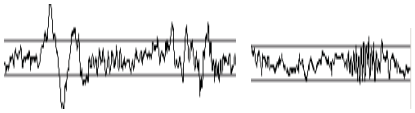
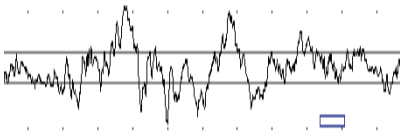
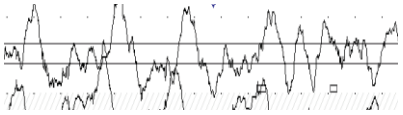
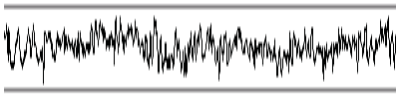
Sleep is not simply a passive state opposing the active state of wake. The advent of the electroencephalogram (EEG) in the 1930s allowed scientists to record and view electrical activity from the cortex (Loomis, Harvey, & Hobart, 1937). These early studies showed that the brain clearly remains active during sleep. The term *sleep architecture* refers to sleep stages and depth defined by the EEG. Eye movements and muscle tone are also recorded through electrodes, these attached around the eyes and chin to help define particular stages of sleep. The process of measuring sleep in this way is called *polysomnography*. Polysomnography typically takes place in a laboratory setting and it is considered the gold standard for measuring sleep (Collop, 2006). Additional channels measuring airflow, oxygen saturation and limb movements can also be included to diagnose particular sleep disorders (see section 1.3).

Studies using polysomnography identify five sleep stages within two distinctly different sleep states: non-rapid eye movement sleep (NREM) and rapid eye movement sleep (REM). The four stages of NREM sleep are on a continuum of depth and threshold for arousal (from the lightest, Stage 1, to the deepest, Stage 4). Sleep is entered through NREM stage one, often accompanied by slow rolling eye movements. Stage 2 is a light stage of sleep characterised by EEG phenomena known as sleep spindles and K-complexes. Slow wave sleep (SWS) consists of stages 3 and 4. Slow wave sleep is characterised by slow frequency and high amplitude delta waves (≤2 hertz). It is the deepest stage of sleep, therefore being

roused from SWS is particularly difficult. The state of REM sleep is very different from NREM. The frequency and amplitude of the EEG is similar to that of wake. However REM is differentiated by the loss of muscle tone in the body, an increase of eye movements, and the experience of dreaming (Rechtschaffen & Kales, 1968). Table 1.1 gives an overview of these sleep stages as well as some example EEG traces.

Table 1.1

Description of EEG Activity of Young Adults During Wakefulness, NREM Stages 1-4, and REM sleep

Sleep stage		Description	EEG trace example
Wake		low voltage, fast activity	
NREM	1	Alpha waves (8-13 hertz) mixed with theta waves (3-7 hertz)	
	2	K complex wave forms and sleep spindles (12-14 hertz) appear	
	3	High amplitude delta waves (0.5-2 hertz) range make up 50% or less of the 30 second epoch	
	4	Delta waves make up over 50% of the 30 second epoch	
REM		Low voltage, fast activity, eye movements and low muscle activity differentiates from waking EEG	

Patient data, reused with permission

Within the sleep period, the stages of sleep have an approximately 90-minute cycle. In healthy adults, this cycle begins with NREM sleep which moves progressively into deeper states. After about 90 minutes, individuals move back into Stage 2 sleep and then on into REM. Although the cycles of sleep remain approximately 90 minutes long throughout the sleep periods, those during the first half of the night typically have more SWS compared to REM, whereas the second half of the night has more REM and Stage 2 sleep compared to SWS (Carskadon & Dement, 2011; Gander, 2003). *Figure 1.1* shows a graphical example (hypnogram) of the sleep of a healthy adult.

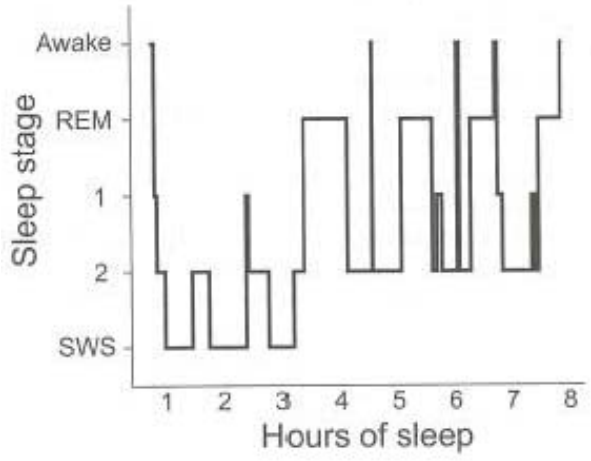


Figure 1.1. Hypnogram example of younger adults' NREM / REM cycle across the night.

(Wright & Frey, 2008, p. 6).

Changes to sleep architecture with ageing.

Previous research using EEG with older participants has typically reported a reduction in the duration of total sleep time and the amount and density of SWS compared to younger adults (Carrier et al., 2011; Dijk, Duffy, Kiel, Shanahan, & Czeisler, 1999; Landolt, Dijk, Achermann, & Borbély, 1996). With the reduced slow wave activity, there tends to be an increase in the lighter Stage 2 sleep. However the structure of Stage 2 sleep differs from younger adults in that there is a reduction in the density and frequency of sleep spindles. Research on changes to REM sleep is inconclusive, with some studies showing significant reductions or changes to timing, whereas others show no change (Bliwise, 2011a; Carrier, 2010; Landolt & Borbély, 2001). *Figure 1.2* shows an example hypnogram of the sleep stages of an older person. These changes mean that older people have lighter sleep, so are more likely to be woken at night (e.g., by external noise or temperature changes) and have increased time spent awake after sleep onset. Older people are thus more likely to report feeling tired in the daytime and more likely to nap (Foley et al., 2007). A number of studies have associated Stage 2 and REM sleep with the processing and consolidation of memories (Fogel & Smith, 2011; Peigneux & Smith, 2011; C. Smith, Aubrey, & Peters, 2004; Stickgold, 2005). Changes or deprivation to these stages of sleep could therefore contribute to common problems in cognitive functioning with ageing (see section 2.2.1 for more detail).

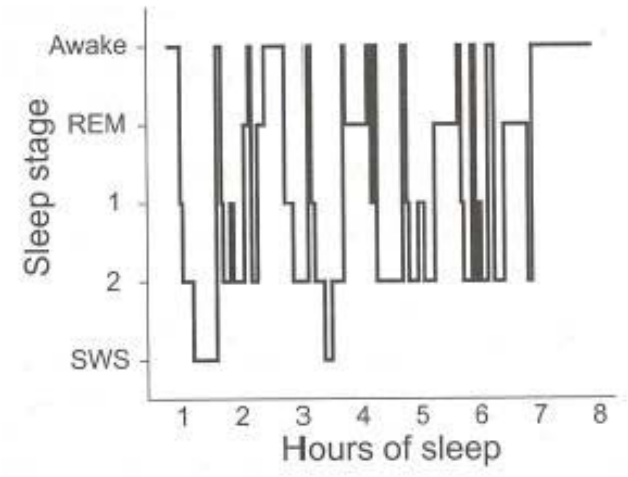


Figure 1.2. Hypnogram example of older adults' NREM / REM cycle across the night.

(Wright & Frey, 2008, p. 6).

1.1.2 Basic Neurophysiology of Sleep and Changes with Ageing

The stages of sleep are dependent on neural sleep and wake promoting systems. These systems are mutually inhibitive and produce either states of NREM sleep, wake, or REM sleep. They involve many areas of the brain and associated neurotransmitters, which alternate in patterns of release and inhibition (see McGinty & Szymusiak, 2011).

Wakefulness is associated with activity within the ascending reticular activating system (ARAS) and brainstem. More specifically these areas include the raphe nuclei, hypothalamus and locus coeruleus. These areas widely project excitatory neurotransmitters (noradrenaline, serotonin, histamine, acetycholine, and orexin) to the cortex accounting for global wakefulness. The noradrenergic, dopaminergic, histaminergic, and serotonergic systems are all more active during wakefulness, and inactive during both NREM and REM sleep states. The cholinergic system is also active during wakefulness and during REM sleep as well. Rather than projecting directly to the cortex, the cholinergic system activates via pathways within the thalamus, hypothalamus and basal forebrain (Fuller, Gooley, & Saper, 2006; Marks, 2006; McGinty & Szymusiak, 2011; Saper, Chou, & Scammell, 2001).

During sleep, activity within the ARAS is reduced. The neurotransmitter adenosine is associated with the onset of sleep and has inhibitory effects on the basal forebrain. Collections of neurons within the ventrolateral preoptic nucleus (VLPO) are active during sleep. These are considered to play a role in the

generation of sleep by their transmission of gamma-amminobutyric acid (GABA) and galin which have inhibitory effects on the cortex, thereby reducing levels of arousal. The VLPO nucleus appears to have reciprocal connections to the wake-related structures mentioned above. During sleep the VLPO inhibits the expression of orexin which is considered to play an important role in facilitating wakefulness (Fuller, et al., 2006; Marks, 2006; McGinty & Szymusiak, 2011; Saper, Scammell, & Lu, 2005). Therefore, it is hypothesised that the sleep and wake systems have a mutually inhibitive relationship serving to maintain the cortex in one or the other state. When the activity in one outweighs the other, the state is switched, hence a common analogy used is a see saw or "flip flop switch" (McGinty & Szymusiak, 2000; Saper, et al., 2001). *Figure 1.3* shows a schematic diagram of this model.

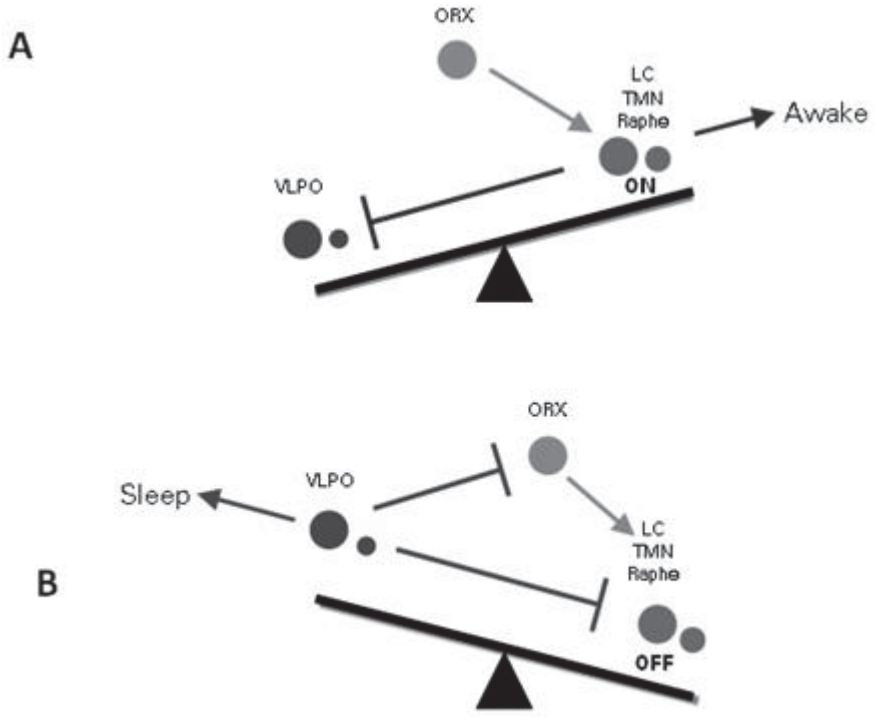


Figure 1.3. The flip-flop switch model of sleep and wake.

Modified from Saper, Scammell et al. (2005).Figure 1.3.A. indicates that inhibition of the ventrolateral preoptic nucleus (VLPO) creates disinhibition of the wake promoting systems in the raphe nuclei, tubermammillary nucleus (TMN) and locus coruleus (LC). The neurotransmitter, orexin (ORX) facilitates this activity and the wake promoting system is regarded as "on". Figure 1.3.B. shows the reverse, the inhibited wake-promoting systems results in disinhibition of the VLPO and therefore disinhibited ORX. Therefore the wake promoting system is "off" and sleep is the dominant state.

The production of REM versus NREM sleep is related to specific patterns of neurotransmitter release. During NREM all of the waking systems are inhibited via the release of GABA and galin, producing a deep level of sleep which is more difficult to be roused from. However, during REM sleep, high levels of acetylcholine and orexin are still present. Activity within the medulla oblongata and pons is associated with the phasic events of REM such as eye movements and muscle twitches, as well as the tonic states of widespread neural activity and muscle atonia. Although many of the areas and neurotransmitters associated with wakefulness are active during REM, the aminergic systems remain inhibited via the transmission of GABA and galin from the VLPO. Therefore the transmission of noradrenaline, serontonin, and histamine is inhibited, maintaining a state of sleep (Fuller, et al., 2006; Marks, 2006; McGinty & Szymusiak, 2011). This phenomena is described as another sleep switch, the "REM switch", which is modulated by the cholinergic (REM-on) and aminergic (REM-off) systems enabling smooth transitions between REM and NREM sleep (McCarley, 2004; Saper, Scammell, et al., 2005).

Abnormalities in one or more of the components of the sleep, wake, or REM systems have been shown to cause unstable states of consciousness and/or disordered sleep. With healthy ageing, a reduction in the number of neurons in the VLPO has been observed, as well as a reduction in acetylcholine. This is likely to be related to the changes in sleep architecture mentioned above, particularly the reduction in REM sleep and increased wake time after sleep onset (Saper, Scammell, et al., 2005; Sarter & Bruno, 2004). More significant abnormalities have been observed with neurological diseases such as dementia (see section 2.2). Medications could also affect the stability of the systems (Saper, Scammell, et al., 2005). Wake-promoting drugs such as amphetamines and modafinil block the reuptake of the neurotransmitters which usually promote successful sleep; conversely, sedating drugs such as benzodiazepines and antihistamines supress the wake system by blocking the monoaminergic and GABA receptors (see section 3.1.1). The changing activity within the sleep promoting or wake promoting systems is modulated by the circadian drive for wake and the homeostatic pressure to sleep.

1.2 The Regulation of Sleep Timing and Changes with Ageing

The timing and maintenance of sleep and wake is regulated by two mechanisms: an endogenous *circadian wake drive* from the circadian pacemaker in the suprachiasmatic nuclei (SCN) of the hypothalamus, and a *homeostatic sleep drive*. In healthy young adults, these drives interact to create a consistent rhythm of consolidated sleep during the night and consolidated wake during the day (Czeisler & Buxton, 2011). With age the sleep/wake cycle becomes less pronounced. This is evident in more fragmented patterns of activity and inactivity and in changes to the timing of sleep and wake (Van Someren & Riemersma-Van Der Lek, 2007).

1.2.1 Circadian Rhythms

Most aspects of physiology and behaviour fluctuate across the 24-hour day/night cycle. This has been associated with a molecular-level circadian timing mechanism that is present in tissues and cells throughout the body. The four *clock genes* and oscillations in the concentrations of their proteins have been related to the length, timing, and consolidation of rest/activity periods and express circadian timing at a molecular level (Ko & Takahashi, 2006). The clock genes achieve this via a negative feedback loop involving the transcription of the genes and the translation into their proteins (Rosenwasser & Turek, 2011; Shearman et al., 2000).

Circadian rhythms all oscillate individually but are under the influence of an internal circadian pacemaker in the suprachiasmatic nuclei (SCN) of the anterior hypothalamus. The SCN consists of thousands of neurons and has the most robust and independent expression of circadian timing. These neurons have been shown to have a synchronised and self-sustaining rhythm of rest and activity, which is slightly longer than 24 hours (Ralph, Foster, Davis, & Menaker, 1990). The neurons within the SCN appear to be chemically split into two groups: those that are internally synchronised and generate a core signal of circadian timing which is imposed on the rest of the body, and those that are involved with the entrainment of the SCN to the environmental cycle of day and night via external stimuli (Saper, Scammell, et al., 2005).

Internally the SCN imposes its signal of circadian timing via neural outputs. Neurotransmitters synthesised in the SCN include vasopressin, vasoactive intestinal polypeptide, arginine, and gamma-aminobutyric acid. These have been highlighted as useful for regulating aspects such as blood pressure, temperature, appetite, and the expression of the sleep/wake cycle (Gooley & Saper, 2011). Vasopeptide neurons have been identified as for receiving information from the RHT and modulating the rhythmicity within the SCN, as well as being involved with the majority of outputs (Ingram et al., 1998; Wu & Swaab, 2007). The neural outputs from the SCN are mostly mediated via two areas within the hypothalamus: the supraventricular zone (SPZ), and the dorsomedial nucleus of the hypothalamus (DMH). The SPZ has been associated as key for temperature regulation and sleep/wake timing. It projects in a similar but amplified fashion as the SCN. The DMH has an intermediate role which involves integrating the circadian signal from the SCN (via the SPZ) along with other cues such as eating, socialising, and ambient temperature. The DMH then projects to other areas of the brain via the expression of multiple neuropeptides. This includes a projection directly to the VLPO and ARAS, aiding the expression of states of sleep and wake as described above (Gooley & Saper, 2011). This multifaceted process allows for the potential of flexibility within the timing of rest/activity rhythms, for example when travelling to a different time zone (Rosenwasser & Turek, 2011; Saper, Lu, Chou, & Gooley, 2005).

Neurons within the SCN are sensitive to environmental light via a monsynaptic projection from dedicated ganglion cells in the retina. These cells monitor light intensity and promote an increased firing rate in the SCN cells, via a pathway known as the retino-hypothalamic tract (RHT). This promotes increased rates of clock gene transcriptions within the feedback loop, helping to sustain the timing of the SCN pacemaker in synchrony to the cycle of light and darkness (Czeisler & Buxton, 2011; Duffy & Czeisler, 2009). Light also suppresses synthesis of the hormone melatonin, which is secreted by the pineal gland and feeds back to the SCN, aiding internal synchronisation and contributing to feeling more alert in the daytime, or when exposed to light during the night (Guardiola-Lemaitre & Quera-Salva, 2011).

The wavelength, intensity, and timing of light exposure influence its effects on the SCN. The retinal ganglion cells are particularly sensitive to blue light (460-685 nanometres; Jewett, Kronauer, & Czeisler, 1994). For healthy circadian entrainment, regular light exposure to light to an intensity of at least 500 lux across the day is deemed necessary (the intensity of light in normal rooms is between 100-300 lux, outdoor light on an overcast day has an intensity of around 10,000 lux, and at noon on a sunny day more like 100,000 lux; Sack et al., 2007; Wirz-Justice, Benedetti, & Terman, 2008). Light above about 100 lux is sufficient to suppress melatonin synthesis (Zeitzer, Dijk, Kronauer, Brown, & Czeisler, 2000). Light exposure can be manipulated to shift the timing of activity (phase) within the SCN, thereby having a subsequent effect on the phase of sleep and wake (Khalsa, Jewett, Cajochen, & Czeisler, 2003). This can be achieved with multiple or long doses of light of around 500 lux, otherwise shorter exposures at higher intensities can have a similar effect. For example, 2,500 lux for 1-2 hours, or 10,000 lux for 30 minutes (Wirz-Justice, et al., 2008). The effects of light are most pronounced during periods of reduced activity within the SCN, when the synthesis of melatonin is typically higher. Depending on the timing of exposure, bright light can delay (i.e. shift to a later time) or advance (i.e. shift to an earlier time) the phase of the SCN pacemaker by 1-3 hours (Jewett, et al., 1994). This can be useful for treating disorders of sleep timing (discussed in section 3.3.2).

There are other environmental stimuli that serve as non-photic circadian time cues to the SCN. These are arousal-related inputs including physical activity, social interactions, and eating. Non-photic time cues are considered to influence the activity within the SCN via a pathway from the lateral geniculate nucleus within the thalamus. Rodent studies have identified this area as necessary for successful circadian entrainment via physical activity (Wickland & Turek, 1994). Such activities also stimulate the production of serotonin in the brainstem which mediates the effect they have on the SCN (Rosenwasser & Turek, 2011; Webb, Antle, & Mistlberger, 2014). Physical activity could also influence the SCN indirectly via changes in temperature, melatonin, and metabolism as well as increase the need for physical restoration that can be achieved during sleep (Buxton, Lee, L'Hermite-Balériaux, Turek, & Van Cauter, 2003; Kondratova & Kondratov, 2012; Mrosovsky, 1996). Non-photic time cues are not as influential on SCN activity as light. Exercise has the potential to shift sleep timing by a matter of minutes

rather than hours (Czeisler & Buxton, 2011). As with light, the size of effect is related to the timing and intensity of the exercise. In opposition to light, exercise during the biological night promotes an increase in melatonin synthesis and decrease in neural activity within the SCN, promoting sleep (Frank et al., 2013; King, Oman, Brassington, Bliwise, & Haskell, 1997), see section 3.2.2 for more detail.

It is not possible to measure the activity of the SCN pacemaker directly, so other rhythms are measured as markers of its cycle, most commonly melatonin or core body temperature (Gander, 2003). When the circadian timing system is fully synchronised to the 24-hour day, melatonin synthesis begins to rise approximately two hours prior to bedtime (as recorded by the concentration levels of melatonin in plasma or saliva). Melatonin is considered to promote sleep via its inhibitory effects on the firing rate of the SCN as well as other parts of the brain and central nervous system. Melatonin synthesis peaks around six hours after sleep onset, then synthesis stops, as reflected by low levels of melatonin concentration in plasma and saliva during the day (Guardiola-Lemaitre & Quera-Salva, 2011). Conversely, body temperature increases across the day, peaking in the evening, and then beginning to decline prior to sleep onset, reaching its minimum approximately six hours after sleep onset (Czeisler & Buxton, 2011; Rajaratnam & Arendt, 2001). The subsequent rest/activity cycle is typically monitored by *actigraphy* which uses a small wrist-worn device to record the amount of movement in specified epochs, typically every minute. Patterns of activity can be monitored for days or weeks and interpreted with the aid of a sleep diary, to determine sleep and wake (Ancoli-Israel, Cole, et al., 2003; Littner et al., 2003). Validation studies have indicated reasonable agreement rates between actigraphy and polysomnography for estimating sleep timing and duration but less reliability for estimating sleep quality (Pollak, Tryon, Nagaraja, & Dzwonczyk, 2001; Sadeh, Sharkey, & Carskadon, 1994; Signal, Gale, & Gander, 2005).

Circadian variation is also evident in the propensity for sleep stages. This has been achieved via protocols of constant routine (using a rigorously controlled environment with the absence of external time cues) and recordings of core body temperature and polysomnography. Sleep efficiency, the production of REM, and Stage 2 sleep spindles can all be predicted based on the timing of circadian phase (Scheer & Shea, 2007). This means that, during appropriate circadian timing (i.e. biological night) sleep is typically less fragmented and more restorative. Similarly, waking performance in relation to reaction times has also

been demonstrated to fluctuate depending on circadian phase, with poorer performance occurring during biological night rather than day (X. Zhou et al., 2011). In these experiments the amount of time spent in prior wakefulness was also identified as having an independent influence on the quality of sleep as well as waking performance. This indicates that there is a secondary homeostatic process which helps to regulate the timing of sleep and wake.

1.2.2 The Homeostatic Sleep Process

The homeostatic sleep process reflects an increasing need for sleep that accumulates during wakefulness which subsequently dissipates during sleep (Achermann & Borbély, 2011). The increased need after a long bout of wakefulness acts as a pressure for sleep, restricting the amount of wake during the first hours of sleep. This has been exemplified in studies using polysomnography that show a positive correlation between time spent asleep with an increased percentage of wakefulness during the sleep period (Scheer & Shea, 2007). As the need for sleep declines across the sleep period the amount of wakefulness increases, until final wake up occurs.

No definitive physiological or chemical markers have been identified for the homeostatic sleep process. However, an indicator of sleep pressure is the amount of time it takes to fall asleep at different times of day or after previous sleep has been restricted, with shorter sleep onset latency indicating greater pressure (Taylor, Jenni, Acebo, & Carskadon, 2005). The amount and depth of SWS is also considered to be a reliable indicator, with deeper and longer SWS associated with greater sleep pressure. Slow wave sleep typically occurs during the first third of the night, acting to maintain a state of consolidated sleep during this time, whereas REM and Stage 2 activity are more prevalent as sleep time progresses (Achermann & Borbély, 2011; Scheer & Shea, 2007). Similarly, during wake an increase in EEG theta activity across the wake period has been observed and considered related to the drive for slow wave activity during subsequent sleep (Finelli, Baumann, Borbély, & Achermann, 2000).

As noted above, the neurotransmission of adenosine is associated with enabling sleep onset. Its concentration increases with prolonged wakefulness, promoting the release of inhibitory neurotransmitters which suppresses levels of arousal. Its transmission has also been associated with duration and depth of

SWS. The role adenosine plays regarding sleep homeostasis is also exemplified by the stimulating effects that caffeine, a potent adenosine receptor antagonist, has for staving off the pressure for sleep and subsequently disrupting SWS quality (Landolt, 2008).

Exercise promotes the homeostatic sleep pressure by increased need for physical restoration (Mrosovsky, 1996). This is reflected in studies using polysomnography showing that sleep onset is faster after exercise and sleep in general is deeper (an increase in SWS sleep and a decrease in Stage 1 sleep), compared to sleep without prior exercise (Driver & Taylor, 2000; Kubitz, Landers, Petruzzello, & Han, 1996; Youngstedt, O'Connor, & Dishman, 1997). These findings reinforce the positive impact that physical activity can have on sleep.

The homeostatic sleep process is considered to interact with the circadian drive for sleep, creating opposing periods of consolidated sleep and wake during biological night and day. This is known as the *opponent processes model* and is illustrated in *Figure 1.4* (Dijk & Edgar, 1999). This model ascertains that, as sleep pressure (or "load") increases across periods of wake, the circadian signal for alertness also increases in order to maintain a state of wakefulness, despite the heightened need for sleep. Sleep occurs after the circadian peak of this signal, when the pressure for sleep is greatest. During periods of sleep, circadian activity is reduced and the alerting signal low. As the pressure for sleep declines across the sleep period wakefulness becomes more likely. Once the lowest threshold of circadian activity has occurred, and the alerting signal begins to slowly rise again, final wake up is facilitated.

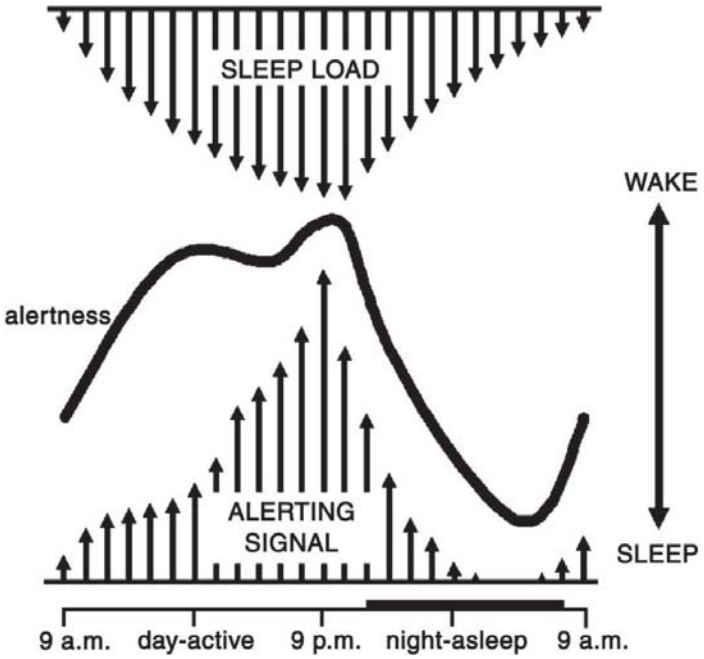


Figure 1.4. The opponent processes model of sleep/wake timing.

(Dijk & Edgar, 1999, p. 122).

1.2.3 Sleep Regulation and Ageing

With ageing, the circadian and homeostatic processes appear to become less synchronised, less robust, and reduced in amplitude compared to in younger age. This means that older adults are likely to sleep more than younger people across the average 24-hour day, but also have more fragmented sleep at night and increased likelihood of napping in the day compared to younger adults (Bliwise, 2011a). Older adults typically report going to sleep and waking up several hours earlier in the 24-hour day, and are more likely to describe themselves as "morning types" compared to younger adults (Czeisler et al., 1992; Duffy, Dijk, Klerman, & Czeisler, 1998; Paine, Gander, & Travier, 2006).

At a circadian level, changes to sleep timing have been related to changes in the transcription and translation of the clock genes within the SCN (Kolker, Vitaterna, Fruechte, Takahashi, & Turek, 2004; Kondratova & Kondratov, 2012), as well as anatomical deterioration within the area (Pandi-Perumal, Spence, & Sharma, 2010). Research using cell recordings from mice found that the SCN firing rate was less clearly distinguished between night and day within the older compared to younger mice (M. Nygård, Hill, Wikström, & Kristensson, 2005). Studies using autopsies of human brains have found that the reception and expression of particular chemicals within the SCN could also change with ageing. For example, one

study found that the vasopressin cells were particularly affected in older adults (Hofman & Swaab, 1994). Another found that melatonin receptors were more likely to diminish (Wu, Zhou, Van Heerikhuize, Jockers, & Swaab, 2007). Such changes equate to declining functionality in the area relating to neural projections from the SCN. A general reduction of the amplitude of circadian rhythms measured in temperature and melatonin profiles has been observed in older versus younger adults (Dijk, et al., 1999; Kondratova & Kondratov, 2012; Touitou & Haus, 2000; Welsh & Ptácek, 2010).

Age-related phase advances have been objectively measured in the timing of temperature rhythms of older adults which could account for the increased likelihood of earlier sleep timing compared to in younger age (Bliwise, 2011a; Bliwise, Ansari, Straight, & Parker, 2005). However, studies using constant routine protocols reveal that sleep timing of older people might actually be delayed relative to their different peaks in melatonin synthesis (Duffy et al., 2002). The findings concerning circadian amplitude and timing are generalised for older people. It should be noted that studies of the melatonin and temperature profiles of people of advanced age with good health status show that many had negligible differences in the expression of circadian markers compared to younger samples (Ferrari et al., 2008; T. H. Monk, 1995). Furthermore there does not appear to be any difference in the length of circadian timing, as indicated by temperature profiles (Czeisler et al., 1999). Together these findings indicate that changes to the homeostatic sleep profile and individuals' health and sociological status could be responsible for changes to sleep timing rather than age per se.

It has been proposed that the homeostatic pressure for sleep might dissipate more quickly for older compared to younger people, or older people could be less sensitive to the effects of sleep pressure. Faster dissipation of sleep pressure has been demonstrated with polysomnographic studies showing that older participants have less SWS at the beginning of the night, and an increased number of awakenings at night, as well as increased sleep time in day compared to younger participants (Bliwise, 2011a; Dijk, Duffy, & Czeisler, 2001). Studies examining the effects of sleep deprivation on performance have found that while older and younger participants both have poorer performance after sleep deprivation compared to baseline, older participants appear less sensitive to the effects of sleep pressure as exemplified by their performance, alertness and micro naps than younger participants (Duffy, Willson, Wang, & Czeisler,

2009; Frank, et al., 2013). They are also less likely to report subjective sleepiness at times of sleep deprivation (Silva, Wang, Ronda, Wyatt, & Duffy, 2010).

As well as the structural and physiological changes contributing to weakened sleep regulation, the timing and intensity of external time cues is also a key factor. Older adults typically have less time exposed to bright light compared to younger adults (Campbell, Kripke, Gillin, & Hrubovcak, 1988). This reduced exposure has an effect on how robust the SCN's entrainment is to the environmental light/dark cycle and the synthesis of melatonin (Duffy & Wright, 2005; Wirz-Justice, et al., 2008; Zeitzer, et al., 2000). Reduced light exposure with ageing has been related to lifestyle and health changes, with the lowest intensities of light recorded in PWD in nursing homes (Campbell, et al., 1988; Mishima, Okawa, Shimizu, & Hishikawa, 2001; Shochat, Martin, Marler, & Ancoli-Israel, 2000). The timing of light exposure may also change. Older people, practically morning-types, have more light exposure in the early morning than in the evening (Kawinska, Dumont, Selmaoui, Paquet, & Carrier, 2005; Staples, Archer, Arber, & Skene, 2009), which corresponds to the time when the SCN pacemaker cycle is phase advanced by light (see section 3.3.2 for more information). More napping in the evening has also been related to reduced light exposure and desynchronised sleep/wake timing with ageing (Yoon, Kripke, Youngstedt, & Elliott, 2003). Finally, aging of the visual system as well as deterioration within the SCN mean that older people can be less sensitive to the effects of light. Reduced size and functionality of the pupil, as well as changes to the shape, opacity, and density of the lens reduce the absorption and transmission of light, particularly light of shorter-wave lengths (Bradford, 2004; Herljevic, Middleton, Thapan, & Skene, 2005). Pathologies of the eye, such as cataracts or glaucoma are also more common with ageing and further contribute to weakened reception of light and make bright light more glaring and uncomfortable to look at (Dowling & Mastick, 2010; Herljevic, et al., 2005). So older people typically have reduced light exposure and the light that they are exposed to is often less intense and has a reduced effect on the circadian system compared to younger people. It is hypothesised that more environmental light is required to have an equivalent effect on the SCN pacemaker of older people, compared to those of younger age.

With ageing, there is often a reduction in the amount of physical activity, as well as of general activities of daily living, particularly among those with physical or mental health problems (Sirven &

Mancall, 2008). Changes to physical and social activity are likely to contribute to poorer sleep quality and regulation due to the mediating effects non-photic cues have on the circadian and homeostatic processes (Driver & Taylor, 2000; King, et al., 1997; Rosenwasser & Turek, 2011). Daytime and early evening napping is likely to increase (Yoon, et al., 2003). Planned afternoon naps versus unintended dozing in the evening affect subsequent night time sleep differently, due to the impact the nap has on homeostatic drive (Dijk & Edgar, 1999). Differences in napping behaviour have been related to older individuals either accepting or resisting the sociological concept of napping in older age (Venn & Arber, 2011). Degrees of acceptance are likely associated with the individual's preconceptions regarding napping and healthy, active ageing.

In summary, normal aging is associated with physiological changes, increased risk of pathologies, and changed patterns of behaviour and exposure to environmental time cues that affect the circadian system. These changes can lead to disruptions to sleep/wake timing. Trouble getting to sleep can be caused by trying to sleep at inappropriate times in the SCN pacemaker cycle or when the homeostatic sleep pressure is not sufficient to bring rapid sleep onset. Misalignment of the circadian and homeostatic processes can cause more shallow and fragmented sleep and difficulty in staying asleep. Early morning awakenings can occur due to an earlier phase position of the SCN pacemaker with respect to the day/night cycle, which can be exacerbated by early morning exposure to light. Finally daytime sleepiness and napping is more common with ageing, due to declining homeostatic sleep drive for sleep and poorer sleep at night (Carrier, 2010; Frank, et al., 2013; Pandi-Perumal, et al., 2010; Touitou & Haus, 2000).

1.3 Sleep Disorders and Ageing

Primary sleep disorders are those originating from a disturbance of the sleep/wake system itself, as opposed to being secondary to a condition such as depression or pain. Sleep disorders are further classified as *dyssomnias* (disorders of sleep timing or quality) or *parasomnias* (disorders related to unusual behaviours or experiences during sleep; American Academy of Sleep Medicine, 2005). Sleep apnoea, restless legs syndrome (RLS), periodic limb movements of sleep (PLMS), and insomnia are among the most common dyssomnias. These have all been found to become more prevalent with age and are summarised below. Parasomnias include activities such as sleep talking and walking, and night terrors.

Parasomnias are associated with malfunctions of the neurophysiological systems responsible for NREM and REM sleep. They are typically more common in childhood when sleep is maturing (Mahowald & Schenck, 2000). However, disorders such as REM behaviour disorder and nightmares can become more likely with age-related pathologies and as side effects of medications (Gagnon et al., 2002; M. Singer, Romero, Koenig, Förstl, & Brunner, 2005), see Chapters 2 and 3 for more detail.

1.3.1 Sleep Apnoea

Sleep apnoea is defined by repetitive cessations of breathing (apnoeas) during sleep. Obstructive sleep apnoea (OSA) is caused by a physical obstruction in the airway, whereas, central sleep apnoea (CSA) is related to the cessation of breathing due to impaired respiratory mechanisms in the central nervous system (Loewen, Poulin, & Hanly, 2010). Sleep apnoea is diagnosed as having five or more apnoeas per hour (with 5-15 episodes per hour classed as mild, 16-30 as moderate, and more than 30 as severe; American Academy of Sleep Medicine, 2005; Ruehland et al., 2009). The sleep apnoea syndrome includes these events along with daytime sleepiness which is associated with fragmented sleep and increased hypoxia.

A NZ study using overnight monitoring estimates that sleep apnoea is prevalent in 12.4% of Males and 3.4% of females aged 30-59 years (with approximatly a third of these estimated to have the full syndrome; Mihaere et al., 2009). Māori men were identified as having increased likelihood of OSA, suggesting that there may be factors associated with increased likelihood of sleep apnoea (e.g., factors related to socioeconomic status, and body mass) that make it of particular concern for Māori. Mihaere et al. (2009) also found that increased age was related to increased likelihood of reporting symptoms of sleep apnoea. A large community-based study in the United States of America (USA), using questionnaires as well as polysomnographic recordings with 40-98 year olds, has also found age-dependent changes with sleep apnoea (Young et al., 2002). Young et al. found that 21% of 70-79 year olds had moderate-severe sleep apnoea compared to 10% of 39-49 year olds. Subsequent research indicated that much of the age-related increase in apnoea is related to central rather than obstructive events, and that older people appear less likely to report any associated daytime sleepiness (Bixler, Vgontzas, Ten Have, Tyson, & Kales, 1998).

This may mean that older people's sleep is less affected for an equivalent level of respiratory disturbance, and/or they may be less likely to be aware of or report daytime sleepiness compared to younger people.

The increased prevalence of sleep apnoea with age is thought to be related to physiological changes to the airway itself (e.g., weaker upper airway musculature), as well as a less stable respiratory response to changes in breathing (Loewen, et al., 2010). Having sleep apnoea increases the likelihood of other comorbidities, including cardiovascular disease and depression, and can also have a significant effect on individuals' daytime functioning including alertness, and cognition. This has implications for older people regarding risk of falls and road traffic accidents (Gander, Marshall, Harris, & Reid, 2005b; Stone, Ensrud, & Ancoli-Israel, 2008). Patients who are compliant with treatments for sleep apnoea (e.g., via continuous positive airway pressure; C. Sullivan, Issa, Berthon-Jones, & Eves, 1981), or non-invasive ventilation (Morgenthaler, Gay, Gordon, & Brown, 2007) typically have a reduction in the number of respiratory events as well as improved waking symptoms (Loewen, et al., 2010).

1.3.2 Restless Leg Syndrome and Periodic Limb Movements of Sleep

Restless legs syndrome is characterised by discomfort in the legs along with an irresistible urge to move them. Symptoms increase in the evening, prior to sleep. Decreased activity typically exacerbates the symptoms whereas moving the legs improves them. Therefore RLS causes difficulty in relaxing and getting to sleep (Bliwise, 2006). Conversely PLMS are characterised by involuntary jerks which occur *during* sleep (NREM), causing brief arousals or awakenings.

In an international multi-city study using telephone interviews, Ohayon and Roth (2002) found that the prevalence of self-reported symptoms of RLS in the general population (aged 15-100 years) is 5.5%. Symptoms of RLS significantly increased with age (8-9% in people aged over 60 years). In a similarly designed study, Phillips et al. (2000), found that Americans of advanced age had even higher rates of self-reported RLS (19%). Self-reported symptoms of PLMS do not appear to increase with age (Ohayon & Roth, 2002). This may be because of the lack of awareness of symptoms occurring during sleep. The estimated prevalence of PLMS is 3.9% using self-reports (Ohayon & Roth, 2002). Previous studies using polysomnographic recordings reveal that the prevalence (as defined by >5 limb movements per hour

during sleep) could be between 45% and 85% for people aged over 60 years (Ancoli-Israel et al., 1991; Youngstedt, Kripke, Klauber, Sepulveda, & Mason, 1998) compared to 11% of the general population (Bixler et al., 1982).

The reasons why RLS and PLMS appear to increase in age are unclear, but may involve age-related comorbidities and behaviours more than the ageing process per se. Restless legs syndrome has been associated with iron deficiency, cardiovascular disease, diabetes, mental health conditions, arthritis, as well as reduced exercise and the side effects of medications, all of which increase in likelihood with age (Bliwise, 2006; O'Keeffe, Gavin, & Lavan, 1994; Ohayon & Roth, 2002; Phillips, Hening, Britz, & Mannino, 2006; Phillips, et al., 2000). The presence of RLS and/or PLMS contributes to older peoples' sleep disturbances, including trouble getting to sleep, waking at night and daytime sleepiness (Bliwise, 2006). The symptoms of RLS and PLMS can be alleviated through pharmacological treatments (e.g., dopaminergic agonists; Hening, Allen, Earley, Picchietti, & Silber, 2004). However, the importance of healthcare professionals addressing any underlying conditions (such as iron deficiency) prior to medicating is stressed (Bliwise, 2006).

1.3.3 Insomnia / "Poor Sleep"

Insomnia is defined as having difficulty initiating and maintaining sleep despite having an adequate sleep opportunity. *Primary insomnia* is sleeplessness related to psychological maladaptive behaviour associated with sleeping, misperceptions associated with sleep, or idiopathic insomnia which originates from childhood. *Secondary insomnia* is defined as when the sleeplessness is attributed to another medical or psychological condition (e.g., depression, pain, or pharmacology). Insomnia is often associated with environmental factors and the behavioural regime surrounding sleep. This is defined as insomnia related to poor "sleep hygiene" (American Academy of Sleep Medicine, 2005). Polysomnographically, insomnia can be defined as having a sleep onset latency of 30 minutes or more, spending 30 minutes or more awake after sleep onset, having a sleep efficiency of less than 85%, and/or having a total sleep duration of less than 6 hours, occurring more than three nights per week for six months or more. However, insomnia is frequently diagnosed by the patient's subjective sleep complaints which often appear more severe than

these quantitative changes alone (Lee-Chiong & Harrington, 2008; Lichstein, Durrence, Taylor, Bush, & Riedel, 2003; Ruiter, Vander Wal, & Lichstein, 2010).

The Pittsburgh Sleep Quality Index (PSQI;Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) is often used in both clinical and research settings to measure subjective sleep quantity and quality over the previous month. This brief standardised questionnaire gives an indication of self-satisfaction with sleep (on a scale of 0, "no sleep difficulty", to 21, "severe sleep difficulties") and is used to distinguish "good" and "poor sleepers" using a threshold score of 5 (Buysse, et al., 1989; Tsai, Wong, & Ku, 2008). This questionnaire is useful compared to using an insomnia questionnaire alone (e.g., the Insomia Severity Index; Bastien, Vallières, & Morin, 2001), as it highlights if there are any other types of sleep disturbance (e.g., disturbed sleep timing, breathing or restlessness) as well as perceived sleep quality and insomnia (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002).

It is difficult to give reliable estimates of the prevalence of insomnia due to the different ways in which clinicians and researchers define it. Clinically significant insomnia symptoms had been recorded in 4-6% of young-middle aged adults, and up to 20% of adults aged 65 years or more (Arber, Bote, & Meadows, 2009; Lichstein, Durrence, Riedel, Taylor, & Bush, 2004; Lichstein, Taylor, McCrae, & Ruiter, 2011; Ohayon, 2002). Prevalence estimates for general insomnia complaints (difficulty falling asleep, staying asleep, early morning awakenings, and unsatisfactory sleep) and reports of "poor sleep" range between 9-27% for young-middle aged adults, and 30-60% among older adults (Ohayon, 2002; Paine, Gander, Harris, & Reid, 2005; Venn & Arber, 2009). Research using the PSQI within samples of healthy participants corroborates a negative relationship between age and subjective sleep reports. Healthy adults of advanced age (aged > 80 years) have higher mean and more variable PSQI scores (mean = 4.8, standard deviation (SD) = 3.0) compared to younger adults (aged 20-30 years, mean score = 2.5, SD = 1.5; Buysse et al., 1991). Buysse et al. also found that 31.9% of adults aged over 80 years were defined as poor sleepers (indicated by scoring >5 on the PSQI) compared to 2.9% of adults aged 20-30 years ($p=0.0001$).

Although the prevalence of insomnia/poor sleep increases with age, age alone is not considered the issue. Rather, the comorbidities which are more common with ageing increase the likelihood of

secondary insomnia. Therefore, healthy older adults are not necessarily at any greater risk than younger adults (Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001). Once symptoms of insomnia develop in older age, they are often exacerbated by poor sleep habits. For example, spending extended time awake in bed, reduced daily routines, or light exposure during the night (Ruiter, Vander Wal, et al., 2010).

Previous research indicates that prevalence of insomnia/poor sleep varies by sex, socioeconomic positon and ethnicity (Arber, et al., 2009; Bixler, Kales, Soldatos, Kales, & Healey, 1979; Ohayon, 2002). For younger and older age groups, women have increased likelihood of reporting symptoms of insomnia compared to men (1.4-1.7 times more likely, respectively), and are twice as likely to have a diagnosis of insomnia compared to men (Ohayon, 2002; Ruiter, Vander Wal, et al., 2010). This has been associated with the hormonal effects of menopause as well as women being more likely to have misperceptions of their sleep quality (Vitiello, Larsen, & Moe, 2004). More recent research also suggests that socioeconomic factors (e.g., employment and educational status) are also major contributors to women being more likely to report sleep problems (Arber, et al., 2009).American-based research indicates that African-Americans report less symptoms of insomnia than Caucasian Americans (Durrence & Lichstein, 2006; Grandner et al., 2010; Ruiter, DeCoster, Jacobs, & Lichstein, 2010).

New Zealand-based studies of 20-59 year olds consistently find disparities in sleep health of Māori compared to non-Māori individuals (Paine & Gander, 2013). One study using a self-completed postal questionnaire amongst 2,100 Māori and 1,900 non-Māori found that 33.0% of Māori reported symptoms of insomnia compared to 26.4% of non-Māori (Paine, et al., 2005). In this, and similar studies, socioeconomic position was measured by the New Zealand Deprivation index (NZDep), which categorises people based on the home address on a scale of 1 (least deprived) to 10 (most deprived; Salmond, Crampton, & Atkinson, 2007). Increased deprivation is consistently shown to be an independent predictor of reporting insomnia, and Māori are typically over represented in the most deprived deciles (Ministry of Health, 2012a; Paine, et al., 2005).The study did not include older New Zealanders and so it is not known whether these ethnic and socioeconomic disparities continue into older age.

Common diseases with ageing such as arthritis, heart disease, diabetes, and respiratory disease have been associated with increased likelihood of insomnia. The presence of such conditions is likely to place additional stress and discomfort on the individual as well as promote the use of medications which might also have negative side effects on sleep (Wolkove, Elkholy, Baltzan, & Palayew, 2007; see section 3.1).

Polypharmacy (i.e. taking ≥ 5 medications at once) and drug misuse is also an area of concern in relation to the sleep of older people (Culberson & Ziska, 2008; Jyrkkä, Enlund, Korhonen, Sulkava, & Hartikainen, 2009). While some older people are legitimately required to take several medications, careful consideration of the side effects is recommended, as well as the time of day they are being taken, to avoid unnecessary sedation during the daytime and arousal at night. Excessive caffeine and alcohol consumption have also been associated with insomnia, and withdrawal from these as well as other recreational substances also needs to be considered as potential contributors to poor sleep (Kim, Tofade, & Peckman, 2009).

Life events such as retirement, bereavement, and loneliness have also been associated with poor sleep hygiene and depression among older people. In turn, depression and anxiety are considered interrelated with symptoms of insomnia throughout the lifespan (Ruiter, Vander Wal, et al., 2010). Jimenez, Perez, Prieto and Martinez-Osorio (1989) conducted an exploratory study to understand the factors associated with depression and anxiety amongst 207 adults aged over 65 years. They used the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983), which is a brief scale to assess likelihood of these affective disorders (on a scale from 0-21, with scores > 7 representing increased affect), as well as a self-completed behavioural questionnaire including sleep duration and whether they wake at night. They found that older people sleeping fewer than seven hours at night had significantly higher scores for depression (mean = 7.9, SD = 4.8), compared to those sleeping more than seven hours (mean = 6.4, SD = 3.8). Furthermore, those who woke at night had significantly higher anxiety scores (mean = 8.3, SD = 4.4), compared to those who did not wake (mean = 6.5, SD = 4.1).

The health and lifestyle factors described above can be applied to Speilman's 3P model for insomnia which ascertains that there are *predisposing*, *precipitating* and *perpetuating* factors associated with the

condition (Spielman, Caruso, & Glovinsky, 1987). For older people, predisposing factors include the changes to sleep architecture and regulation of sleep noted in sections 1.1 and 1.2; precipitating factors including life events and comorbid illnesses occurring with ageing; and perpetuating factors include changes in sleep hygiene behaviours to compensate for the lack of sleep, such as activities and light exposure at night or daytime napping.

Insomnia has an impact on daytime sleepiness and poorer perceived functionality (Hauri, 1997; Lichstein, Heith Durrence, Riedel, & Bayen, 2001). These symptoms contribute to the risk of motor vehicle accidents and falls as well as cognitive impairment (Gander, et al., 2005b; Ohayon & Vecchierini, 2002; Stone, et al., 2008), which are areas of particular concern for older people. The relationship between insomnia and mental health is considered bi-directional and so insomniacs are often in a vicious circle of poor sleep health and depression and/or anxiety (Choi & Irwin, 2008; Ford & Kamerow, 1989). Finally, chronic sleep restriction has been associated with premature death (Dew et al., 2003; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002).

There is no single strategy for treating insomnia due to its diversity of causes. Interviews with older British adults revealed a tendency to resist medicating sleep problems. This was due to negative connotations participants associated with sleeping pills including potential side effects, as well as the normalisation of sleep problems (Venn & Arber, 2012). Self-management of sleep hygiene appears preferable, and strategies are likely to vary by gender (Venn, Meadows, & Arber, 2013). Trials of non-pharmacological strategies (including cognitive behavioural therapy) have identified that they are effective in addressing the perpetuating factors (P. Montgomery & Dennis, 2003; Morin, Colecchi, Stone, Sood, & Brink, 1999). Despite these factors, many clinicians prescribe hypnotic medications for their sedating influence (see Chapter 3; Kim, et al., 2009; Lee-Chiong & Harrington, 2008; Reynolds, Kupfer, Hoch, & Sewitch, 1985).

1.3.4 Sleep of Older Carers

Life expectancy is increasing at an international level (Oeppen & Vaupel, 2002), therefore the number of older people in poor as well as good health is rising. In NZ it is predicted that between 2011

and 2026, the population aged over 65 years will increase by 121.8% for Māori and 60.3% for non-Māori (Ministry of Health, 2012b). Nationally as well as internationally, there are limited services and support for everyone requiring aged care, as well as an increased need and desire for older people with disabilities to be cared for within their own homes (Jorgensen, Parsons, Jacobs, & Arksey, 2010; Kiata, Kerse, & Dixon, 2005). This indicates that there is an increase in the amount of *informal caring* occurring. Informal care is care assistance provided by family or friends to someone with whom they often live. Informal carers are typically spouses or adult children. They are not necessarily trained caregivers, nor are they paid for their work. Informal carers often have to sacrifice employment or social commitments and undergo significant lifestyle changes in order to provide care for their family member (Brunton, Fouché, & Jordan, 2007).

Previous research has found that many informal carers report financial strain and isolation (Jorgensen, et al., 2010). Unfortunately, not all carers are provided with support such as additional care assistance, social workers, or respite. These factors contribute to the objective burden of caregiving, i.e. having to physically and mentally respond to the care recipient and having negative caregiving experiences (Vitaliano, Russo, Young, Becker, & Maiuro, 1991). Interviews with 300 informal carers (the majority of whom were aged 30-69 years) in NZ, revealed that just 4% of those providing care for an older person reported that they were happy with the amount of support they received (Jorgensen, et al., 2010). Overarching these stressors is the emotional impact of providing personal care for a family member with a physical or mental disability (Arber & Venn, 2011).

Changes to carers' subjective quality of life, as well as their objective environment and time constraints, can have an impact on degrees of positive and negative affect experienced (Lawton, Winter, Kleban, & Ruckdeschel, 1999). Quality of life for older people is considered to be affected by four domains outlined by Lawton (1991). These are behavioural competence: " the social normative evaluation of the person's functioning in their health, cognition, time use, and social dimensions" (p.8); perceived quality of life: "the persons subjective evaluation of function in any of the behavioural competence dimensions" (p.10); psychological wellbeing: "the weighted evaluated level of the person's competence and perceived quality in all domains of contemporary life" (p.11); and the objective environment (and

whether that environment helps or hinders their behaviours and influences over quality of life). When one or more of these domains is out of balance, quality of life is affected. This could be particularly likely for carers and care recipients.

Previous research consistently finds that informal caring is associated with increased likelihood of stress, anxiety, and depression and has an impact on quality of life (Goodhead & McDonald, 2007; Jorgensen, et al., 2010; Vitaliano, Zhang, & Scanlan, 2003). In NZ, Jorgensen et al. (2010) found that symptoms of depression and stress were common across all age groups of carers. For older carers, depression was of particular concern, especially for female carers. Informal carers are required to physically "work" for potentially long hours. Caring for someone with a severe disability or neurological disorder could mean providing care assistance during the night. Although research has shown that older carers lead a routine lifestyle (T. H. Monk et al., 2006), some are required to be available to work 24 hours a day, seven days a week. Therefore, informal caring is comparable to the lifestyle of a first time parent or shift-worker. Many informal carers are also older people Informal carers, so may also have to cope with age-related changes to their own sleep and comorbidities, which could interfere with their ability to adapt to sleep challenges associated with caregiving.

In a large survey of carers in the United Kingdom (UK), Maher and Green (2002) found that the amount of time spent providing care was significantly related to self-reported sleep disturbances. Almost half (47%) of those providing care for 50 hours or more per week reported disturbed sleep, compared to 24% of those providing care for 20-49 hours, and 7% of those providing less than 20 hours care per week. Studies using polysomnography have found that the sleep architecture of carers differs from non-carers in that they have significantly reduced sleep duration at night, reduced SWS and REM sleep, as well as increased time spent in Stage 1 sleep and wakefulness compared to older non-carers (Fonareva, Amen, Zajdel, Ellingson, & Oken, 2011; Rowe, McCrae, Campbell, Benito, & Cheng, 2008).

Recent research from the UK has highlighted the reasons why sleep is disrupted for older informal carers (Arber & Venn, 2011). Interviews with male and female carers helped to identify a six-fold typology which includes providing physical care at night; the anticipation of being required to provide care at night; monitoring and checking on the care recipient during the night; having sleep disturbed by the activities of

the care recipient; providing emotional support to the care recipient or their own worries about their situation; and the impact of caring after their role finishes (the "legacy of caregiving", p.158). Those caring for someone with a more severe disability encountered most of these types of sleep disruption. Spousal carers appeared to have particularly affected sleep. This could be related to the intense nature of spousal caring as well as increased likelihood of these couples sharing a bed or bedroom, or changes to the couple's routines around bedtime and sleep (Crossley, 2004, as cited in Kotronoulas, Wengström, & Kearney, 2013, p. 580). Female carers also appeared to be more likely to report more sleep disturbances at night compared to males. In their interviews, female carers were more likely to report that their sleep was affected by the emotional aspects of caring and worrying at night (Arber & Venn, 2011). This is important as females appear to be more likely to take on the role of informal carer compared to males (Brunton, et al., 2007; Kotronoulas, et al., 2013). As noted above, women are also more likely to report symptoms of insomnia (Ohayon, 2002; Ruiter, Vander Wal, et al., 2010).

Sleep of informal carers is important for several reasons. As noted above, sleep disturbances are associated with daytime sleepiness and negatively affect cognitive performance, as well as physical and mental health (Foley, Ancoli-Israel, Britz, & Walsh, 2004; Hauri, 1997; Lichstein, et al., 2001; K. Reid, Martinovich, Finkel, Harter, & Zee, 2006). Sleep deprivation or disruption for these individuals could have an exacerbating effect on the levels of burden and depression they are already experiencing, as well as potentially jeopardizing their health to the point where they are no longer able to provide sufficient care for their family member. Informal care situations can last many years depending on the trajectory of the care recipient's disease. Carers' subjective and objective sleep can be improved during periods of respite care. However, their sleep returns to being just as disrupted once their relative returns home to their care (D. Lee, Morgan, & Lindesay, 2007). Previous research highlights that sleep disturbances are one of the key reasons given by carers for their relative moving into a care facility (Kesselring et al., 2001; Pollak & Perlick, 1991). Research on the sleep of carers in NZ is lacking, so is a key consideration when addressing the sleep of older people living in the community.

1.4 Rationale for Studies 1 and 2

The sleep health of the younger, and particularly middle aged (20-59 years) members of the New Zealand population is well documented (e.g., Borlase, Gander, & Gibson, 2013; Gander, 2003; Gander, Marshall, Harris, & Reid, 2005a; Gander, et al., 2005b; Gibson, Elder, & Gander, 2012; Gibson, Gander, & Elder, 2012; Mihaere, et al., 2009; Paine & Gander, 2013; Paine, Gander, Harris, & Reid, 2004; Paine, et al., 2005; Paine, et al., 2006). However, to date, no research appears to have been designed or analysed to address the sleep of older New Zealanders as a primary outcome. Although the main focus of this thesis is a trial of therapies for improving the sleep of older people with dementia and their carer's, it was considered important to first address the question: "How are older New Zealander's sleeping?"

At the time of the thesis, two longitudinal studies were being conducted with older New Zealanders. Te Puawaitanga o Nga Tapuwae Kia Ora Tonu, Life and Living in Advanced Age: A Cohort Study in NZ (LiLACS NZ) has been using face-to-face surveys to collect information to evaluate factors that contribute to successful ageing among Māori and non-Māori of advanced age (Hayman et al., 2012). This research was led by Professor N. Kerse in the Faculty of Medical and Health Sciences at Auckland University. The LiLACS NZ cohort began in 2010 with 421 Māori (aged 79-90 years) and 516 non-Māori (aged 84-86 years). A large battery of standardised questionnaires was included in the LiLACS NZ survey, including general questions about current sleep problems (including the type of problem), as well as past sleeping problems, and caregiving status. Through collaboration with Professor Kerse and the LiLACS NZ team, I was able to analyse the first LiLACS NZ survey to provide the first description of the sleep health of New Zealanders of advanced age. This constitutes Study 1 and is presented in section 1.5 as a paper which has been submitted to the Australasian Journal of Ageing for consideration for publication.

The second cohort available was the Health Work and Retirement (HWR) study. Surveys have been used to collect information on the physical and mental health of older workers and retirees, with the global aim of finding what factors are associated with New Zealanders living a more healthy, wealthy, and independent retirement. This research was led by Professor F. Alpass in the School of Psychology at Massey University. This cohort began in 2006 with 6,662 adults aged 55-70 years. The second wave of

data collection took place in 2008 with 2,473 participants. This wave included some standardised sleep questions from the Short-Form Health Survey (Ware, Kosinski, & Dewey, 2000) as well as caregiving status. This was of particular interest for the current project as the LiLACS NZ dataset only had a small sample of carers and so the relationship between caregiving and sleep could not be reliably assessed. Collaboration with Professor Alpass and the HWR team allowed me to analyse this data and contribute to the description of the sleep health of older New Zealanders (by including people aged < 80 years, the "young old"), as well as assess the impact of caregiving on participants reports of feeling tired, worn out, or dissatisfied with their sleep. This constitutes Study 2 and is presented in section 1.6 as a paper which has been accepted for publication in the Australasian Journal of Ageing by John Wiley & Sons, Inc.

1.5 Study 1: Sleep of Māori and non-Māori of Advanced Age

1.5.1 Abstract

Objectives: To estimate prevalence and identify predictors and outcomes of reporting sleep problems in Māori and non-Māori of advanced age. Methods: Participants were 251 Māori and 398 non-Māori adults (79-90 years) from Life and Living in Advanced Age: A Cohort Study in New Zealand. Multiple logistic regression was used to identify predictors of reporting a current sleep problem, and to investigate relationships between current or past sleep problems and indices of poorer physical and mental health. Results: 26.3% of Māori and 31.7% of non-Māori reported a current sleep problem (odds ratio (OR) = 0.52, 95% CI=0.30-0.90). Reporting a past sleep problem was associated with reporting a current problem (OR=2.67, 95% CI=1.25-5.72). Sleep problems were significantly related to poorer physical and mental health, and falling. Conclusions: Early recognition and management of sleep problems could reduce risk of falls and improve symptoms of mood disorders and cognitive impairment.

1.5.2 Introduction

Getting older is generally associated with more sleep disturbances, with 20%-70% of 50-80 year olds reporting a sleep problem (Foley, et al., 2004; Foley et al., 1995; Middelkoop, Smilde-van Den Doel, Neven, Kamphuisen, & Springer, 1996; K. Reid, et al., 2006). Older women (aged ≥ 50 years) are more likely to report disturbed sleep with insomnia-type symptoms, whereas older men typically report more sleep-disordered breathing and daytime sleepiness (Foley, et al., 1995; Middelkoop, et al., 1996; Phillips & Ancoli-Israel, 2001). Studies that have measured sleep objectively corroborate these reports, showing that sleep quality and duration generally decrease with age (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). However, limited research has focused on the prevalence of sleep problems among people of advanced age (>80 years). A recent USA-based study found that successful ageing might be associated with a *reduction* in reporting sleep problems (Grandner et al., 2012).

The likelihood of having health problems associated with sleep disturbances increases with age (House et al., 1994). These include depression, pain, respiratory and cardio-vascular diseases, cognitive

impairment and dementia (Foley, et al., 1995; Phillips & Ancoli-Israel, 2001). Sleep problems are negatively associated with self-rated physical and mental wellbeing, mood, and quality of life (Foley, et al., 2004; Paine, et al., 2005; K. Reid, et al., 2006). Poor sleep degrades daytime alertness and performance, thereby increasing the risk of incidents such as road traffic accidents and falls (Gander, et al., 2005b; Stone, et al., 2008). When older people are also required to care for someone with an illness, sleep problems are likely to be exacerbated (Arber & Venn, 2011; Gibson, Gander, & Jones, 2014). The proportion of older people in the population is increasing and it is becoming more common for people to provide care to their family members at home (Cornwall & Davies, 2004), making the sleep of older carers of particular interest. People of advanced age are effectively 'survivors' - they are likely to have had exceptionally good health - and the predictors and consequences of sleep problems may not be the same as for younger populations.

A partnership programme of epidemiological research to understand disparities in sleep health between Māori (the indigenous population of New Zealand, (NZ)) and non-Māori adults (aged 20-59 years) has revealed significant differences in the prevalence of common sleep problems and disorders (Paine & Gander, 2013). For example, a national survey revealed that 28.6% of Māori and 24.6% of non-Māori reported having a sleep problem lasting at least 6 months ($p=0.033$) (Paine, et al., 2005). There are also consistent relationships between poorer sleep health and socioeconomic position and age, as well as differences by sex (Paine & Gander, 2013).

The current study used data from the inception interviews (Wave 1) of Te Puāwaitanga o Ngā Tapuwae Kia Ora Tonu. Life and Living in Advanced Age: A Cohort Study in NZ ("LiLACS NZ"). This is the first study of its kind, using face-to-face surveys to collect information on factors that contribute to successful ageing for Māori and non-Māori of advanced age (Hayman, et al., 2012). Sleep-related information in this dataset provides a unique opportunity to describe sleep health in advanced age, and to contribute to better health services to recognise and manage geriatric sleep problems.

The aims of these analyses were to estimate the prevalence of self-reported sleep problems among Māori and non-Māori of advanced age; to identify the demographic and health-related predictors of

reporting sleep problems; and to determine whether self-reported current or past sleep problems increased the likelihood of reporting other adverse health-related outcomes.

1.5.3 Methods

This project was approved by the Northern X Regional Ethics Committee (NXT 09/09/088). The LiLACS NZ Wave 1 survey was completed with 421 Māori (aged 79-90 years) and 516 non-Māori (aged 84-86 years). The Māori cohort included participants who identified themselves as Māori, either alone or as one of multiple ethnicities. They had a broader age range than the non-Māori cohort to account for known differences in longevity between the two populations (Dyall et al., 2013; Statistics New Zealand). The participants were living in the Bay of Plenty and Lakes District Health Board areas of NZ in 2010. All Māori born between January 1st 1920 and December 31st 1930, and all non-Māori born between January 1st and December 31st 1925, were identified from the electoral roll, primary care databases, word of mouth, Māori tribal networks, and through local publicity. Someone known to them invited all participants. Those who gave informed consent completed the face-to-face survey in their own home, with a trained interviewer, using standardised techniques. An overall response rate of 57% was achieved (Dyall, et al., 2013). The populations recruited approximated the age and gender distribution of the underlying population, except that women were over-represented.

The sample used for the present analyses included all participants who completed the question: "Do you have trouble with your sleeping (on at least 3 nights per week) such that it interferes with your activities the following day (e.g., unrefreshed in the morning, fatigue, poor concentration, or irritability)?" Participants who answered *yes* were asked to indicate the types of problems they were experiencing from a list of eight symptoms (*Figure 1.5*), and were also asked: "How much trouble did you have with sleeping when you were young?" Answers were dichotomised into *yes* (*a little, some,* or *a lot*) versus *no* (*none at all*).

Study procedures allowed for participants to complete a shortened core survey that did not include the sleep questions. For example, if they were considered incapable of answering the interview questions for themselves, or in residential care (Dyall et al., 2014). Of the 937 participants in Wave 1, 261 completed the core survey only and 5 did not complete either. There were also 22 instances of participants

who completed the full survey but did not answer the sleep questions. These groups were combined as 288 participants who did not answer the sleep questions. The analyses presented here focus on the remaining 251 Māori and 398 non-Māori (the "sleep sample").

Demographic measures included: ethnicity, sex, age, the NZ Deprivation index 2006 (NZDep 06, an area-based measure of socioeconomic position; Salmond, et al., 2007), and caregiving defined by the question: "How often do you currently provide care or assistance for other people?" Carers included those responding *occasionally, less than once a week, once a week, two to five times weekly,* or *daily (six to seven times weekly)*. Non-carers were defined by answering *never*. The physical and mental health variables considered for multivariate analyses can be found in Table 1.2. These include standardised scales as well as: a global score from a 5-point likert scale rating coping in different situations (*times of loss, financial hardship, on-going health problems, times of trouble for family and friends*, and *overall)*; a 5-point liker scale rating the experience of ageing ("On the whole has growing older been a positive or negative experience for you?"); and single item questions to define those who had fallen, were a current or past smoker, or drinker (see Table 1.2 for details).

Analyses.

The sleep sample and the participants who did not answer the sleep questions were compared by ethnicity, sex, age and NZdep 06 (Salmond, et al., 2007) using sequential logistic regression modelling with 98.3% of the total observations (n=922). Chi square analysis was used to describe the types of sleep problems reported between Māori and non-Māori. Uni-variate associations between reporting a current sleep problem and demographic and health-related variables were undertaken to identify factors related to sleep problems at the $p<0.10$ level. These were included in the multiple logistic regression models along with variables selected based on *a priori* evidence, including all of the demographic variables. Dichotomised variables were defined either by pre-determined standardised cut off scores, or by scoring within the 25th percentile of the particular scale (Table 1.2)

Table 1.2

Predictors and Outcome Variables Considered for Logistic Regression Models

Variables	Scale (reference)	Description as an independent predictor	Description as dichotomous outcome
Current sleep problem		Yes vs. no (ref.)	Yes vs. no (ref.)
Sleep problem when young		A little, some, or a lot vs. none at all (ref.)	A little, some, or a lot vs. none at all (ref.)
Demographics			
Ethnicity		Māori vs. non-Māori (ref.)	-
Sex		Female vs. Male (ref.)	-
Age		One year increments (79-90 years)	-
Socioeconomic position	NZdep 06(Salmond, et al., 2007)	1 (least deprived) to 10 (most deprived)	-
Caregiving status		Carer vs. non-carer (ref.)	-
Mental health	Short-Form Health Survey (SF-12, Fleishman, Selim, & Kazis, 2010)	Range 14.9-79.3	Low ≤25% QI vs. mod-high >25% QI (ref.)
Cognition	Mini Mental Status Exam (MMSE, Folstein, Folstein, & McHugh, 1975);	Range 0-30	Low <25 vs. mod-high ≥25 (ref.)
Depression rating	Geriatric Depression Scale(GDS-15, Sheikh & Yesavage, 1986)	Range 1-14	Mod-high risk >8 vs. low risk ≤8 (ref.)
Depression diagnosis	General Practitioner record review	Depressed vs. not depressed (ref.)	Depressed vs. not depressed (ref.)
Perceived coping		Range 10-25	Reduced ≤25% QI vs. Mod-high>25% QI (ref.)
Perceived control	Pearlin Mastery Scale (PMS, Pearlin & Schooler, 1978)	Range 9-35	Reduced ≤25% QI vs. mod-high>25% QI (ref.)
Ageing experience†		Negative-very negative vs. neutral-very positive (ref.)	Negative-very negative vs. neutral-very positive (ref.)
Physical health	SF-12 (Fleishman, et al., 2010)	Range 6.0-65.8	Reduced ≤25% QI vs. mod-high>25% QI (ref.)
Pain interference	SF-12 (Fleishman, et al., 2010)	Range 0-100	Increased ≤25% QI vs. mod-low>25% QI (ref.)
Physical activity†	Physical Activity Scale for the Elderly (PASE, Washburn, Smith, Jette, & Janney, 1993);	Range 0-607.9	Reduced ≤25% QI vs. mod-high >25% QI (ref.)
Falls in past 12 months		0- ≥4	Fallen ≥1 times vs. not fallen (ref.)
Alcoholic drinking		Monthly or less - ≥4 times per week) vs. never (ref.)	-
Smoking status†		Current or past smoker vs. never smoked (ref.)	-

†Variable not used in final logistic regression models due to non-significant relationship to the outcome variable at the univariate level. Ref. (reference group), - (not applicable)

Additional logistic regression analyses were conducted to identify whether reporting a sleep problem (current or when younger) was significantly associated with reporting poorer physical or mental health, after controlling for the demographic variables. For outcome variables with low prevalence (depression rating >8, depression diagnosis, and cognitive impairment), two separate versions of the models were run: one including reporting a current sleep problem, and the other including reporting a past sleep problem.

Models included between 89.7% and 98.8% of all observations, due to missing values. Co-linearity between dependent variables was investigated using the tolerance and variance inflation factors, and by Pearson's or Spearman's correlation. None of the models had issues with co-linearity by these criteria. Odds ratios (ORs) and 95% confidence intervals (95%CIs) were estimated for groups of interest. Analyses were undertaken in SAS® (2011, Version 9.3, Cary NC).

1.5.4 Results

Demographics.

Of the 649 participants who responded to the question concerning a current sleep problem, 38.7% were Māori and 61.3% were non-Māori. Their demographic characteristics are summarised in Table 1.3

Sequential logistic regression analyses revealed that, after controlling for sex and NZdep 06, Māori were more likely to be within the group who did not answer the sleep question compared to non-Māori (OR=3.00, 95% CI = 2.16-4.16, p = <0.0001). The likelihood of not answering the sleep question also increased with age (OR = 1.18, 95% CI = 1.10-1.27, p = <0.0001).

Table 1.3

Demographic Characteristics of Māori and Non-Māori Participants in the Sleep Sample

Variable		Māori (n=251) n (%)		Non-Māori (n=398) n (%)	
Females		153	(61.0%)	211	(53.0%)
Carers†		84	(33.5%)	107	(26.9%)
NZ_Dep06					
Least deprived:	Decile 1	4	(1.6%)	12	(3.0%)
	Decile 2	2	(0.8%)	13	(3.3%)
	Decile 3	6	(2.4%)	28	(7.0%)
	Decile 4	24	(9.6%)	47	(11.8%)
	Decile 5	11	(4.4%)	16	(4.0%)
	Decile 6	31	(12.4%)	85	(21.4%)
	Decile 7	20	(8.0%)	67	(16.9%)
	Decile 8	34	(13.6%)	59	(14.8%)
	Decile 9	34	(13.6%)	37	(9.3%)
Most deprived:	Decile 10	85	(33.9%)	34	(8.5%)

† n = 249 for Māori carers, & 386 for non- Māori carers

Reporting sleep problems.

In the sleep sample, 26.3% of Māori and 31.7% of non-Māori reported having a current sleep problem (chi square = 2.13, p = 0.145). Women were more likely to report a current sleep problem than men (33.0% vs. 25.3%, chi square= 4.55, p = 0.033). *Figure 1.5* shows the proportions of Māori and non-Māori reporting a sleep problem who endorsed each sleep symptom. Both Māori and non-Māori endorsed a median of three sleep symptoms.

All participants reporting a current sleep problem endorsed at least one symptom of insomnia (waking up in the early hours of the morning, taking a long time to get to sleep, and/or lying awake most of the night). "Other sleeping problems" noted by participants included pain, restless legs, symptoms of sleep disordered breathing, hallucinations, taking medications, and care giving. In total, 5.2% of Māori and 6.8% of non-Māori reported having a sleep problem when they were young. Table 1.4 shows the results of the multiple logistic regression analyses investigating factors associated with reporting a current sleep problem.

Table 1.5 summarises the findings from the 13 logistic regression models investigating current and past sleep problems as independent risk factors for poorer health. The demographic variables (ethnicity, age, sex, NZdep 06 and caregiving status) were also included in each model.

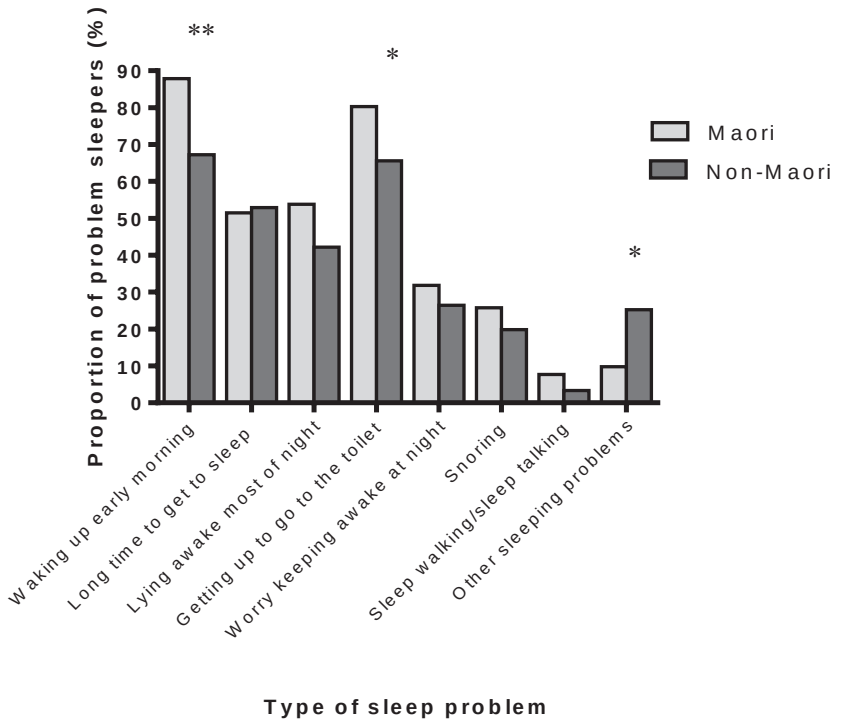


Figure 1.5. The proportion of sleep symptoms endorsed by Māori (n=66) and non-Māori (n=126) participants who reported having a current sleep problem.

* = p (chi square) <0.05, ** = p (chi square) <0.01.

Table 1.4

Independent Associations Between Reporting a Current Sleep Problem and Demographic and Health-Related Factors

Independent variables	Categories/range	Adjusted OR	95%CI
Ethnicity	Māori	0.52*	0.30-0.90
	Non Māori (Ref.)		
Sex	Females	1.48	0.98-2.24
	Males (Ref.)		
Age	79-90	0.94	0.82-1.07
Deprivation (NZdep 06)	1-10	1.05	0.99-1.14
Caregiving	Carer	1.52	0.99-2.32
	Non Carer (Ref.)		
Sleep problem when young	Yes	2.67*	1.25-5.72
	No		
Mental health (SF-12)	14.9-79.3	0.95***	0.92-0.98
Cognition (MMSE)	2-30	0.96	0.89-1.03
Depression rating (GDS)	1-14	1.09	0.96-1.24
Depression diagnosis	Yes	1.37	0.73-2.54
	No (Ref.)		
Perceived Coping (PMS)	5-25	1.04	0.96-1.14
Perceived Control	9-35	0.98	0.92-1.05
Physical health (SF-12)	6.0-65.8	0.97*	0.94-1.00
Pain interference (SF-12)	0-100	1.00	0.99-1.01
No. Falls	0-3	1.14	0.92-1.42
Alcohol	Drinker	0.81	0.53-1.24
	Non drinker (Ref.)		

N = 582, 89.7% of full sample. * $p<0.05$, ** $p<0.01$, *** $p<0.001$
Abbreviations: SF-12 (Short Form 12 Item Survey), MMSE (Mini Mental State Exam), GDS (Geriatric Depression Scale), PMS (Pearlin Mastery Scale), OR (Odds Ratio),CI (confidence Interval), Ref. (Reference group). A version of the model was run with the interaction term sex X ethnicity, but the interaction was non-significant.

Table 1.5

Independent Associations between Reporting Current or Past Sleep Problems and Poorer Health

Model	Health outcome variable	n, (%) with condition	Observations	Sleep problem	Adjusted OR	95%CI
1	Depression, (GDS)†	49 (7.6%)	641	Current	4.42***	2.40-8.14
2	Depression, (GDS)†	49 (7.8%)	629	Past	2.70*	1.07-6.92
3	Depression diagnosis†	62 (9.8%)	631	Current	1.96*	1.15-3.37
4	Depression diagnosis†	62 (10.0%)	621	Past	4.19***	1.94-9.08
5	Cognitive impairment†	76 (12.1%)	629	Current	1.49	0.89-2.61
6	Cognitive impairment†	61 (11.5%)	618	Past	0.95	0.32-2.83
7	Poor mental health	155 (25.5%)	608	Current	2.45***	1.65-3.63
				Past	0.86	0.40-1.88
8	Poor coping	144 (23.5%)	612	Current	1.80**	1.20-2.71
				Past	0.85	0.38-1.87
9	Poor control	167 (27.4%)	610	Current	1.82**	1.23-2.68
				Past	2.49**	1.26-4.92
10	Poor physical health	148 (24.3%)	608	Current	1.64*	1.10-2.46
				Past	1.91	0.94-3.88
11	Fallen in last year	215 (34.5%)	624	Current	1.64**	1.14-2.36
				Past	1.77	0.89-3.48
12	Reduced physical activity	147 (23.7%)	624	Current	1.36	0.89-2.06
				Past	1.57	0.75-3.29
13	Increased pain interference	181 (29.2%)	619	Current	1.89**	1.30-2.76
				Past	2.41*	1.22-4.77

† Complete models had too small a sample size for the outcome variable and were restricted to 6 independent variables to maintain statistical power. Therefore two versions of the model were run, the first with the outcome variable of reporting a current sleep problem, including the five demographic variables (ethnicity, age, sex, NZdep06 and caregiving status); and the second with the outcome variable of reporting a past sleep problem, including the five demographic independent variables.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

1.5.5 Discussion

This is the first study to estimate the prevalence of reporting sleep problems among Māori and non-Māori of advanced age. Among 251 Māori and 398 non-Māori aged 79-90 years, we found that 25.5% of Māori and of 31.7% non-Māori reported a sleep problem. Reporting a current sleep problem was more likely among non-Māori and those who reported a sleep problem when younger, poorer physical or mental health, and depression. The associations between sleep problems and health are likely to be bi-directional (Kondratova & Kondratov, 2012), highlighting the importance of sleep for the health-related quality of life of our rapidly ageing population.

In contrast to previous research involving 20-59 year olds (Paine & Gander, 2013; Paine, et al., 2005), the present study found that Māori were *less* likely to report sleep problems than non-Māori, and that socioeconomic deprivation (NZdep 06) was not associated with sleep problems. The reasons for these differences are unknown. Response bias in the current study may have been a factor, since Māori were three times more likely than non-Māori to be amongst those who did not respond to the sleep questions, and people with poorer health were less likely to complete the full questionnaire with the sleep questions. The lower prevalence of reporting sleep problems among Māori could also reflect the fact that the LiLACS NZ sample includes exceptionally long lived Māori, the majority of whom strongly identify with their culture (e.g., many being fluent in speaking Te reo Māori, and frequently visiting a marae) compared to younger generations (Dyall, et al., 2014). Previous LiLACS NZ research has identified cultural engagement (but not socioeconomic position) as an independent predictor of better physical health-related quality of life (Dyall, et al., 2014). Further research is required to clarify relationships between lifestyle and cultural practices and sleep problems.

In contrast to previous research (Paine & Gander, 2013), in the current study NZdep 06 was not associated with reporting sleep problems. The Māori participants in the present study were over-represented in the most deprived NZdep 06 deciles, whereas the non-Māori sample had a more normally distributed NZdep 06 profile. These distributions are similar the general population of Māori and non-Māori aged over 50 years (Ministry of Health, 2012a). However they differ from previous studies of 20-59

year olds where non-Māori were over represented in the least deprived deciles (Paine & Gander, 2013). It is also possible that NZdep 06 alone is not the most reliable way to measure socioeconomic position in people of advanced age, who are more likely to live with family members or in rest homes or institutions that may not reflect their true position (Statistics New Zealand, 2006b). Alternatively, socioeconomic position may be less closely related to health-related quality of life in advanced age (Dyall, et al., 2014). Additional research using other or multiple methods of defining socioeconomic position is needed to clarify this.

The prevalence of reported sleep problems is lower in the LiLACS NZ sample than in previous studies of adults aged 60-80 years (Foley, et al., 1995; K. Reid, et al., 2006). Grandner et al. (2012) propose that this pattern could be related to the exceptional health that leads to living longer, or a reduction in the effects of life or societal stressors on sleep in advanced age. Expectations and perceptions of good or acceptable health are also thought to change with age (Brouwer, Van Exel, & Stolk, 2005) and older people as well as their family members may downplay or be less likely to complain of sleep disruptions (Gibson, et al., 2014). It is therefore important for healthcare professionals to actively ask their older patients about sleep (K. Reid, et al., 2006).

More symptoms were endorsed by people reporting sleep problems compared to previous studies (median = three for Māori and non-Māori; K. Reid, et al., 2006). However, the list included trips to the toilet and waking early, which are not necessarily sleep "problems" *per se*. All of the participants reporting a current sleep problem reported at least one insomnia symptom. Insomnia is common with ageing and reflects physiological and psychological changes that increase the likelihood of sleeplessness (K. Reid, et al., 2006).

Carers are considered likely to have disturbed sleep (Arber & Venn, 2011; Gibson, et al., 2014). However in the present study, caregiving (including carers providing any type or amount care) was not associated with reporting a current sleep problem. Future research should consider gathering more detailed information on the amount and type of care being provided.

Those who reported having a sleep problem when they were younger were more likely to report a current sleep problem, although the definition of "younger" used in the survey is ambiguous. This relationship has been reported throughout the lifespan and highlights the importance of early diagnosis and treatment of sleep problems as a way of preventing any negative impact in later life.

With each point of increase on the mental health related QOL scale there was a 6% reduction in the likelihood of reporting a sleep problem. Those who reported a current sleep problem were more likely to score within the lowest quartile for mental health, have a diagnosis of depression, have a moderate-high self-rated depression score, and had poorer perceived control compared to those who did not. Moreover, participants reporting a sleeping problem when they were younger were more likely to have a current diagnosis of depression and poorer perceived control compared to those who did not. Cognitive functioning and mood have consistently been related to sleep problems, these relationships have been attributed to physiological processes as well as to the effects of sleep deprivation (Kondratova & Kondratov, 2012). Reporting sleep problems and cognitive impairment were not significantly associated in the present study, possibly due to those with more severe impairment being less likely to have completed the long form of the survey which included the sleep questions.

With each point increase on the physical health related QOL scale there was a 3% reduction in the likelihood of reporting a sleep problem. Participants reporting a current sleep problem were more likely to score within the poorest quartile for physical health and pain that interferes with daytime functioning. Those who reported a past sleeping problem were also more likely to report pain. This relationship is likely to be multifactorial, as pain is common with ageing and is also related to poorer physical and mental health, mobility problems, and quality of life (Foley, et al., 2004). Participants reporting a current sleep problem were more likely to have fallen in the past year than those who did not. This relationship may be due to higher daytime sleepiness causing issues with balance and reaction times, getting out of bed whilst drowsy, and the residual effects of sleeping medications (Stone, et al., 2008).

A limitation of this study is that the LiLACS NZ survey was not designed with sleep as a primary focus and therefore the data are difficult to compare to previous studies using different questions. Future studies would be strengthened by the use of validated and standardised sleep questions and scales.

Objective sleep monitoring would help identify any potential discrepancies with self-reported sleep problems, but such an intensive protocol may dissuade people of advanced age from participation (Harris & Dyson, 2001).

The present analyses did not address the use of sleeping medications, physical exercise, light exposure, doctor's visits, or comorbidities. These factors have been highlighted as significantly associated with sleep problems in previous studies (Foley, et al., 2004; Foley, et al., 1995). Risk factors for, and symptoms of, sleep disordered breathing were also not examined in detail, although the proportion of snorers was small.

Despite these limitations and the potential response biases, LiLACS NZ provides a large sample to investigate the factors related to reporting sleep problems in Māori and non-Māori of advanced age. The unique design and the researchers' commitment to engaging and recruiting this specific group has provided an outstanding opportunity for the first investigation of sleep of this age group in NZ.

Conclusions.

These analyses show that 26.3% of Māori and 31.7% of non-Māori of advanced age report current sleep problems. Sleep problems are a significant marker for poorer health status in advanced age. It is important for clinicians as well as family members to explore whether the elders in their care have sleep problems, which may exacerbate physical and mental health problems as well as increase the risk of falling.

Acknowledgements.

We would like to acknowledge the participants of LiLACS NZ. We are very grateful to the LiLACS NZ research team and Leadership Group for their willingness to share their data with us and for their advice and support. Funders of LiLACS NZ include the Health Research Council of New Zealand programme grant, and Ngā Pae o te Māramatanga (New Zealand's Māori Centre of Research Excellence) project grant.

1.6 Study 2: The Effect of Caregiving Status on the Sleep of Older New Zealanders

1.6.1 Abstract

Objectives: To investigate relationships between caregiving and subjective sleep reports among older New Zealanders. Methods: Participants were 434 carers (177 home-based) and 1,747 non-carers who completed the 2008 Health Work and Retirement survey (aged 55-72 years). Sleep outcomes (feeling worn out; tired; dissatisfied with sleep; or having a diagnosed sleep disorder) were explored by caregiving status. Logistic regression was used to investigate caregiving as an independent predictor of poor sleep health after controlling for other demographic variables. Results: Home-based carers were more likely to report feeling tired some/all of the time than non-carers (OR 1.55, 95%CI 1.10-2.16). Being Māori, female, younger, or more socioeconomically deprived (NZdep 2006), were also significant independent predictors of poor sleep outcomes. Conclusions: Carers are at increased risk of feeling tired in the daytime. Early recognition and management of underlying sleep problems is particularly important for older people providing care at home.

1.6.2 Introduction

Approximately one quarter of adults aged less than 60 years in Aotearoa/New Zealand (NZ) report a sleep problem lasting at least 6 months (Paine, et al., 2005). International research indicates that these numbers are higher for those aged over 60 years (Foley, et al., 2004). When older people are also required to care for someone with an illness, a situation is created where sleep problems are likely to be exacerbated (Alpass et al., 2013; Arber & Venn, 2011). For example, carers of people with dementia report sleep problems related to being disturbed by the person in their care as well as the physical and psychological burden of caregiving (Gibson, et al., 2014). Being a carer within the family home is likely to create a situation where longer hours of support are being provided and carers more are likely to be working at night, compared to those providing care to people living in another house or hospital (Arber & Venn, 2011). The proportion of older people in the population is increasing and more are requiring in-home care (Cornwall & Davies, 2004). Poor sleep has been related to increased physical and mental health

problems as well as institutionalisation of care recipients (Foley, et al., 2004; McCurry, Logsdon, Teri, & Vitiello, 2007; K. Reid, et al., 2006) and so research is required to better understand and manage the sleep of older carers.

Previous NZ-based research has demonstrated that there are significant differences in the prevalence of common sleep problems and disorders between Māori and non-Māori adults (aged 20-59 years). There are also consistent relationships between sleep and socioeconomic position, sex and age (Paine & Gander, 2013). These relationships have yet to be investigated in older New Zealanders and need to be considered when investigating the relationship between sleep and caregiving status.

The Health Work and Retirement (HWR) study was designed to better understand the health of older adults in the general population as they move through retirement to older age (Alpass et al., 2007). Information concerning sleep and caregiving in the 2008 dataset provides a unique opportunity to examine aspects of the sleep health of older carers and non-carers in NZ. The aims of the present study were to estimate the prevalence of factors indicating poor sleep health among carers and non-carers aged 55 years or more, and to give a descriptive overview of the independent demographic predictors of poorer sleep health. Based on previous research, it was hypothesised that at least 25% of the sample would self-report factors indicating poor sleep, and that carers, particularly home-based carers, would have poorer sleep health compared to non-carers. It was also predicted that participants who were Māori, female, older, or more socioeconomically deprived would be more likely to report poor sleep.

1.6.3 Methods

Participants were recruited into the HWR study via the electoral roll, Māori were oversampled in order to maximise the explanatory power between Māori and non-Māori populations. A postal survey was used to collect information concerning health (including sleep) and use of healthcare services; individual and household demographics; work and retirement; independence; and social participation (including caregiving responsibilities; Alpass, et al., 2013; Alpass, et al., 2007). The sample's physical and mental health scores from the Short-Form 12 Question Health Survey (SF-12) are described below. For further information regarding the status of the overall cohort please see Alpass et al. (2013; 2007).

Demographic variables included in the present analyses were: ethnicity, socioeconomic position, sex, and age. Participants were classified as Māori (if they identified themselves as Māori, either alone or as one of multiple ethnicities) or as non-Māori. Socioeconomic position was defined by the NZ Deprivation index 2006 (NZdep) which categorises individuals on a scale of 1 (least deprived) to 10 (most deprived) based on the geographical area (census meshblock) in which they live (Salmond, et al., 2007).

Carers were defined by a positive response to the question: "Do you regularly provide care or assistance (e.g., personal care, transport) to any of the following people because of their long-term illness, disability, or frailty?: someone who lives with you; someone who lives elsewhere; someone who is now in a nursing home or hospital". Non-carers were defined as those who identified as not caring for someone with a long-term illness, disease, or frailty; being a carer but not actively providing care for more than 12 months; or those who currently care for someone as part of their paid work (note the definition of 'carer' differs slightly from that used by Alpass et al., 2013). Carers also indicated what their relationship was to the person in their care, as well as the frequency and duration of caregiving.

The following sleep-related questions were extracted from the Short Form 36 Question Health Survey (SF-36; Ware, et al., 2000) and the World Health Organisation Quality of Life Measure (Skevington, Lotfy, & O'Connell, 2004) and were categorised to represent poor sleep health versus good sleep health: "During the past 4 weeks did you feel worn out?" (*some / all of the time* vs. *none / a little of the time*); "During the past 4 weeks did you feel tired?" (*some / all of the time* vs. *none / a little of the time*); "How satisfied are you with your sleep?" (*dissatisfied / very dissatisfied* vs. *satisfied / neither way*). Participants were also asked to indicate if they had been diagnosed with a sleep disorder.

Responses to the four sleep-related questions were compared according to caregiving status using chi square analysis. Logistic regression models were used to investigate whether caregiving status was independently associated with measures of poor sleep, after controlling for ethnicity, NZdep, sex, and age. Odds ratios (ORs) and 95% confidence intervals (95%CIs) were estimated for groups of interest. Study analyses were undertaken in SAS® (2011, Version 9.3, Cary NC).

1.6.4 Results

Among the 2,473 participants who completed the 2008 survey, 88.2% could be defined as either being a carer or a non-carer (N = 2,181). Of these, 38.8% were aged 65 years or over (median = 63 years, range = 55-72 years), 51.2% were Māori, and 53.7% were female. On average, participants' rated their health as *very good* from a scale of *excellent-poor*. Participants' mean physical and mental health scores on the SF-12 were 45.3 (SD = 15.2) and 46.7 (SD = 15.1) respectively, indicating just below average quality of health within the sample (Quality Metric, 2013).

The demographic profile of the participants differed significantly by ethnicity. Māori participants were over-represented in the most deprived NZdep deciles (chi square = 176.69, p<0.0001) and were also more likely to be female (54.9% vs. 50.4%, chi square = 4.43, p = 0.035). The Māori and non-Māori participants were not significantly different in age.

Of the 434 carers, 40.8% provided care to someone who lived with them (*home carers*) while the remainder provided care to people living privately elsewhere or in a nursing home or hospital (*other carers*). Carers were significantly more likely to be Māori (chi square = 13.19, p = 0.001) and female (chi square = 29.52, p = <0.0001) than non-carers. The NZdep profile of participants also differed significantly by carer status. Carers, particularly those caring for someone in their own home, were more likely to be over-represented in the most deprived deciles compared to non-carers (*Figure 1.6*; chi square = 33.52, p = 0.014). Details of the caregiving relationship, as well as frequency and duration of care for home-based and other carers can be found in Table 1.6.

Within the previous four weeks, 38.2% of the full sample reported feeling worn out some or all of the time and 54.2% reported being tired some or all of the time. At the time of completing the survey, 20.4% reported being dissatisfied or very dissatisfied with their sleep, and 8.1% had been diagnosed with a sleep disorder. Responses to the sleep-related questions are broken down by carer status in Table 1.7.

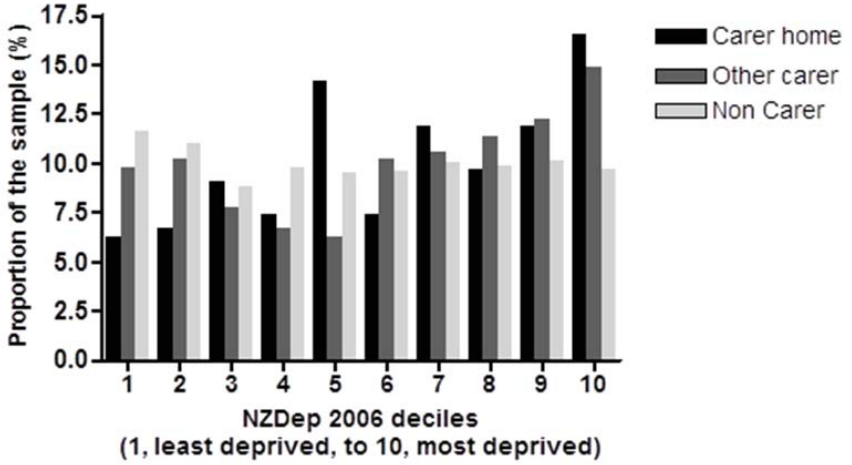


Figure 1.6. Zealand Deprivation Index 2006 (NZdep) profile for carers at home, other carers and non-carers (n=2,164).

Table 1.6

Description of Carers

	Home carers	Other carers
Māori	59.9%	58.4%
Female *	55.4%	68.1%
Age (years, range)	64 (56-72)	62 (56-72)
Relationship of care recipient ***		
Spouse/partner	59.4%	2.2%
Parent	10.0%	30.0%
Other relative	24.4%	35.7%
Friend or Someone else	6.3%	32.2%
Age of care recipient (years, range) ***	66 (1-98)	82 (5-99)
Recipient has AD/dementia **	5.1%	15.6%
Care frequency ***		
Every day	82.0%	14.2%
Once a week-several times a week	13.8%	64.0%
Once every few weeks-less often	4.2%	21.8%
Usual care duration per occasion ***		
All day and night	32.3%	7.1%
All night	0.6%	1.7%
All day	7.3%	5.9%
An hour to several hours	59.8%	85.3%

Groups compared using chi square analyses or Wilcoxon 2 way analyses, *p <0.05. ** p<0.001. ***p<0.0001.

Table 1.7

Sleep Responses by Caregiving Status

	Carers		Non-carers		
	Home carer	Other carer		$\chi 2$	P
Worn out (some or all of the time)	44.7%	43.0%	36.9%	6.77	0.034
Tired (some or all of the time)	64.7%	57.2%	52.7%	10.11	0.006
Dissatisfied-very dissatisfied with sleep	24.4%	23.7%	19.5%	4.24	0.120
Diagnosed sleep disorder	11.9%	8.2%	7.7%	3.81	0.149

Logistic regression models were run to identify independent risk factors for poorer sleep (Table 1.8). Versions of the models were also run with the following interaction terms: caregiving status*ethnicity; caregiving status*NZ Dep; and caregiving status*sex. None of these interaction terms were significant so they were removed from the final models summarised in Table 1.8. The only significant relationship with carer status was that home carers were more likely than non-carers to report feeling tired some or all of the time.

Participants were also dichotomised by age (≥65 years vs. <65 years) and by retirement status (yes/no). Chi square analyses revealed that participants who were aged ≥65 years were significantly *less* likely to report feeling worn out (chi square = 6.30, p = 0.012) or dissatisfied with their sleep (chi square 16.14, p = <0.0001) compared to those aged <65 years. Retirement status was not significantly related to the sleep variables at either the univariate level or in alternative versions of the logistic regression models.

Table 1.8

Associations Between Reporting Feeling Worn out, Tired, Dissatisfied With Sleep, or Having a Diagnosed Sleep Disorder, with Caregiving and Other Demographic Factors

Variable	Worn out: Some or all of the time (ref. none-a little of the time)		Tired: Some or all of the time (ref. none-a little of the time)		Dissatisfied-very dissatisfied with sleep (ref. Neither way-very satisfied)		Sleep disorder (ref. no sleep disorder)	
	OR	95%CI	OR	95%CI	OR	95%CI	OR	95%CI
Home carers (ref. non-carers)	1.26	0.91-1.75	1.55*	1.10-2.16	1.39	0.95-2.01	1.44	0.88-2.36
Other carers (ref. non-carers)	1.19	0.90-1.57	1.12	0.85-1.47	1.24	0.90-1.71	1.01	0.62-1.64
Māori (ref. Non-Māori)	1.37**	1.14-1.65	1.12	0.93-1.34	0.95	0.76-1.19	1.76*	1.26-2.47
NZDep06 (1,least deprived to 10, most deprived)	1.10***	1.07-1.14	1.07***	1.04-1.11	1.01	0.97-1.05	1.08*	1.03-1.15
Female (ref. male)	1.05	0.88-1.27	1.19*	1.00-1.43	1.35*	1.09-1.69	0.84	0.61-1.15
Age (55-72 years)	0.97*	0.95-0.95	0.98*	0.96-1.00	0.94***	0.91-0.96	1.01	0.98-1.05

*p <0.05. ** p<0.001. ***p<0.0001.

CI, confidence interval; NZdep, New Zealand Deprivation Index 2006; OR, odds ratio; ref., reference group.

1.6.5 Discussion

This is the first study to investigate differences in the prevalence of sleep problems of New Zealand carers. Using a sample of 177 home-based carers, 257 other carers, and 1,747 non-carers (aged 55-73 years), we found that 20% of the sample reported being currently dissatisfied with their sleep, and 8.1% had a diagnosed sleep disorder. The prevalence of feeling worn out or tired (some or all of the time over the last four weeks) was 38.2% and 54.2% respectively. Providing care at home independently increased the risk of reporting feeling tired some or all of the time. As with previous research, poorer self-reported sleep was also associated with being Māori, more socioeconomically deprived, and female (Gander, et al., 2005a; Paine & Gander, 2013; Paine, et al., 2005). Conversely, age was found to have a negative relationship with reporting poorer sleep.

Previous research shows that sleep disturbances are common among family carers (Gibson, et al., 2014; McCurry, et al., 2007; Pollak & Perlick, 1991). The types of sleep disturbance are varied and typically involve interactions between the effects of having a disrupted sleep schedule due to providing care assistance overnight, carer burden, and the comorbidities of the carer (Gibson, Gander, Alpass, & Stephens, in press; McCurry, et al., 2007). In this sample, home-based carers were more likely to be caring for their spouse/partner, and were also more likely to provide care every day as well as all day and night, compared to those providing care elsewhere. Due to reduced statistical power and the lack of standardised questions, the variables were not stratified any further. However it is anticipated that the relationship of the carer with the care recipient, level of dependency, and carer work-load at night might increase carer stress and subsequent sleep problems (Arber & Venn, 2011). Previous research has identified sleep as a particular issue for carers of people with dementia. Sleep problems of these carers have been related to having increased disturbances at night and the psychological impact of care giving (Arber & Venn, 2011; Chenoweth & Spencer, 1986). Further research is required with a larger group of dementia-related carers to verify this in the NZ context.

Carers were more likely to be Māori, more socioeconomically deprived, and female. These demographic groups were also identified as having increased likelihood of reporting poor sleep outcomes.

The interaction terms for these relationships were not significant in the logistic regression models. However, it is important to recognise that a range of factors may operate to increase the risk of carers having sleep problems, making them a particularly vulnerable group.

These analyses found that Māori were 1.4 times more likely to report feeling worn out in the last four weeks (some or all of the time) and were 1.8 times more likely to have a diagnosed sleep disorder. These findings corroborate previous research with younger Māori adults (aged 20-59 years). Ethnic disparities in sleep health could be associated with sleep disordered breathing, physical and mental health or socioeconomic position (Gander, et al., 2005a; Ownby et al., 2010; Paine & Gander, 2013).

Females were 1.2 times more likely to report feeling tired some or all of the time and 1.4 times more likely to report being dissatisfied to very dissatisfied with their sleep. These findings support previous research which identifies older women (aged 50 years and over) as being more likely to report disturbed sleep, particularly insomnia-type symptoms. Such complaints have been associated with increased worry at night, mental health problems, hormonal changes, and with use of sleeping medications (Foley, et al., 1995; Lichstein, et al., 2011; Middelkoop, et al., 1996).

This study has the unique finding that increasing age was associated with reduced likelihood of reporting poor sleep health. With each year of increased age there was a reduction in the likelihood of reporting feeling worn out (3%) or tired (2%) some or all of the time, and being dissatisfied or very dissatisfied with sleep (6%). Age did not statistically differ by ethnicity, NZDep, or sex. Previous research using objective measures consistently shows that sleep quality and duration reduce with age (Ohayon, et al., 2004). However, as the present study shows, subjective reports may not reflect these physiological changes. Grandner et al. (2012) propose that a seemingly lower prevalence of self-reported sleep problems in older age could be a reflection of fewer life stressors or societal influences on sleep compared to younger, working adults. Expectations and perceptions of good or acceptable health are also thought to change with age (Brouwer, et al., 2005). Therefore older people may be less likely to complain about sleep disruption and subsequently be less likely to be assessed for sleep disorders. Sleep problems have been consistently related to poorer physical and mental health (Foley, et al., 2004; K. Reid, et al., 2006),

making it important for healthcare professionals to actively ask their older patients about sleep (K. Reid, et al., 2006).

A limitation of this study is that the survey was not designed with sleep as a primary measure, and the data are difficult to compare to previous studies using different questions. Identification of carers was limited by some participants failing to answer the caregiving module of the questionnaire and some responding in contradictory ways to different questions about their caregiving status. As a result, the final data set included only 434 participants identified as carer. Given the limitations of the sleep-related questions and the limited number of carers, we chose not to control for comorbidities or other potential confounding factors (e.g., physical and mental health, medications, or quality of life; Foley, et al., 2004; K. Reid, et al., 2006) in the logistic regression models. Additional research is needed to address these factors, using validated questions on sleep and with robust definitions of care-giving status. Future studies would also be strengthened by having more information on the nature of participants' sleep disorders (diagnosis and treatment), and assessments of carer burden. Objective monitoring of sleep (e.g., using actigraphy or polysomnography) would help clarify any discrepancies with self-reported sleep problems and could also be used to investigate associations between the sleep of the care recipient and that of their carers. However, older people may also be discouraged by the high demands of quantitative sleep study protocols (Harris & Dyson, 2001).

In summary, sleep-related information from the 2008 HWR survey has provided a unique descriptive overview of the sleep of older carers and non-carers. This is the first study of its kind in NZ (with previous studies focused on adults aged 20-59 years). These findings highlight the need for further locally-based research specifically designed to investigate sleep with ageing and caregiving.

1.7 Overview of the Findings from Studies 1 and 2

Key points from studies 1 and 2 showed that around a quarter of older New Zealander's reported a current sleep problem, or being dissatisfied with their sleep. Sleep problems are associated with reduced physical and mental health as well as an increased risk of falling. In the younger sample of Study 2, Māori and females were more likely to report feeling worn out or having a sleep disorder, which is similar to the findings of previous studies (Paine, Harris, & Mihaere, 2007). Carers were more likely to report feeling tired. Such demographic disparities in sleep health were not evident in the sample of New Zealanders of advanced age (Study 1). It appears that with increased age, the likelihood of reporting a sleep problem declines. It may be that demographic predictors for sleep are less relevant with older age, and greater focus needs to be paid to the physical and mental health predictors for sleep problems, as well as caregiving status.

2 SLEEP AND DEMENTIA

In 2011, an estimated 48,182 New Zealanders were living with dementia (approximately 1.1% of the population; Alzheimers New Zealand Incorporated, 2012). This is 1.2 times higher than in 2008, and it is projected that by 2050 this number will triple. Dementia has a significant economic impact. The estimated national cost of dementia in 2011 was approximately $954.8 million, the majority of which was associated with healthcare expenditures including residential care (Alzheimers New Zealand Incorporated, 2012).

Dementia rarely occurs before the age of 50 years and increases in likelihood with increasing age. At the age of 65-70 years approximately 2% of individuals have dementia, whereas those aged 85 years or over have a 20-40% prevalence (Alzheimers New Zealand Incorporated, 2012; Jorm & Jolley, 1998; Kawas, Gray, Brookmeyer, Fozard, & Zonderman, 2000). Life expectancy is increasing, so the number of cases of dementia, particularly of the Alzheimers type, will also increase (Alzheimers New Zealand Incorporated, 2012; Statistics New Zealand, 2012; Tobias, Yeh, & Johnson, 2008). Women have a longer life expectancy and therefore also have increased likelihood of experiencing dementia. In NZ it is estimated that 60% of people with dementia (PWD) are female (Alzheimers New Zealand Incorporated, 2012; Statistics New Zealand, 2012). Similarly, New Zealanders who are non-Māori, non-Asian, or non-Pacific Islanders also have a higher prevalence of dementia (accounting for 90.3% of estimated cases in 2011). However, with the increasing number of Māori and Pacific Island people living into advanced age, prevalence rates are expected to also rise for these ethnic groups (Alzheimers New Zealand Incorporated, 2012; Ministry of Health, 2012b).

This chapter begins with an overview of dementia including a description of the symptoms, risk factors, outcomes, and management. These are presented to give a context for the effects that dementia can have on sleep and to highlight the challenges for conducting research with PWD. The second half of the chapter describes the sleep-related changes specific to dementia, as well as the sleep of those providing informal care to PWD. The methodological limitations of existing research are outlined within an ethical and person-centred philosophy of research. This highlights gaps in our understanding about how PWD as well as their carers personally experience sleep disturbances. To address some of these gaps, the chapter

concludes with a published paper from Study 3, which used focus groups to gather information about dementia-related sleep disturbances from the viewpoint of the PWD as well as their carer.

2.1 What is Dementia?

The term *dementia* is used to describe a collection of symptoms representing a cognitive disorder. It is "a progressive loss of memory, intellectual and linguistic skills, usually accompanied by radical changes in personality and sometimes in motor skills" (Stuart-Hamilton, 2006, p. 219). Dementia therefore affects a person's functional abilities within activities of daily living. In the early stages this could manifest as an inability to focus on work or reading. In the moderate stages, lapses in short-term memory are more frequent and PWD may be unable to safely and reliably perform housework. In the final stages, the cognitive impairment is such that learning and memory systems are devastated and the ability to maintain personal care and hygiene are affected (Woodruff, 2008).

Dementia is caused by degeneration within areas and networks within the brain, typically associated with neuropathological disease or vascular damage (see below). As sleep is regulated by diverse areas and networks within the brain (as highlighted in section 1.1.2), it is unsurprising that sleep disturbances are a common symptom of dementia (see section 2.2). Dementia has profound effects on the individual's memory and *sense of self* (Garratt & Hamilton-Smith, 1995). Maintaining somebody with dementia's place in society, honouring their views and contributions, while protecting their sense of self, are key considerations for the ethical, sensitive, and reliable conduct of dementia-related research (Sabat, 2005, see sections 2.3 and 3.4 for more detail). These psychosocial changes have also been associated with sleep disturbances and so are considered briefly below.

Like sleep, memory is a complex series of phenomenon which rely on multiple areas of the brain to register, encode and store information, as well as retrieve stored information in response to cues. If any one aspect of these mental processes is affected, then memory begins to falter (Baddeley, 2012). Memory can be separated into two types of learning. *Non-declarative* learning refers to knowing *how* to do something, for example, riding a bicycle or tying a shoelace. Non-declarative information is acquired through procedural processes or conditioning. This is known as *implicit* memory. On the other hand,

declarative learning refers to the ability to recognise and name people and things, as well as learn and recall short-term memories and events. Declarative memory involves explicit learning and recall - the individual needs to cognitively work to know *what* something is.

The formations of declarative and non-declarative memories are associated with different areas of the brain and neurological processes (Squire, 2004). The way dementia affects these areas and processes differs. Non-declarative memory is associated with diffused activity throughout the cortex (including the striatum, isocortex and cerebellum) rather than a specific location. Therefore, PWD's implicit knowledge and ability to perform procedural tasks is typically relatively well maintained, until dementia progresses to the severe stages when more of the brain is affected. Conversely, declarative memory relies more on the medial temporal region including the hippocampus, which is necessary for the learning and long-term consolidation of declarative memories (Baddeley, Bressi, Sala, Logie, & Spinnler, 1991; Squire & Zola, 1996). The medial temporal region and cholinergic system are where the most profound neurodegeneration associated with dementia takes place, particularly with Alzheimer's disease (Braak, Thal, Ghebremedhin, & Del Tredici, 2011; Geula, Nagykery, Nicholas, & Wu, 2008), see below. These changes, together with damage to the frontal and temporal lobes of the cortex, affect PWD's abilities to pay attention or make associations between memories and cues (Morris, 1994). As the dementia progresses, memory failure becomes more frequent and the ability to learn new information impaired (Baddeley, et al., 1991; R. Perry & Hodges, 1999; Stopford, Thompson, Neary, Richardson, & Snowden, 2012).

To avoid confusion or embarrassment associated with memory failure, some PWD might simply block attempts at recall, or provide false memories. This further weakens the neural networks required for memory (Garratt & Hamilton-Smith, 1995). These changes mean that the memory of PWD in the early or middle stages can be selective. Fragments of information are recalled on the basis of what the individual considers relevant to them at the time of exposure. Attention and mood also play a role (Garratt & Hamilton-Smith, 1995; R. Perry & Hodges, 1999). For the individual, changes to memory can be debilitating with regards to functioning in society as they would have done in the past. This can have an impact on wellbeing and quality of life (Lawton, 1991).

The *self* is a construct of how we see and identify with our person. Sense of self varies between and within individuals due to a combination of inherent factors as well as social and environmental influences and experiences. The construct and expression of the self varies between how we consider ourselves internally (the personal self) and how we present ourselves externally to others (the social self; Garratt & Hamilton-Smith, 1995; Sabat & Harŕe, 1992). For PWD, the personal self remains relatively intact (at least as far as the individual is concerned), whereas the social self is often affected (Baldwin, 2011; Kitwood, 1997; Kitwood & Bredin, 1992). This is due to psychosocial changes that are commonly associated with a diagnosis of dementia, including the social stigma associated with the disease, changes to relationships with friends and family, and poorer self-esteem. Impaired memory and ability to do things for oneself can affect feelings of orientation in relation to reality. This further affects the strength and continuity of sense of self. Due to the fragmentation of the elements necessary for an integrated sense of self, PWD often exhibit particular symptoms and behaviours to maintain or protect their social self. These include confusion, confabulation, or denying symptoms or need for assistance; the development of peculiar rituals in order not to forget or falter; as well as frustration, fear, or mania during situations which threaten the sense of self (Garratt & Hamilton-Smith, 1995; Sabat & Harŕe, 1992).

The most common types of dementia are Alzheimer's disease (AD), vascular dementia (VaD), and Lewy body dementia (LBD). Together these types account for over 90% of dementia cases (see below). Mild cognitive impairment (MCI) is recognised as an intermittent state between normal cognitive functioning and dementia, usually of the AD type (Lyketsos et al., 2002; Petersen, Stevens, et al., 2001).

Issues with the clinical diagnosis of dementia are outside the scope of this thesis. It should be noted, however, that reliable diagnosis is complicated. This is due to the variability between patients with regard to their symptoms, the rate and severity of disease progression, as well as comorbidities. Many have multiple causes of dementia. For example, about half of those with AD also have another type of dementia (Jellinger, 2006). A patient history, neurocognitive and psychiatric testing, as well as neuroimaging, are used to help diagnosis. Despite this, many people with suspected dementia are misdiagnosed or have an undefined type of dementia. In the case of AD, a definite diagnosis is unreachable until autopsy (Gurland &

Toner, 1983; Stuart-Hamilton, 2006). Management of the symptoms of dementia should be considered on a case-by-case basis, honouring the individual's experience (Fazio, 2013).

2.1.1 Alzheimer's Disease

The most common type of dementia is AD (around 77%, Barker et al., 2002), and so it is the main focus of this chapter. Characteristic symptoms of AD include problems with short-term memory and recognition; being unable to make new declarative memories or perform procedural tasks; and impaired language skills, for example, a loss of words or inability to conduct comprehensive conversations. Behavioural and psychological symptoms are also present in many cases. These symptoms include agitation, wandering, apathy, depression, anxiety, irritability, and psychosis (Cummings & McPherson, 2001; Liperoti, Pedone, & Corsonello, 2008; Malamut & Ryan, 2008; McKhann, Drachman, & Folstein, 1984). These symptoms typically increase as the cognitive impairment progresses. People with AD are usually moved into a care facility within six years of diagnosis, due to the lack of resources made available to informal carers (see section 2.2.5). Death usually occurs a few years later and is often associated with pneumonia or vascular disease (although the prognosis varies; Heyman, Peterson, Fillenbaum, & Pieper, 1997; Kukull et al., 1994; Stuart-Hamilton, 2006; Woodruff, 2008).

Alzheimer's disease is caused by cell decay in the brain over and above that of typical ageing. Characteristic bodies called neurofibrillary tangles and senile plaques are left behind, which are thought to interfere with the transmission of information within and between the associated neurons respectively. During the initial stages of AD, some plaques have been identified in the subcortical areas (the hippocampus, amygdala, and the brain stem), together with neurofibrillary changes in the entorhinal region (which is thought to serve as an interface between the hippocampus and the rest of the cortex; Squire & Zola, 1996). In the moderate stages of AD, plaques can be found in virtually all areas of the cortex and are particularly dense around the parietal, frontal and temporal lobes. Neurofibrillary degeneration is more dense in the entorhinal region, and the hippocampus itself is also affected. In the final stages of AD, plaques are present throughout the cortex. Neurofibrillary tangles continue to increase in the entorhinal region and hippocampus, and some are also present in the cortex itself (Braak & Braak, 1991; Woodruff, 2008). As the cells die, neural activity is affected particularly in the cholinergic system. The

brain shrinks as the disease progresses, leaving cavities, and becoming smaller and lighter compared to non-AD brains (Braskie et al., 2010; Stuart-Hamilton, 2006). These neuropathological changes translate to the progression of neuropsychiatric symptoms.

Other than age and being female, risk factors identified for AD include diabetes, cardiovascular diseases, and depression (Caselli, 2008). Genetics is also considered to contribute to the occurrence of AD. In particular, the presence of a specific allele of the gene Apolipoprotein E (ApoE) on chromosome 19 can account for approximately half of AD cases. The presence of the allele ApoE 4 predicts worsening memory, and each additional copy of the gene is correlated with a slightly earlier onset of the disease. Conversely, the presence of the allele ApoE 2 is associated with *reduced* susceptibility to AD (Farrer et al., 1997). However, the onset of dementia is not related to genetics alone. The risk of developing the disease is higher for the children or twin siblings of people with AD (Brickell et al., 2007; Brickell et al., 2006).

There is currently no cure for AD. Ways to prevent or delay the onset are therefore important, and treatments for managing or masking the symptoms are prescribed. Preventative measures include continued learning, higher education, and cognitive challenges in order to increase cognitive reserve and brain plasticity. A healthy diet, and plenty of exercise are also considered key (Middleton & Yaffe, 2009; Scarmeas et al., 2009; Stern et al., 1994). Medications for dementia tend to increase the supply of acetylcholine (e.g., Donepezil, Rivastigmine, and Galantamine). Acetylcholine cholinesterase inhibitors (AChEIs) can improve clarity of thought, functionality, mood and behaviour. They seem to be effective for some PWD, however they do not slow the progression of the disease and can have unpleasant side effects. The side effects are usually gastric related (e.g., nausea, vomiting, diarrhoea), but AChEIs can also aggravate heart conditions, cause dizziness and respiratory problems, and can cause sleep-related side effects (see sections 2.2 and 3.1; Francis, Palmer, Snape, & Wilcock, 1999). Symptoms of AD may also be improved by the drug memantine. This drug prevents the release of glutamic acid in the brain, an acid which damages brain cells and is typically released when damage has occurred in the brain. Memantine can slow (but not stop) the cognitive decline of dementia, however it is not state-funded in NZ and side effects can include hallucinations, confusion, headaches, as well as sleep disruptions (Reisberg et al., 2003).

Antipsychotics (e.g., Clozapine, Risperidone, or Olanzapine) are sometimes used to treat the psychological and behavioural symptoms of dementia. They are typically used when the behaviours are deemed challenging or dangerous to the PWD or others, as well as to sedate PWD who are more active at night (see section 2.2). These often work by stimulating acetylcholine activity in the brain whilst supressing or stabilising the activity of dopamine or serotonin. However, previous trials have shown mixed results regarding their effectiveness for PWD (Ballard, Waite, & Birks, 2012; Liperoti, et al., 2008; Schneider, Dagerman, & Insel, 2006). Long-term use of antipsychotics is not recommended as side effects include Parkinsonian-type movements, cerebrovascular events, sleepiness (see section 3.1), as well as overall increased mortality (Ballard, Creese, Corbett, & Aarsland, 2011; Ballard, et al., 2012).

Further considerations for AD and other dementias include the consumption of antioxidants (such as found in ginko biloba, and green tea) for increasing blood supply to the brain, and taking anti-inflammatories to reduce secondary neural damage. Psychosocial therapies such as cognitive behavioural therapy, memory aids, or reality orientation, as well as activity and support groups, are often used (Garratt & Hamilton-Smith, 1995). Finally, environmental considerations, as well as education, are considered key for PWD and their carers managing to cope with the symptoms of dementia (see Chapter 3).

Mild cognitive impairment: preclinical AD.

Mild cognitive impairment is a condition diagnosed when an individual does not meet the clinical criteria for dementia. People with MCI typically have some dysfunctional aspects to their memory or cognition, however the impact on daily living is not as marked compared to PWD (Petersen, Doody, et al., 2001; Woodruff, 2008). Longitudinal studies in Europe and the USA have estimated that 14-18% of the population aged over 70 years have a form of MCI (Busse, Hensel, Gühne, Angermeyer, & Riedel-Heller, 2006; Lopez et al., 2012). Symptoms of MCI typically increase with age until the individual meets the criteria for dementia, usually of the AD type (Busse, et al., 2006; Petersen, Stevens, et al., 2001).

Research comparing neuroimaging and autopsy data of people with MCI to those with AD or normal ageing have found that the type and location of deterioration to the brain is similar to AD (e.g., the presence of neurofibrillary tangles, and cortical atrophy). However, in this preclinical stage, only the hippocampus and the medial temporal lobe are affected, whereas the full temporal, parietal, and frontal

lobes are spared. These neuropathological changes are greater than is expected in healthy ageing, but lesser than in early AD (Jack et al., 1999; Mueller et al., 2010; Petersen et al., 2006). Like AD, the common pharmaceutical treatment option for MCI is also AChEIs etc., though clinical trials have reported mixed results (Petersen et al., 2009; Petersen et al., 2005).

2.1.2 Vascular Dementia

Vascular dementia accounts for approximately 18% of all dementias (Barker, et al., 2002). This refers to dementia caused by damage to the blood vessels resulting in an inadequate blood supply to the brain. This damage can originate from thrombosis, an embolism, or haemorrhage. As the brain cells are deprived of oxygen and nutrients they die. This can occur chronically or acutely. Most VaD is due to multiple successive infarcts causing cell death, known as multi infarct dementia (Stuart-Hamilton, 2006). Infarcts can occur anywhere in the brain, either randomly or in a focused region. Unresponsive blood vessels lead to either a reduction in blood when required, or damage to the blood vessel walls during times of heightened blood pressure, subsequently causing small bleeds and damage to the brain. Acute damage occurs as a result of an abrupt blockage such as that caused by a blood clot. This can cause a transient ischaemic attack or, worse still, a complete infarct (stroke).

The symptoms of VaD are more unique than those of AD, and depend on the locality of the damage. For example, damage specifically to the cortex is associated with intellectual impairment, whereas damage to the subcortical regions is associated with movement, and parietal damage with recognition. Memory is often affected in the early stages of any type of dementia, due to its reliance on so many areas of the brain. Mood-related symptoms are more common with VaD than AD, including depression, aggression, and apathy (Groves et al., 2000). Although the symptoms can appear quite similar to AD, with VaD they occur more abruptly, due to the sudden death of cells. The progression is described as "stepwise" compared to AD, as each infarct removes a portion of cognitive ability before a temporary plateau (Groves, et al., 2000; Malamut & Ryan, 2008; Stuart-Hamilton, 2006). This trajectory can make it difficult to diagnose or give a prognosis for VaD (Metter and Wilson 1993). It is common for people with VaD to also have AD, which further complicates the process (Knopman et al., 2003). Death occurs on average

four years after diagnosis, typically due to cerebrovascular events such as strokes (Fitzpatrick, Kuller, Lopez, Kawas, & Jagust, 2005).

The likelihood of VaD also increases with ageing. Unlike AD, the prevalence is greater for males than females. This has been associated with the other risk factors for VaD which have been identified as more common in males. For example, stroke, high blood pressure, heart disease, diabetes, smoking, as well as increased risk of head trauma (Yoshitake et al., 1995). Steps to reducing VaD include taking measures to avoid these underlying causes.

As with AD, there is no cure for VaD. Medications which preserve the blood vessels and prevent clotting, as well as those which stabilise blood pressure and lower cholesterol are recommended. Treatment options depend on the type and location of damage. As with AD, symptoms of cognitive impairment can be relieved through prescribed AChEIs and Memantine, as well as psychosocial therapies (Baskys & Hou, 2007).

2.1.3 Lewy Body Dementia

Lewy body dementia accounts for approximately 26% of dementia cases (Barker, et al., 2002). Early symptoms include attention and visuospatial difficulties. Memory is affected, but not to the same degree or as early as in AD. Patients with LBD typically experience more hallucinations and delusions than those with other types of dementia (Ballard et al., 1999; Malamut & Ryan, 2008; McKeith, 2008). Parkinson's disease is a common precursor. In both LBD and Parkinson's disease the substantia nigra of the midbrain is affected. This area is important for the control of movement, and so motor-related symptoms such as rigid limbs, changes in posture, and shuffled walking are common. These movement-related symptoms are often exacerbated by antipsychotic medicines which may be used in an attempt to treat hallucinations (Malamut & Ryan, 2008; Woodruff, 2008). Unlike AD or VaD, the symptoms of LBD fluctuate. However, the progression is similar in that the cognitive functioning gradually declines (Walker et al., 1999).

Lewy bodies are intraneuronal inclusions affecting the neocortex and subcortical areas of the brain. In Parkinson's disease alone, they are found in the subcortical part of the brain, the area used for

movement, balance, and consciousness. Whereas with LBD they are present throughout the brain. People with LBD also have plaques (as in AD) and some vascular changes. Together, these changes cause the cell death, brain shrinkage, and cognitive impairment typical of dementia (McKeith, 2008). There may be some genetic risk factors associated with the ApoE 4 gene LBD and Parkinson's disease runs in families, however the genetic mechanisms involved with LBD are less understood than in AD (Caselli, 2008).

As with AD and VaD, levels of acetylcholine are reduced in LBD. The transport and uptake of the neurotransmitter dopamine is also affected, a change which is associated with the movement-related symptoms. Therefore, treatment options include AChEIS for cognitive impairment, but also Levodopa to increase dopamine concentrations (McKeith, 2008). The effectiveness of Levodopa in LBD is questionable, with greater reduction of motor symptoms in people with Parkinson's disease alone (90-100% vs. 50-60% respectivley, Bonelli et al., 2004; Lucetti et al., 2010). Furthermore, the side effects can include exacerbated symptoms of dementia, particularly psychosis, rigid movement, as well as sleep disturbance (see section 2.2, Gulmann & Korsgaard, 1976; Melamed, 1979; Nausieda, Weiner, Kaplan, Weber, & Klawans, 1982).

In summary, dementia can be defined as a suite of symptoms caused by neuropathological deterioration associated with disease or vascular events. These symptoms vary between individuals dependent on the type and stage of dementia. However, across the board memory is impaired. Cognitive functioning, mood and personality are also affected, having implications for daily living and sense of self. The areas of the brain and neurotransmitters affected by dementia are also of importance for maintaining and regulating sleep (particularly the subcortical regions and cholinergic system).

2.2 Dementia-Related Sleep Disturbances

People with dementia are typically older and so more likely to experience the physiological changes and sleep disorders which increase in prevalence with ageing (as outlined in Chapter 1). Many of the sleep disturbances experienced by PWD are exaggerated forms of these age-related changes. Sleep disturbances have been observed in all types of dementia. The most profound changes are those observed in AD. This chapter focuses on the AD-related changes to sleep, which underpin the design of the final intervention study (Study 4).

Typical dementia-related sleep changes include reduced sleep quality and duration, more fragmented sleep/wake patterns and/or a shift in sleep timing, as well as exacerbated primary sleep disorders (Yesavage et al., 2003). Dementia-related sleep disturbances have been related directly to the neuropathological degeneration associated with the disease, but can also vary dependent on other intrinsic, extrinsic, and mediating factors which are reviewed below and form a conceptual framework presented in the summary of this chapter (*Figure 2.5*).

Sleep disturbances have been associated with a particular collection of dementia-related behaviours known as *sundowning*. Sundowning is poorly defined, but behaviours include wandering or seeking activities, agitation, hallucinations, and automatic behaviours such as checking doors and dressing and undressing (Bliwise, 2004; Song, Dowling, Wallhagen, Lee, & Strawbridge, 2010; Yesavage, et al., 2003). Those who sundown have been found to also exhibit a more anxious, disoriented, or aggressive temperament. This makes these behaviours particularly difficult for carers to manage (Khachiyants, Trinkle, Son, & Kim, 2011). Sundowning behaviours typically occur from dusk on throughout the night and are estimated to be prevalent in 20-30% of PWD (Bliwise, Yesavage, & Tinklenberg, 1992; Vitiello & Borson, 2001). Due to the non-specific definition of sundowning and variance in the temporal distribution of dementia-related behaviours, estimates of prevalence may not be reliable. *Figure 2.1* shows data from nursing staffs' observations of sundowning-type behaviours across the 24-hour day, amongst 18 PWD (Nowak & Davis, 2007).

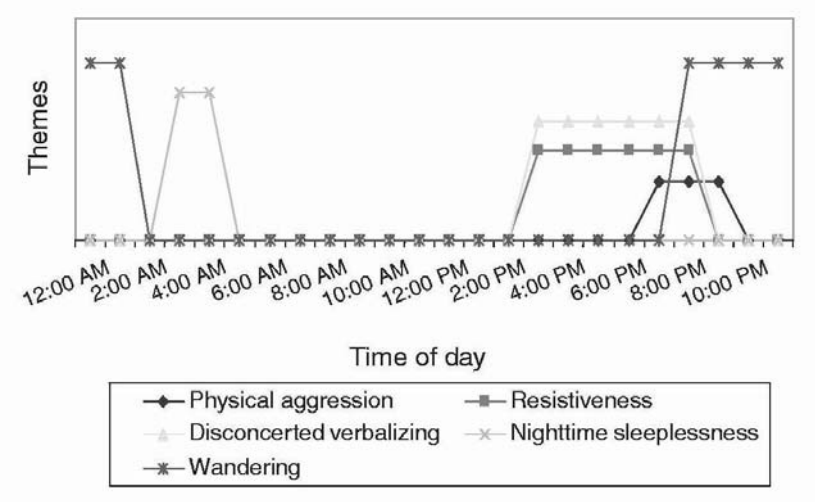


Figure 2.1. The temporal patterning of sundowning behaviours.

(Nowak & Davis, 2007, p. 258)

Dementia-related sleep disturbances have been associated with increased impairment of cognition and functioning, as well as poorer mood and aggressive behaviours, thereby contributing to poorer quality of life (Lawton, 1983, 1997; McCurry et al., 1999; Moe, Vitiello, Larsen, & Prinz, 1995; Moran et al., 2005). Sleep disturbances appear to be more likely and severe for those who were poor sleepers prior to dementia (Yesavage et al., 2002). The frequency and severity of sleep disruptions generally progresses with severity of the disease. However, a peak of disruptive sleep behaviours has been reported during the moderate stages, when many dementia-related behavioural disturbances are also more severe, prior to movement becoming more limited and the final stages of dementia (Chen, Borson, & Scanlan, 2000; Van Someren et al., 1996; Vitiello & Borson, 2001). As sleep becomes more disrupted, it becomes increasingly difficult for informal carers to provide continuous and safe care (see section 2.2.5). This is of importance as sleep disturbances, particularly night time activity, have been highlighted as amongst the behavioural predictors for PWD being moved into institutionalised care (Hope, Keene, Gedling, Fairburn, & Jacoby, 1998; Pollak & Perlick, 1991; Strain, Blandford, Mitchell, & Hawranik, 2003). If sleep can be addressed, this may extend the time that a PWD can continue to live at home with their family carer.

The estimated prevalence of sleep disturbances varies between studies, probably due to the different sampling strategies, methodologies, and definitions used for defining sleep problems. Many

research samples include heterogeneous groups of PWD with various types and severities of dementia, as well as a range of comorbid health conditions. Many studies focus on institutionalised settings (i.e., nursing homes or hospitals). These studies are particularly useful as they include larger samples, often with more rigorous or quantitative protocols than are possible in community-based samples (i.e. PWD still living in their own homes). However, the sleep of institutionalised PWD does not necessarily reflect the sleep of community-dwelling PWD, due to differences in age, stage of dementia, as well as environmental factors in the nursing home which could negatively affect sleep (Meadows et al., 2010). For example, in institutionalised settings, routine nursing checks and shift changes may govern bedtimes and exacerbate confusion around these times, which could contribute to sundowning symptoms and changes to sleep regulation (Bachman & Rabins, 2006; Schnelle et al., 1998).

A growing body of research is focusing on the sleep of community-dwelling PWD, with the global aim of creating a better understanding of sleep disturbances and treatment options for PWD *before* institutionalised care is considered necessary.

2.2.1 Changes to Sleep Architecture with Dementia

Polysomnography can be challenging to conduct with PWD. Symptoms of dementia can create practical and technical barriers to achieving a low-risk recording process with reliable results. The controlled laboratory environment and equipment necessary for polysomnography may cause to become confused, agitated, or distressed (Rose et al., 2011; Yesavage, et al., 2003). This creates an unpleasant situation for the PWD and compromises the validity of the sleep study. It is likely that a carer would need to accompany the PWD for the night and technical staff would require some skills and experiences with dementia-related symptoms in order to conduct the study in an appropriate manner. The few polysomnographic studies that have been successfully conducted have contributed pivotal information relevant to mapping the neurological origins of the sleep disturbances experienced by PWD. They have also helped to explain some of the cognitive impairment associated with dementia.

A key finding from polysomnographic studies in dementia is that there is a general slowing of EEG activity across all stages of sleep and wake. This slowed activity has been associated with the degeneration

of the cortical areas of the brain, including the hippocampus, ARAS, and cholinergic system (Avidan, 2007; Khachiyants, et al., 2011; Vitiello & Borson, 2001; Weinert, 2000). The constant slow wave activity is considered likely to create blurred boundaries between states of consciousness, causing inadequate transitions between states of wake, NREM, and REM. As the cortical activity of the brain is dampened by the slower firing, this increases the likelihood of confused awakenings and hallucinations in the night with little or no recollection by the individual. Sundowning behaviours have been related to weakened capacity to respond to an event or situation (e.g., a sudden noise or change in environment) due to the brain showing neurophysiological signs of sleep during states of physical wakefulness (Bachman & Rabins, 2006; Kavanau, 2001; Walker, et al., 1999).

Another consistent finding is the changes in sleep architecture, over and above those of normal ageing. Sleep quality is impaired due to reductions in the duration of SWS and REM sleep, as well as increased latency until REM sleep. There is also a reduction in the amount of Stage 2 sleep and its characteristic sleep spindles and K complexes. For example, Prinz, Peskind, and Vitaliano (1982) recorded EEG in 44 institutionalised people with mild-severe AD versus controls. Rapid eye movement sleep made up 17% of the total time in bed of controls, whereas those with mild AD had 14% REM and those with severe AD just 7%. Similarly, 9% of control participants' time in bed was spent in SWS compared to 5% of the mild AD group and 2% for those with severe AD. In addition, Prinz et al. reported that participants with AD spent up to 40% of their time in bed awake, compared to 21% of the age-matched controls. These changes are evident in the earliest stages of dementia (particularly of the AD type) and continue to worsen as the disease progresses. More frequent and abrupt awakenings have been associated with nightmares and sundowning behaviours during the night among PWD (Prinz, Vitaliano, et al., 1982; Vitiello & Borson, 2001).

Changes to PWD's sleep architecture are related to deterioration within the basel forebrain and VLPO which is typical with dementia, particularly of the AD type. This causes changes in the transmission of acetylcholine and adenosine and therefore problems with sufficient activation or suppression of the wake/NREM and REM states (Hofman & Swaab, 1994; Wulff, Gatti, Wettstein, & Foster, 2010). The reduction of architectural landmarks as well as diffused delta activity can make scoring the

polysomnographic sleep studies of PWD challenging. The background of slow wave activity could mean that time spent asleep is overestimated using traditional scoring rules (Rechtschaffen & Kales, 1968). Revised guidelines are necessary, and some studies simply differentiate between states of REM and NREM (Bliwise, 2004; Eeles, 2006).

There is consistent evidence that learning non-declarative tasks and emotional memory are associated with REM sleep, while learning declarative information is associated with SWS (Plihal & Born, 1997; Stickgold, 2005). Thus, the reduction in REM and SWS with dementia may contribute to the waking symptoms and cognitive decline. Rapid eye movement sleep is thought to contribute to learning and processing through bursts of theta activity and the reactivation of the brain regions used during initial learning, via an increase in acetylcholine. Conversely, SWS's rich delta waves originating from the hippocampal area, together with a reduction in acetylcholine, are considered beneficial for blocking interference and retaining information during the consolidation of declarative memories. The amount and density of spindles during Stage 2 sleep are also considered important with regards to the consolidation of both declarative and non-declarative memories. This is because sleep spindles are believed to facilitate neural communication between the hippocampus and the rest of the cortex, thus aiding the synaptic changes necessary for brain plasticity and long-term learning and memory (Buzsáki, 1998; Fogel & Smith, 2006, 2011; Gais, Mölle, Helms, & Born, 2002; Peigneux & Smith, 2011). Based on this previous literature, *Figure 2.2* was drawn to give a schematic representation of declarative and non-declarative learning, and how sleep states augment the learning processes and plasticity required for memory consolidation.

As dementia progresses, further neuropathological degeneration, as well as increased behavioural sleep disturbances, mean that the process of memory consolidation during sleep is further affected. In turn, more dementia-related symptoms are present during waking, creating a vicious cycle for PWD. This conundrum highlights the importance of understanding and treating the sleep disturbances of this population.

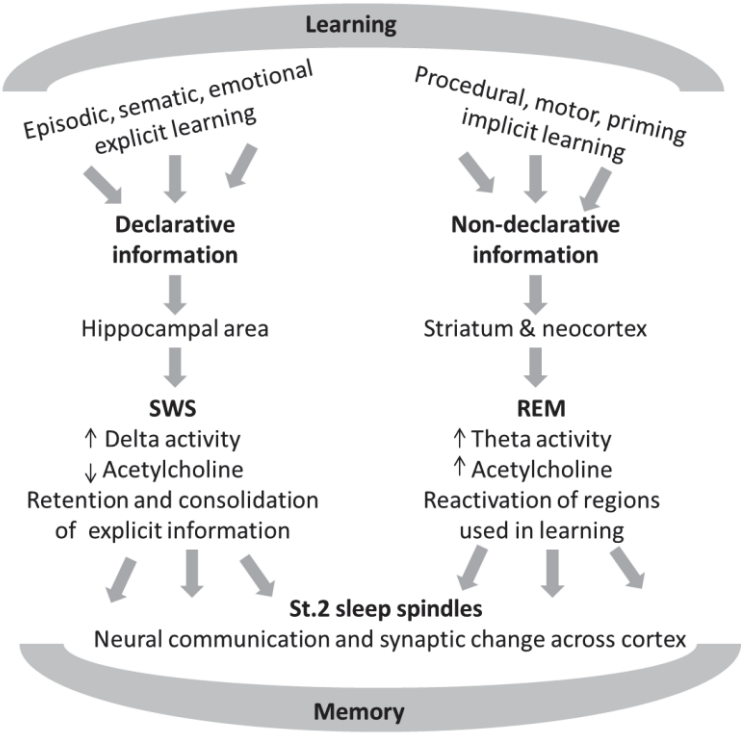


Figure 2.2. Categorisation of learning processes and how sleep contributes to consolidated memory.

2.2.2 Changes to Sleep Regulation with Dementia

Previous studies have consistently shown that PWD have increased awakenings and time awake during the night (McCurry, et al., 1999). Due to this fragmented nature of sleep, many PWD do not get more than six hours sleep at night (Yesavage, et al., 2003). Previous studies of people in late stages of AD report sleep efficiencies of around 60% (Vitiello & Borson, 2001). More awakenings during the night create disruption to the expression of REM and SWS and so account for some of the changes noted above.

Poorer sleep efficiency at night also increases the likelihood of daytime sleepiness and napping. Although napping is common amongst PWD, the quality of this sleep is poor and unlikely to compensate for the REM or SWS reduction during night time sleep. Daytime sleep may also have detrimental effects on sleep regulation as it prematurely reduces the homeostatic drive for sleep (Achermann & Borbély, 2011). This is a notable problem as daytime sleeping is seldom considered an issue by carers (Bonanni et al., 2005; Eeles, 2006; J. Lee et al., 2007; McCurry, et al., 1999; Prinz, Peskind, & Vitaliano, 1982; Song, et al., 2010). Changes to sleep timing have been related to increased likelihood of sleep disorders

(see section 2.2.3). More importantly, degenerative changes in the circadian timing system occur with dementia and contribute to fragmented sleep/wake patterns.

Few studies have attempted objective recording of circadian rhythms with PWD. The highly controlled protocols required may increase the chance of participants becoming agitated and withdrawing from the research (Harper, 2010). Also, the synthesis of melatonin is affected in dementia due to a reduction of receptors within the SCN, compromising its reliability as a circadian marker (Wu, et al., 2007). Studies typically have to use temperature or movement as indicators of circadian phase and stability. Less rigorous protocols have been used for PWD compared to other populations. These typically involve fewer restrictions around participants' exposure to factors which could mask the expression of their circadian rhythms (Harper et al., 2001; Satlin, Volicer, Stopa, & Harper, 1995). Although such protocols affect the reliability of estimating circadian phase and amplitude, these modifications are considered necessary for recruiting adequate sample sizes and for conducting ethical research with this population. Studies that have measured the circadian rhythms of PWD, report mixed results. However, it appears that more than half of PWD have some kind of circadian abnormality (Motohashi, Maeda, Wakamatsu, Higuchi, & Yuasa, 2000).

Compared to healthy older adults, phase delays of 2-3 hours in the circadian temperature rhythm and in sleep timing have been reported in PWD, particularly those with AD. These delays are most evident in constant routine protocols using full unmasking (Harper et al., 2005). Phase delays appear to be more extreme in people who also exhibit symptoms of sundowning behaviour (Harper et al., 2004; Satlin, et al., 1995; Volicer, Harper, Manning, Goldstein, & Satlin, 2001). Other studies report no differences or phase advances of sleep timing (Harper, et al., 2001; Sloane et al., 2007), which might reflect an exacerbation of the progressively early sleep times associated with normal ageing.

Differences between studies may be explained by differing methodologies and/or differences in the types and stages of dementia of the participants and their comorbidities (Bliwise, 2004). For example, Sloane et al. (2007) found that those with mild-moderate stages of dementia were more likely to have advanced circadian phase compared to participants with severe or very severe dementia. Variations between living environments and schedules are likely to be important. Those living in institutionalised care

may be more likely to follow a routine schedule in which they receive less exposure to phase-delaying bright light later in the day, eat earlier in the evening, and have earlier bedtimes dependent on the shift changes of staff (Ellmers, Arber, Luff, Eyers, & Young, 2013). These factors would predispose them to falling asleep earlier in the evening. Conversely PWD living at home might be encouraged not to get up too early in the morning, have limited exposure to phase advancing morning light, and attempt to stay up in the evening with family carers, or have bedtime schedules that slip behind due to the high volume of care-related duties in the evening. These factors would predispose them to falling asleep later in the evening.

A more consistent finding across studies is a reduction in the amplitude and robustness of circadian rhythms with dementia (Harper, 2010; Witting, Kwa, Eikelenboom, Mirmiran, & Swaab, 1990). Sleep regulation often becomes uncoupled from environmental time cues and more random , by comparison with healthy ageing. Some studies have calculated measures of circadian stability and variability to help describe dementia-related sleep disturbances. *Interdaily stability* is calculated from continuous data over several weeks (e.g., continuous monitoring of activity or core body temperature), with higher values representing more day-to-day rhythm stability. The interdaily stability of the sleep/wake pattern of PWD has been found to be significantly lower than that of older people without dementia ($p = 0.11$; Witting, et al., 1990). As the severity of dementia increases, the sleep/wake pattern becomes less defined or consistent (Carvalho-Bos, Riemersma-van Der Lek, Waterhouse, Reilly, & Van Someren, 2007; Harper, et al., 2004). One study measuring the circadian melatonin rhythm recorded a reduced amplitude of this rhythm in PWD (Uchida, Okamoto, Ohara, & Morita, 1996), which might contribute to the observed changes in sleep/wake regulation. *Intradaily variability* is another factor often calculated to represent the variability in the amount of sleep and wake per hour across the 24-hour day, with higher values indicating more consolidated sleep/wake timing. Witting et al. found that PWD had significantly less consolidated periods of sleep and wake across the 24-hour day compared to healthy older people ($p = 0.02$). Studies using actigraphy monitoring of nursing home residents found that those in more advanced stages of dementia have no complete hours during the day without bouts of sleep, and no complete hours during the

night without bouts of wake (see Figure 2.3; Jacobs, Ancoli-Israel, Parker, & Kripke, 1989; Pat-Horenczyk, Klauber, Shochat, & Ancoli-Israel, 1998).

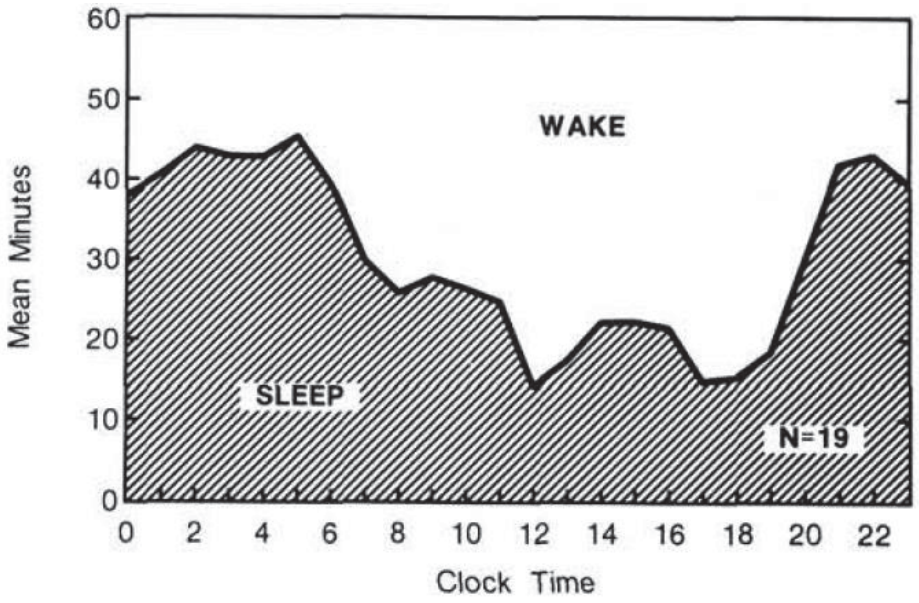


Figure 2.3. Average amount of time spent sleeping in each hour across a 24-hour period.

Derived from 24 hours of actigraphy data from 19 nursing home residents with moderate-severe stages of dementia (Jacobs, et al., 1989, p. 354).

The "circadian dysrhythmias" outlined above have been associated with neuropathological and physiological changes, as well as reduced or inappropriate timing of environmental time cues (these are summarised in *Figure 2.4*). Studies using autopsies (Wu et al., 2003; J. Zhou, Hofman, & Swaab, 1995) and immunoreactivity (Swaab, Fliers, & Partiman, 1985) have found that the SCN, hypothalamus, and associated areas visibly shrink with dementia, reducing in volume and neural activity. The senile plaques and neurofibrillary tangles associated with AD are evident in the SCN (Stopa et al., 1999), and people with AD have been estimated to have just one-third of the expression of vasopressin of healthy older people (Liu et al., 2000; Wu & Swaab, 2007). These changes indicate that there is reduced activity and functionality with regards to the transcription and translation of the clock genes within the SCN. Research using rodents supports the notion that mutations of clock genes are associated with dysrhythmia of circadian timing commonly observed in ageing and dementia (Oster, Baeriswyl, van der Horst, & Albrecht, 2003). Neuropathological changes have been noted to occur at a younger age and more dramatically for people

with AD compared to healthy ageing. Furthermore, they are evident prior to noticeable cognitive impairment (Hoogendijk et al., 1996; Swaab, et al., 1985; Wu et al., 2006; Wu & Swaab, 2007). Accelerated degeneration in the number and activity of cholinergic and neroadrenergic neurons within the ARAS and brainstem further contribute to dysrhythmia within the SCN (Vitiello & Borson, 2001; Weinert, 2000). These patterns of degeneration mean that the neural pathways become less functional and the ability of the SCN to regulate sleep-promoting and wake-promoting areas of the brain is affected, leading to the loss of consolidated sleep at night and consolidated wakefulness during the day.

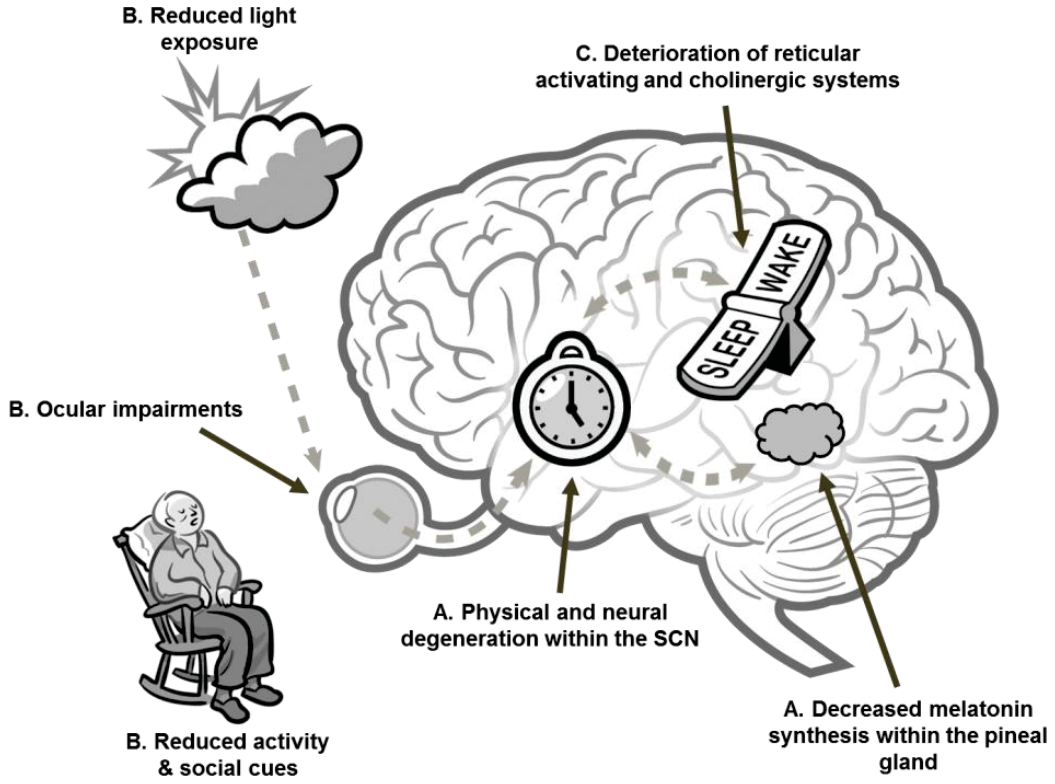


Figure 2.4. Factors associated with poor sleep regulation for people with dementia.

A. The intrinsic age and dementia-related degeneration related to the suprachiasmatic nucleus (SCN), and metabolism of melatonin predisposes people with dementia to irregular sleep/wake timing. B. Reduced exposure to environmental time cues as well as age and dementia-related impairment to the eye and retino-hypothalamic tract precipitate circadian dysrhythmia. C. Deterioration of the neural systems responsible for the transitions between sleep and wake contribute to less distinct states of consciousness, poor sleep architecture, and sundowning behaviours.

Reduced melatonin synthesis in the pineal gland, as well as a degeneration of the melatonin receptors within the SCN, also appear to be greater in AD than in normal ageing. The melatonin levels of people with AD are reported to be about one fifth of those of age-matched controls (Liu, Zhou, Van Heerikhuize, Hofman, & Swaab, 1999; Wu & Swaab, 2007). There are no clear pathological differences between the pineal glands of PWD and those of healthy older people. The changes in melatonin synthesis are thought to be related to the disconnection between the pineal and the SCN, as is evident from the earliest, preclinical stages of AD and likely associated with a disruption of clock genes (Wu, et al., 2006). This highlights the vulnerability of the circadian timing system to dementia, and suggests that reduced melatonin could be a useful marker for disease onset (Liu, et al., 1999; Wu & Swaab, 2005; Wu, et al., 2007; Wulff, et al., 2010). Reactivating the connection between the SCN and pineal has also been highlighted as an important aim for treating circadian rhythm disturbances associated with dementia. In principle, this can be achieved through timed bright light exposure and/or timed physical exercise, and/or timed doses of exogenous melatonin (see Chapter 3).

As noted in section 1.2, synchronisation of the circadian timing system and the sleep/wake cycle to the day/night cycle is achieved through the sensitivity of the SCN circadian pacemaker to light, via dedicated retinal cells and the RHT (Czeisler & Buxton, 2011; Duffy & Czeisler, 2009). The visual impairments typical of normal ageing are often exacerbated for PWD. There are also specific ocular impairments associated with AD which contribute to less light being absorbed by the retina, and degeneration to the RHT itself has also been detected (Bradford, 2004; Valenti, 2004). It is hypothesised that PWD may require more bright light exposure than older people without dementia in order to have the same synchronising effect on the SCN pacemaker.

Unfortunately, as well as the pathological changes affecting the mechanism for light regulation of the circadian timing system, PWD may have reduced opportunities for adequate light exposure compared to older people without dementia (Shochat, et al., 2000). Again, this appears to become more of an issue with the progression of age and dementia and finally with moving into institutionalised care. Community-dwelling PWD in the USA have been found to have about half of the light exposure of healthy older people (approximately 30 minutes of light brighter than 2,000 lux per 24-hours; Campbell, et al., 1988;

McCurry, 2000). Those in the severe stages of dementia who live in institutionalised care in the USA have been found to have an average of just a few minutes of light brighter than 1,000 lux during the day and seldom have exposure to light over 2,000 lux (Ancoli-Israel et al., 1997; Shochat, et al., 2000). In the Netherlands, Van Someren et al. (1997) measured an average light intensity of 436 lux (range = 93-1,417 lux) in the living areas of a dementia institution between 9-11am. Together, these studies suggest that PWD rarely receive adequate light exposure to maintain entrainment of the SCN circadian pacemaker to the 24-hour cycle of light and darkness (Van Someren, Riemersma, Raymann, & Swaab, 2005).

Reduced exposure to bright light could be related to impaired physical or mental health affecting a PWD's ability to go outside or adjust lighting, constraints on care providers being able to initiate outside activities, poor housing architecture with regards to natural light, as well as a lack of motivation, or feelings of discomfort when exposed to light. The lack of *routine* exposure to bright light is a key part of the problem. To maintain synchrony between the internal SCN and the external day/night cycle, ideally bright light exposure needs to occur at the same time every day (Wirz-Justice, et al., 2008). Due to the barriers noted above, routine exposure to light can become increasingly difficult to achieve for PWD. As sleep timing becomes more disrupted, PWD are more likely to sleep in the day and be awake at night and consequently be exposed to light at times of the day which further contribute to antisocial sleep timing (Wu & Swaab, 2007). Inadequate light exposure and ocular degeneration have also been associated with misperceptions of the time of day; and confused, sundowning-type behaviours (Khachiyants, et al., 2011).

People with dementia are typically not as socially or physically active as older people without dementia, limiting exposure to non-photic time cues that can contribute to synchronising the circadian timing system. Reduced physical activity could be related to mental or physical impairments, a lack of motivation, or requirements regarding support and/or safety to reduce risk of injuring or getting lost (Laurin, Verreault, Lindsay, MacPherson, & Rockwood, 2001; Penrose, 2005). These factors also influence social interactions. A lack of public knowledge and acceptance of PWD's symptoms could contribute to feeling stigmatised or less welcome in social situations. As the ability to cognitively engage in some activities becomes impaired, this can make taking part in such activities confusing or frustrating. Consequently PWD are less likely to initiate activities and often require support which may not always be

available (Miranda-Castillo et al., 2010; Orrell, Butler, & Bebbington, 2000; S. Sullivan & Richards, 2004). These factors mean that PWD are more likely to spend their time doing sedentary activities, alone and indoors. This creates a situation in which they might become bored or agitated and more daytime napping occurs. Lawton Moss and Duhamel (1995) conducted a community-based study using interviews with 116 carers to recollect the activities of the older people in their care (including both physical and/or mental impairments, accounting for every 15 minutes of the previous day). They found that older impaired people were reported to be spending 72% of their day in a passive state (of either rest, watching television or listening to the radio, or receiving care assistance), and that they also spent more than half of their day alone.

A reduced or desynchronised SCN circadian pacemaker cycle has implications for cognitive functioning due to the associated disturbances in sleep quality at night and daytime sleepiness (Wu & Swaab, 2007), as well as the possibility of attempting to perform tasks at a time that is not conducive with biological timing (Johnson et al., 1992; X. Zhou, et al., 2011). Circadian dysrhythmia has also been associated with depressed mood (Boivin et al., 1997), which suggests that unstable sleep timing is likely to contribute to some of the waking symptoms of dementia.

In summary, neuropathological, ocular, and environmental factors all contribute to the weakened circadian timing system and phase disturbances that have been documented with dementia. For the individual, these disturbances manifest as increased daytime napping, difficulties getting to sleep and staying asleep at night, as well as increased cognitive impairment during waking. Misaligned sleep timing has been associated with the onset of sundowning symptoms. The peak timing of these behaviours has been found to vary from early afternoon to late night and they are associated with the weakened boundary between the states of sleep and wake, as well as the disrupted phase and lower amplitude of circadian rhythms.

2.2.3 Primary Sleep Disorders and "Problem Sleep" in Dementia

Reliable prevalence estimates are not available for primary sleep disorders among PWD, mainly due to complications associated with polysomnographic and laboratory-based sleep recording. While there

are some studies using objective recordings, symptoms of primary sleep disorders with dementia are often gathered from routine observations and diaries maintained by informal family carers or nursing staff. Although not as reliable as polysomnography, observations are considered one of the more clinically-relevant and ethical methods for monitoring sleep of PWD in more severe stages of disease and living in institutionalised care. Observations are a non-invasive method, and privacy is not breeched any further than is necessary for routine care requirements (Bliwise, 2004; Eeles, 2006). For community-dwelling PWD, informal carers are typically responsible for the data collection, so long-term observations are considered impractical or burdensome. Retrospective questionnaires are often used. These are also deemed non-invasive and can gauge the prevalence of typical symptoms of sleep disorders.

Questionnaires specifically designed to capture the sleep disturbances related to dementia have been developed. The Sleep Disorders Inventory (SDI; Tractenberg, Singer, Cummings, & Thal, 2003) was established to record the frequency and severity of dementia-related sleep problems using carers' reports. When initially used in a trial of supplemental melatonin, C. Singer et al. (2003) validated the results from the SDI against actigraphic measures of sleep duration, sleep efficiency, as well as the proportion of total sleep occurring at night, making this an appealing, non-invasive measure (see section 3.1.2). Measures used to record sleep disturbances in the general population (e.g., the PSQI; Buysse, et al., 1989) have been used amongst older people and PWD and are typically completed by family members or nursing staff on their behalf (e.g., B. Carpenter, Strauss, & Patterson, 1996; McCurry, et al., 1999). See Chapter 4 for more details regarding these scales.

Carers are considered likely to have better recollection of sleep and rise times as well as behavioural symptoms compared to the PWD. Limitations of proxy questionnaires are that they are often completed retrospectively and only the sleep problems that carers are aware of are recorded (e.g., if their own sleep is disturbed because of the behaviour; Eeles, 2006; McCurry, Vitiello, Gibbons, Logsdon, & Teri, 2006; Tractenberg, Singer, & Kaye, 2005). Furthermore, and of more ethical importance, proxy reports lack personal insight into the PWD's sleeping experience (Cotrell & Schulz, 1993). Previous research using interviews with PWD in institutionalised care show that such methodology is not only possible, but can offer a rich understanding of the individual's sleep experiences (W. Martin & Bartlett,

2007), however few such studies have been conducted (see section 2.3). Regardless of the methodological barriers, there is a consensus that the frequency of primary sleep disorders, as well as general reports of "problem sleep", are higher for PWD than amongst older people without dementia (Boeve, 2008).

Sleep apnoea.

In a study of community-dwelling Americans, Hoch et al. (1986) found that the prevalence of sleep apnoea (defined as ≥5 respiratory events per hour) was significantly higher for older people with AD compared to those without AD (41.7% vs. 5.4% respectively, $p < 0.001$). Studies of PWD living in institutionalised settings report an even higher prevalence (63%; Ancoli-Israel, Klauber, Butters, Parker, & Kripke, 1991). Those with more severe sleep apnoea also had more severe dementia. The prevalence of the full sleep apnoea syndrome is more difficult to estimate due to less reliable reports of daytime sleepiness.

The higher prevalence of sleep apnoea with dementia has been related to the degeneration of respiratory neurons in the brainstem and supramedullary respiratory pathways. Degeneration to these areas cause PWD to have less efficient responses to airway obstructions and respiratory arousals during sleep (Avidan, 2007). Furthermore, antipsychotics often used to treat the mood and behavioural symptoms of dementia can exacerbate sleep disordered breathing. This is due to their sedating effects influencing the patency of the airway, and their effects on the serotonergic system having a negative impact on the respiratory function (Shirani, Paradiso, & Dyken, 2011). Those with VaD are considered at greater risk than other types of dementia due to increased likelihood of cerebrovascular comorbidities (Avidan, 2005).

Previous research suggests that the brief arousals and awakenings that sleep apnoea causes could contribute to sundowning behaviours and confusion for PWD (Bliwise, 1994; Bliwise, Yesavage, Tinklenberg, & Dement, 1989; Gehrman et al., 2003). Sleep apnoea has also been related to reduced cognitive functioning, attention, and waking performance. This is due to consistent sleep fragmentation, disruptions to REM and SWS, as well as hypoxemia, and likely daytime sleepiness (Ancoli-Israel, Klauber, et al., 1991; Cooke, Liu, et al., 2006; Hoch et al., 1989; Weaver & George, 2011). Therefore, it can be speculated that with treatment of sleep apnoea, some cognitive functioning may be restored. However

many PWD with sleep apnoea are untreated due to a lack of formal diagnosis and complications with establishing PWD on therapy.

Restless legs syndrome and periodic limb movements of sleep.

Restless legs syndrome and PLMS are probably more prevalent with the presence of dementia. Reliable prevalence estimates are unavailable, due to the subjective nature of RLS, the discreet nature of PLMS, as well as the cognitive impairment associated with dementia (Bliwise, 2006; Boeve, 2008; Petit, Montplaisir, & Boeve, 2011). These conditions are often identified behaviourally for PWD, for example, rubbing or moving legs in the evening, as well as more agitated behaviours at night (Rose, et al., 2011).

Those with LBD are considered to be more likely to experience RLS and PLMS due to the affected dopaminergic system, which is associated with the control of movement. Previous studies using brain scanning technology have identified similar dopamine-related deficiencies within the same areas of the brain for both PWD who wander and people with RLS (Meguro, Yamaguchi, Itoh, Fujiwara, & Yamadori, 1997; Turjanski, Lees, & Brooks, 1999). It is speculated that RLS might manifest as the wandering behaviours commonly observed in PWD who sundown in the evenings (Bliwise, 2006, 2011b). With careful treatment of RLS, some of the dementia-related behaviours and agitation may also reduce (Bliwise, 2006; Boeve, 2008; Rose, et al., 2011). In the case of LBD or Parkinson's disease, sleep disturbances can also manifest as rigidity in the night, or REM behaviour disorder (a disorder characterised by dream enactment and vocalisation during sleep). Those with LBD may also experience frightening nightmares or hallucinations at night, making their confused awakenings disruptive for both them and caregivers. These behaviours have also been related to changes in dopamine, as well as adverse effects of medications causing higher than normal rates of muscle activity during sleep (Gagnon, et al., 2002; Hoque & Chesson A. L, 2010).

Insomnia / "poor sleep".

It appears that insomnia becomes more problematic with dementia. It is difficult to gain reliable prevalence data due to PWD's reduced ability to self-report subjective sleeping problems, or give a reason for why they are awake at night. Previous studies gathering data on sleep disturbances of PWD living in the community may be confounded by carers being unaware of PWD's lack of sleep, if sleeping in separate

rooms or being asleep themselves. Furthermore, as concluded in section 1.5, problem sleep may be underreported by older people in general. For PWD it is likely that underreporting of sleep problems is even more common, due to the cognitive symptoms of the disease taking precedence in clinical consultations, or carers not considering symptoms such as excessive daytime sleeping to constitute a problem (McCurry, et al., 1999; Molano & Vaughn, 2014; Tractenberg, et al., 2005).

Perceptions of disturbed sleep are shaped by distal factors, such as social and cultural values and expectations surrounding sleep and ageing, as well as being influenced by the environment, and demographic factors such as socioeconomic position and level of education (Grandner et al., 2013; Paine & Gander, 2013; Paine, et al., 2007; Venn & Arber, 2011). These factors also play a role in how PWD and their carers perceive and cope with the dementia-causing disease and its symptoms (Garratt & Hamilton-Smith, 1995). It is the complex interaction between these distal factors with the intrinsic context of the PWD's disease pathology, comorbidities, and medications, which contribute to their not only experiencing sleep disturbance, but also considering that experience to be abnormal. Alternatively, some carers may overestimate their family member's sleep disruptions. Such misperceptions could be related to how carers critique and manage dementia-related behaviours, as well as how much their own sleep is disrupted (McCurry, et al., 2006).

Miu and Szeto (2012) found that 84.8% of informal carers in China reported some kind of sleep disturbance for their partner with dementia, however these disturbances were not necessarily considered problematic or frequent. Studies from the USA and Europe estimate that the prevalence of "problem sleep" is between 40-50% for PWD living in the community (Altena, Ramautar, Van Der Werf, & Van Someren, 2010; Boeve, 2008; B. Carpenter, et al., 1996; McCurry, et al., 1999; Tractenberg, et al., 2003; Vitiello & Borson, 2001). Symptoms often include those characteristic of insomnia and have been associated with cognitive decline. When PWD are given the opportunity to self-report sleep problems using the PSQI, they tend to have higher scores than healthy older people (Boddy et al., 2007). This is particularly the case for PWD with Parkinson's disease (mean = 6.1, SD = 3.9) or LBD (mean = 7.5, SD = 4.4) rather than AD (mean = 2.9, SD = 2.9). Poorer self/ proxy-rated sleep is likely related to increased movement and behavioural sleep disturbances which can be particularly problematic for people with Parkinson's or LBD

(Bliwise et al., 1995; Boddy, et al., 2007). Due to the more severe detriments to memory from the early stages of AD, it could also be speculated that these PWD might simply not remember if their sleep was affected recently. This highlights the importance of collecting dual reports concerning PWD's sleep (i.e. from dementia carers as well as PWD, see section 2.3).

For PWD, insomnia is often classed as a sleep disorder secondary to their health comorbidities. Wakefulness at night is likely to be associated with circadian dysrhythmia as well as increased arousals and awakenings caused by sleep apnoea, RLS and PLMS. As noted in section 1.3.3, there are many medical comorbidities and life events associated with ageing that increase the likelihood of insomnia (Ruiter, Vander Wal, et al., 2010; Wolkove, et al., 2007). These are exacerbated with dementia. For example, it is estimated that almost 17% of people with AD and 60% of people with VaD also have depression (Cummings & McPherson, 2001). Increased depression has been associated with the social stigma of having the disease, changes to family roles and relationships, financial stress, and the amount and quality of dementia-related support (Baikie, 2002; Judge, Menne, & Whitlatch, 2010). When PWD do not have comorbid depression, the prevalence of proxy-reported sleep problems is much lower. Rebok, Rovner, and Folstein (1991) estimated just 13%. However, many do have both, and depression has been associated not only with symptoms of insomnia but also symptoms of sundowning, due to the variability of mood across the day which contributes to agitation or aggression (Bachman & Rabins, 2006; Moran, et al., 2005). This is a complicated by the bi-directional relationship between sleep and mental health (Kondratova & Kondratov, 2012). It is speculated that treating sleep disturbances could improve symptoms of depression and vice versa.

Increased likelihood of comorbidities often means increased use of medications and polypharmacy. Medications used to treat symptoms of dementia as well as comorbid conditions have been found to increase wakefulness at night. Acetylcholineinesterase inhibitors and selective serotonin-reuptake inhibitors (used to treat depression) have wake-promoting effects on the cholinergic system. They can decrease the ability to get to sleep, especially if taken at night. But they also increase the expression of REM sleep, which has the potential to be advantageous for PWD. However, results are inconclusive, and many PWD using AChEI's also experience an increase in nightmares due to the influence on REM

(Bliwise, 2004; Inglis, 2002; Kitabayashi et al., 2006). Levodopa, used to manage Parkinsonian-type movements, has inhibitory effects on REM sleep and increases the possibility of extrapyramidal symptoms such as involuntary movements. Conversely, antipsychotics and hypnotics, used to treat dementia-related behaviours, have sedating effects. Therefore they increase the likelihood of drowsiness and falls at night, as well as daytime sleepiness (Dauvilliers, 2007; Molano & Vaughn, 2014). Antipsychotics and hypnotics have also been shown to have paradoxical effects, exacerbating the PWD's existing pathology and increasing sundowning symptoms at night, especially if being taken in combination with other medications (Khachiyants, et al., 2011). People with dementia might also face withdrawal from recreational substances such as caffeine, alcohol, or tobacco, due to limitations created by informal carers or nurses as well as their living environment. Such withdrawal can contribute to a situation in which the homeostatic drive is affected, and insomnia and sundowning behaviours are more likely (Kim, et al., 2009; Landolt, 2008).

Poorer sleep hygiene could be responsible for some of the problem sleep experience with dementia. Lack of knowledge or practice around sleep timing and behaviours could contribute to insomnia. Sleep hygiene might become worse with dementia due to lack of awareness of time of day, inability to get in and out of bed without assistance, eating or taking medications at inappropriate times of day, as well as the changes in exposure to environmental time cues mentioned in Chapter 1. As with all age groups, sleep hygiene needs to be taken into account, as well as treating the underlying causes of insomnia, before considering medications (Dauvilliers, 2007; McCurry, Gibbons, Logsdon, Vitiello, & Teri, 2003; Vitiello & Borson, 2001).

2.2.4 Summary

The exacerbation of disturbed sleep and sleep disorders with dementia has been associated with intrinsic factors, including accelerated age-related changes and comorbidities, and neuropathological degeneration. Sleep problems are mediated by degenerative changes in the circadian timing system, reduced exposure to environmental time cues, poor sleep hygiene, use of medications, and influences of the sleep of others. Overarching these factors is the impact that distal, extrinsic, influences have on experiencing and reporting sleep problems. These include social and cultural factors which are considered to contribute to perceptions and expectations around sleep and disease. After reviewing the literature, a

conceptual framework describing the factors affecting sleep and likelihood of carers reporting sleep problems was constructed (*Figure 2.5*). The presentation of this was based on a transactional model developed by Sadeh and Anders (1993) for infants' sleep.

2.2.5 Sleep of those Caring for a Family Member with Dementia

Previous literature and the results from Study 2 highlight that providing informal care creates a situation in which older people are more likely to experience disturbed sleep and report feeling tired in the daytime (Arber & Venn, 2011; Gibson, et al., in press; Maher & Green, 2002). It appears that sleep is particularly affected for older people providing dementia-related care. Recent literature reviews estimate that more than 50% of carers of PWD report some kind of sleep disturbance, usually related to symptoms of insomnia and daytime sleepiness (McCurry, et al., 2007; Peng & Chang, 2013). By comparison, in the broader sample of informal carers presented in Study 2, 24.4% reported being dissatisfied with their sleep (Gibson, et al., in press).

Many previous studies have used sleep diaries and questionnaires to obtain data regarding the sleep of those caring for PWD. These indicate poorer self-reported sleep compared to non-carers. For example, Von känal et al. (2010) found that carers of someone with AD had an average PSQI score of 7.8 (SD = 4.1) compared to 6.1 (SD = 3.5) in older non-carers. Castro et al. (2009) reported a PSQI score as high as 10.4 (SD = 3.5). As this sample were female, they may have been more likely to have insomnia-type symptoms than a sample including males (Arber, et al., 2009). The PSQI data from the study of Castro et al. showed that dementia carers took significantly longer to fall asleep (50 minutes versus 14, $p < 0.05$), and had more disturbances to their sleep at night compared to the non-carers. Daytime sleepiness is a common complaint amongst dementia carers (Chiu et al., 2014; Peng & Chang, 2013; Rowe, et al., 2008). Some studies using actigraphy and polysomnographic data corroborate these reports (Peng & Chang, 2013). A cross-sectional study found that carers of a family member with AD had reduced and more variable sleep duration at night (mean = 6.1 hours, SD = 1.2) compared to non-carers (mean = 6.5 hours, SD = 0.9, $p = 0.049$). Carers of someone with AD had a significantly lower and more variable average sleep efficiency of 76.9% (SD = 11.2) compared to 81.7% for non-carers (SD = 9.2, $p = 0.04$; Von Känel et al., 2006). However, other studies, including Castro et al. (2009), found negligible

differences between the polysomnographic or actigraphic sleep of carers and non-carers, despite increased self-reported sleep disturbance among carers (McKibbin et al., 2005; Pollak & Stokes, 1997). This could be due to the psychosocial factors that have been associated with the sleep disturbances of dementia carers, and the particular traits of the care recipient's dementia (McCurry, Gibbons, Logsdon, Vitiello, & Teri, 2009; McCurry, et al., 1999). Carers' attitudes, feelings of general happiness, and positive experiences, appear to be more strongly related to whether they report a sleep problem or not, rather than objective/physiological sleep changes (McCurry, et al., 2006; R. Moore et al., 2011; Rowe, et al., 2008; Von Känel et al., 2014).

Due to the nature of dementia, informal carers are more likely to spend long hours providing care and are required to assist with many activities of daily living, compared to carers of someone without dementia (Ory, Hoffman Iii, Yee, Tennstedt, & Schulz, 1999). The amount of assistance required is dependent on the type and stage of dementia. However, there is generally a marked increase in carer duties and vigilance in the late afternoon and evening. This often includes supporting eating, household chores, and preparing for bed, which coincides with the timing of sundowning behaviours for many PWD. Dementia-related changes in sleep timing become an issue with regards to synchronising with that of the carer, and many carers end up adapting their own sleep routines to match those of the care recipient (Kotronoulas, et al., 2013). The restricted lifestyle of carers could also have an impact on their exposure to adequate and routine time cues (e.g., light and physical activity), their physical health (e.g., increased body mass and risk of cardiovascular disease), as well as increased feelings of isolation (McCurry, et al., 2007; Von Känel, et al., 2010; Von Känel, et al., 2006) . Together these factors are likely to affect carers' sleep hygiene practices and contribute to symptoms of insomnia and circadian dysrhythmia.

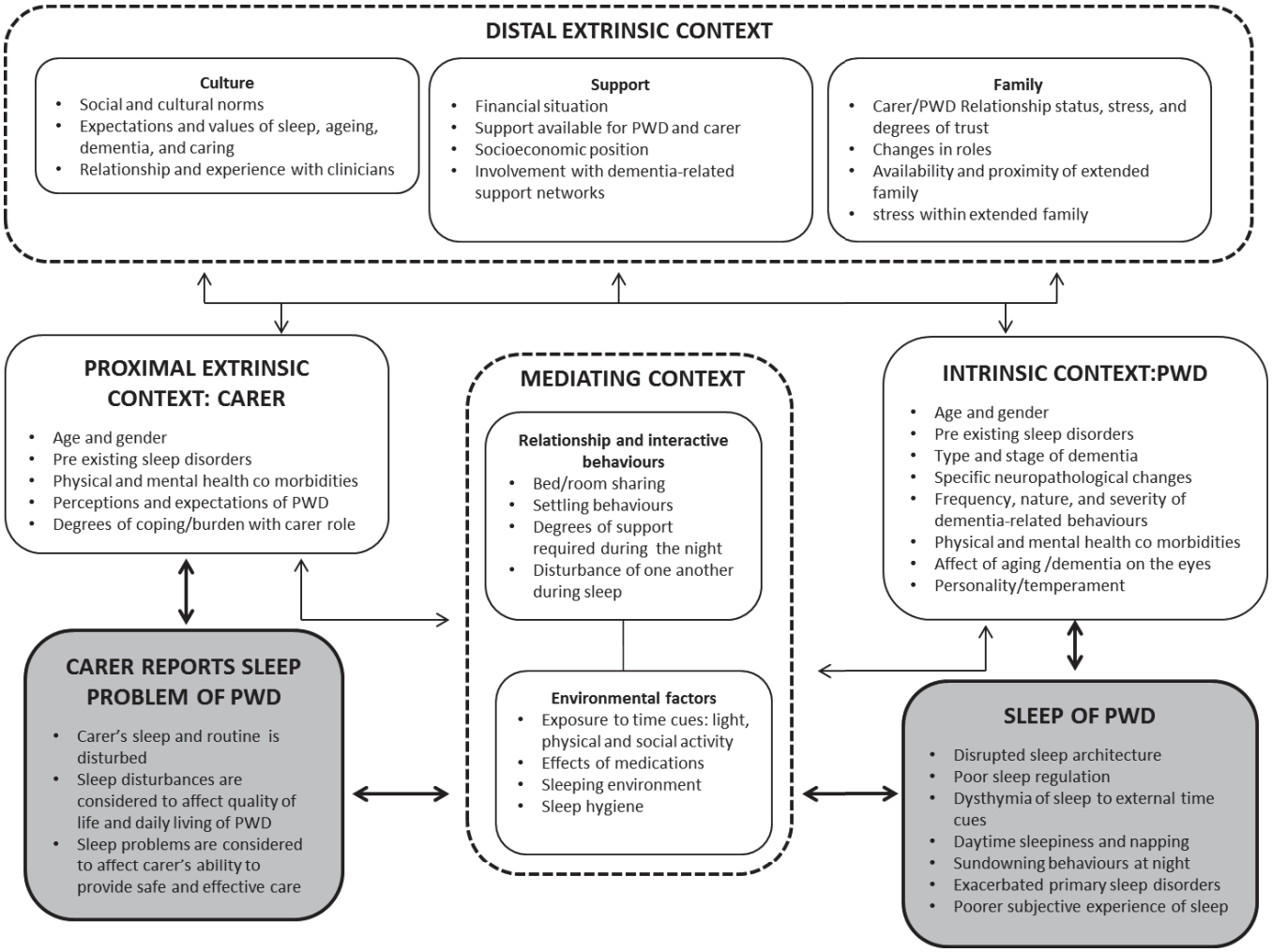


Figure 2.5. Conceptual framework showing the intrinsic and extrinsic factors and mediating context associated with the presentation and reporting of PWD's sleep problems.

Carers' sleep appears to be most affected by disturbances in the night. Creese, Bédard, Brazil and Chambers (2008) found that 63% of AD carers reported that their sleep was disturbed by the sundowning-type activities of the care recipient during the night, as well as by their general restlessness and trips to the bathroom. Sundowning behaviours have been highlighted as particularly difficult for carers to psychologically manage (Logsdon et al., 1998; Tractenberg, et al., 2003). Many dementia carers experience subjective burden related to anger and frustration with their family member and/or their situation, as well as general symptoms of anxiety, stress, and depression (McCurry, Pike, Vitiello, Logsdon, & Teri, 2008; Wilcox & King, 1999). Such factors have been identified as independent risk factors as well as outcomes of poor sleep, and also contribute to the overall reduced health and quality of life of these older individuals (Cupidi et al., 2012; Foley, et al., 2004; D. Lee, 2008; Thompson et al., 2004). Stress associated with sundowning behaviours could, in turn, affect the ability to manage care appropriately, and contribute to symptoms of sleep problems for carers (Gallagher-Thompson, Brooks Iii, Bliwise, Leader, & Yesavage, 1992; Khachiyants, et al., 2011).

McCurry et al. (2007) used Speilman's 3P model to demonstrate that carers are *predisposed* to sleep problems due to demographic and age-related physiological changes to sleep; sleep problems are *precipitated* by changes in routine, being woken by the PWD, and increased daytime napping; then poor sleep hygiene practices and carers' negative reactions or perceptions contribute to *perpetuating* long-term sleep problems, which exist regardless of the sleep and behaviours of the PWD (McCurry, et al., 2007; Spielman, et al., 1987). Increased physical and mental health risks mean that dementia carers are subjected to a "self-perpetuating spiral of sleep and mood disturbances that is difficult to break" (McCurry, et al., 2007, p. 146), which can jeopardise the caregiving role. Indeed, sleep disruptions of both carer and PWD have been identified as one of the reasons that PWD are moved into institutionalised care (Hope, et al., 1998; Pollak & Perlick, 1991; Strain, et al., 2003). This last point is pivotal. If the sleep of PWD *and their carers* can be addressed, this could improve quality of life of the carer and the caregiving relationship, and possibly increase the amount of time that PWD and their carers can continue to live together at home. This is not only an important goal for those affected, but also at a state level where institutionalised care comes at a premium (Alzheimers New Zealand Incorporated, 2012).

It seems that carers' sleep could best be addressed via non-pharmaceutical interventions. The aims of such interventions would be to treat the PWD's sleep problems at a physiological and behavioural level whilst adapting their carers' subjective experience of sleep, and the overall caregiving situation, through sleep hygiene education (McCurry, et al., 2007; Schulz et al., 2002; Van't Leven et al., 2013). Possible interventions are described in Chapter 3.

Much of the previous literature on the prevalence and contributing factors to sleep problems of dementia carers is of a quantitative nature. Considering that much of this research identifies subjective burden and the caregiving experience as key factors associated with sleep, there is comparatively limited qualitative research in this area. One study using focus groups with 15 dementia carers identified three key themes contributing to poorer sleep quality: the fluctuating, unpredictable nature of the PWD's sleep disturbances; carers feeling a need to be hyper-vigilant to their family member's needs throughout the night; and increased worry (e.g., concerning the PWD's symptoms or the future; Simpson & Carter, 2013a). While some carers were aware of good sleep hygiene practice, many appeared to be contributing to their own sleep problems by sacrificing sleep in order to give themselves personal time at night. Furthermore, the caregiver role does not necessarily stop once in bed, making the bedroom environment less conducive to sleep. These findings are similar to that of Arber and Venn (2011) which involved interviews with a broader sample including non-dementia carers (section 1.3.4). It is apparent that as dementia becomes more severe, the prevalence and severity of carers' sleep disturbances also increase (Creese, et al., 2008; McCurry, et al., 2008). In order to understand and treat dementia-related sleep problems, both the PWD and their carers' experience needs to be considered together. This is referred to as a "dyadic approach" (Kotronoulas, et al., 2013; Van't Leven, et al., 2013), and is considered in more detail below (section 2.3.2). Previous research lacks the voice of the care recipient. Through including PWD in qualitative research the complete picture with regards to factors affecting sleep for the carer/PWD pair could be revealed.

2.3 Philosophy of Conducting Research with PWD and their carers

There is a growing body of research concerning sleep and dementia. However, ageing, sleep, dementia, and caregiving are all variable constructs with many personal and psychological factors affecting each individual's experience. When reviewing the literature, it became clear that there were some research gaps regarding the sleep of people affected by dementia living in the community. Firstly, it is considered important to include PWD in research in a more active sense; secondly PWD and carers' experiences should be explored as a dyad rather than independently; and there is a need for more qualitative, person-centred dementia research.

2.3.1 Actively Including People with Dementia in Research

People with dementia deserve the right to be included in research in an active sense, rather than simply being a passive subject (McKeown, Clarke, Ingleton, & Repper, 2010). Much of the early dementia-related research focused largely on the disease rather than the person, and was conducted *about* the PWD rather than *through* them (Downs, 1997). The omission of the PWD's voice in research is an important point. Although they are affected cognitively and psychosocially, the exclusion of PWD from research is not considered ethical. The use of proxy reports alone to understand the sleep experience of PWD is unreliable, unrepresentative, and further marginalises an already neglected group (Booth & Booth, 1996; Clare, 2004; Innes, 2009; S. Smith et al., 2005).

"Society tends to focus on the dementing person's functional disabilities rather than his or her continuing strengths. This tends to alienate the dementing person from normal communication" (Garratt & Hamilton-Smith, 1995, p. 7). This stigma is fuelled by the media as well as the way dementia is understood socially (Kirkman, 2006; Sweeting & Gilhooly, 1997). A common misconception has been that PWD cannot contribute to research in the same way that people without dementia can, because of the unreliability of accounts from someone who forgets (Innes, 2009). Cognitive impairment has been deemed a barrier to collecting reliable data, as well as a barrier to collecting informed consent (see section 3.4). However, this barrier needs to be resolved rather than avoided, because the subjective views of PWD offer

rich insight into the personal experience and impact of living with the disease, which in turn can help inform policy and practice (Clare, 2004; Innes, 2009; Wilkinson, 2002).

Another common misconception has been that there is a complete loss of self with dementia. This is now being challenged (Kitwood, 1997; Kitwood & Bredin, 1992; Wilkinson, 2002). "People with dementia are presented as people with unique biographies, personalities and life circumstances, all of which interact with the neurological impairment" (Downs, 1997, p. 598). With this in mind, researchers are being required to adapt the way they think about and conduct dementia-related research. They are challenged to elicit and respect the views of PWD regarding their experience of their illness, rather than treating them as objects to help understand the trajectory and treatment of dementia (Cotrell & Schulz, 1993; Downs, 1997). After all, they are an expert on experiencing dementia.

Policy and guidelines are being developed to support this movement into person-centred dementia research, and PWD are slowly becoming more involved as participants, as well as key informants (McKeown, et al., 2010; Telford, Beverley, Cooper, & Boote, 2002). A major challenge for conducting dementia-related research is judging degrees of competence to understand, give consent, and take part. People with dementia have been misperceived as completely incompetent and unable to understand situations. Consequently their individual rights are at risk of being compromised. However competence should not be generalised (T. Moore & Hollett, 2003). Everyone has the right to be treated fairly, as well as a right to be heard, and a right to their privacy, regardless of their ability to exercise or appreciate their rights. On this basis, having reduced capacity to understand should not be a suitable justification to exclude people from taking part in research. In fact, doing so is likely denying the individual's right to be involved with an issue that is of fundamental importance to them. Assumptions that PWD have little to offer or are unable take part in research are slowly being challenged (Sherratt, Soteriou, & Simon, 2007; Swain, Heyman, & Gillman, 1998). The focus now is more on the practical issues of *how* to include PWD in the research projects whilst protecting them from undue risks and exploitation (National Ethics Advisory Committee, 2012a; Wilkinson, 2002). Rather than avoiding person-centred dementia research, a carefully designed protocol is required, taking into account an assessment of competence and capacity to consent, as well as judging assent and dissent throughout the research. This is of particular importance for research

that has increased risks, or takes place over a series of time points, such as trialling a medication or therapy (see section 3.4).

2.3.2 Research with PWD/Carer Dyads

Some of the risks associated with including PWD in research, as well as concerns about reliability, can be addressed by actively including both the PWD and their carer in research. This is of particular interest concerning the collection of information about sleep and measuring efficacy of treatments. Due to the loss of consciousness during sleep, many of us are unaware of sleep disorders that do not cause a full awakening, and those who live with us can often provide a different account. Kotronoulas et al. (2013) highlight the importance of considering the sleep of both the care-recipient and the carer at the same time, in order to gather a full understanding of the intrinsic and extrinsic nature of sleep disruptions of these individuals. In this meta-analysis of dyadic studies, the authors found that there is a large amount of variability between individual dyads as well as studies. Key themes concerning dyadic sleeping were highlighted. These included carers changing their sleep timing in order to maintain bedtime rituals and sleep timing of their partner, and their sleep disturbances and timings at night being interrelated. Daytime sleep duration or timing appears less likely to be coupled. Care recipients typically spend more time inactive or napping in the daytime than carers, despite carers' increased reports of sleepiness. Finally, situations in which both the carer as well as care recipient's sleep is disturbed are considered to make the caregiving situation as a whole more difficult:

> A dyad's sleep is a dynamic field of interference of several compounding and interacting variables, which not only affect sleep but are also affected by sleep so that an infinite loop of chronic sleep loss and dysfunction can be established as the dyad moves in time and across health transitions (Kotronoulas, et al., 2013, p. 590).

There is a need to consider both viewpoints and the sociological situation when attempting to understand the types of sleep problems PWD and/or their carers' experience, as well as when measuring for change when trialling treatments or interventions (Van't Leven, et al., 2013; S. Williams, 2005).

Much of the research concerning sleep and dementia, and dyadic sleeping within the informal care situation, has used quantitative methods. Some have incorporated case-studies in order to provide a more comprehensive story of the dyad (e.g., Higgins, Hornick, & Figueiro, 2010; McCurry, Logsdon, Vitiello, & Teri, 2004), however the personal contributions from the PWD and their qualitative experience are still not represented. While many PWD are likely to be competent in completing a questionnaire or survey, a qualitative methodology is considered a more person-centred approach which may feel less clinical to participants, reducing the risk of PWD being uncomfortable with the research-situation (Bamford & Bruce, 2002; Wilkinson, 2002).

2.3.3 A Qualitative, Person-Centred Approach.

Few qualitative studies are available concerning the personal sleep experiences of PWD/carer dyads. Previous qualitative research involving informal carers helps explain some of the variability in their reports of sleep disturbances regarding themselves and the person in their care (Arber & Venn, 2011; Simpson & Carter, 2013a). Studies which have also solicited the viewpoints of PWD have taken place in institutionalised environments (Ellmers, et al., 2013; Kerr, Wilkinson, & Cunningham, 2008; W. Martin & Bartlett, 2007). For example, W.Martin and Bartlett (2007) used interviews to gain insight into PWD's perceptions about their sleep and its relationship to healthcare, vulnerability and memory. These studies highlight the importance of also gaining the PWD's personal insights with regard to their social experience of sleep, their perceptions of their care situation, and their sleeping environment. Gathering rich qualitative sleep data from PWD is not only possible but recommended, in order to understand the full picture with regards to factors affecting sleep.

Qualitative data about the sleep of PWD living in the community appears to be absent or limited at best, particularly from the PWD's or dyad's perspective. Drawing on my experience as a care assistant as well as this literature review it was clear that I needed to maintain a person-centred philosophy and dyadic approach to ethically and reliably gain the views of PWD concerning their sleep. It is imperative to honour the individual's subjective experience in order to understand how important they consider sleep and sleep disturbances to be to them. This includes how they feel their sleep impacts their symptoms of dementia, their waking life, and relationship with their carer, as well as how they feel they could manage

or treat their sleep disturbances. With this understanding I intend to undertake my research involving PWD with integrity, honouring the voices of PWD.

2.4 Rationale for Study 3

The main aim of this thesis was to trial non-pharmaceutical interventions to improve the sleep of community-dwelling dyads (Study 4). However, at the start of this work, there was no available data on the sleep of New Zealanders with dementia. To inform the design of Study 4, it was considered necessary:

- to conduct a qualitative study in order to address the question: "How do PWD and their carers personally experience sleep problems?" to increase our understanding of the impact that sleep problems have on their lives;
- to consult with PWD and carers in the region concerning the proposed topic, methodology and interventions; and
- to conduct a smaller, lower risk study to initially engage with the dementia community, specifically Alzheimers Wellington.

Alzheimers Wellington is a local dementia organisation offering a support and advocacy service for PWD and their carers across the greater Wellington region, and was a key location for contacting and recruiting PWD living locally. This initial low-risk study enabled me to build a relationship with the staff of Alzheimers Wellington and build their confidence in both the project and me personally, before embarking on the larger intervention study.

Study 3 used a focus group methodology, with PWD and carers sharing how they perceived their own sleep and that of their partner, what methods they already had in place for managing their sleep, as well as offering feedback on the suggested interventions for Study 4. The findings of Study 3 offer some of the first qualitative data on the sleep experience of PWD and are presented in section 2.5 as a paper.

(Gibson, R. H., Gander, P. H., & Jones, L. M.). Appendix 2 contains materials related to ethical approval, participants' information sheets, consent forms, the focus group schedule, as well a pamphlet disseminated to participants and community.

2.5 Study 3: Understanding the Sleep Problems of People with Dementia and their Family Caregivers

2.5.1 Abstract

Sleep disturbances are common with dementia and can adversely affect waking function. However, the perspectives of people with dementia and their family caregivers concerning their sleep are under-researched. We conducted three focus groups with 12 community-dwelling pairs (a person with dementia and their family caregiver). Discussions addressed sleep disturbances, coping strategies, and beliefs and attitudes surrounding sleep. Thematic analysis indicated that dementia-related sleep disturbances were common, including confused awakenings and dementia-related behaviours at night, changes to sleep timing, and nightmares. Common issues for caregivers included being woken at night, having problems getting back to sleep, trips to the bathroom, and daytime sleepiness. Participants often normalised their sleeping problems and had developed a number of coping strategies. These findings highlight the impact that sleep disturbances can have on people living with dementia. Their experiences and beliefs need to be considered for developing effective interventions to improve sleep, waking function, and wellbeing.

2.5.2 Introduction

Sleep disturbances are among the most challenging behavioural problems associated with dementia and have been identified as a primary reason for moving people with dementia (PWD) into institutionalised care (Hope, et al., 1998; Pollak & Perlick, 1991). Progressive dementias (e.g., Alzheimer's disease) have effects on sleep that can be distinguished from normal aging, particularly fragmentation of the sleep/wake cycle and disruption to the circadian regulation of sleep (Song, et al., 2010; Vitiello & Borson, 2001). These changes have been related to deterioration of the brain structures and neurotransmitters involved with sleep, as well as the psycho-social and behavioural changes seen with

dementia (Klaffke & Staedt, 2006; Vitiello & Borson, 2001; Wu & Swaab, 2007). Disordered sleep and irregular sleep timing are associated with poorer cognitive performance (Moe, et al., 1995; Naismith et al., 2010), poor physical and mental health (Foley, et al., 1995), and mortality (Gallicchio & Kalesan, 2009; Gehrman et al., 2004) among older people with and without cognitive impairment. Better management of dementia-related sleep problems could thus lead to improvements in waking symptoms and wellbeing.

Past research addressing sleep and dementia has mostly used proxy questionnaires or reports from professional staff or family caregivers (e.g., B. Carpenter, et al., 1996; McCurry, et al., 1999). One previous study included interviews with PWD in care facilities and highlighted the importance of gaining personal insights with regard to the experience of sleep and its relationship to healthcare, vulnerability and memory (W. Martin & Bartlett, 2007). In practice, the personal insights of PWD have rarely been collected, giving a limited perspective of the disease experience (Cotrell & Schulz, 1993). Additional qualitative research is needed to provide insight into how PWD and their family caregivers living together at home recognise, experience and cope with dementia-related sleep problems.

Sleep disturbances are also common among family caregivers of PWD. The types of sleep disturbance are varied and typically involve complex interactions between the effects of having a disrupted sleep schedule due to dementia-related sleep problems, caregiver burden, and the co-morbidities of the caregiver (McCurry, et al., 2007). Situations in which both PWD and caregivers are sleeping poorly tend to be those in which the overall care giving situation is more difficult (Chenoweth & Spencer, 1986; McCurry, et al., 2007). Therefore it is important to not only investigate the sleep experience of those with dementia, but also that of the family caregivers. In the present study we aimed to:

a) increase our understanding of the types of sleep problems being experienced by community-dwelling pairs consisting of a person with dementia (≥65 years of age) and their family caregiver;

b) gain insight into how older PWD and their family caregivers generally view their own sleep and that of their partner;

c) understand what strategies older PWD and their family caregivers use to manage their sleep.

2.5.3 Design and Methods

Participants.

Twelve pairs were recruited via Alzheimers Wellington, a local dementia organization who offer a support and advocacy service for people in the Wellington Region of New Zealand. Convenience sampling was employed to recruit participants who would be willing to openly discuss their sleeping experience. Inclusion criteria were living together as a PWD/family caregiver pair, and the person with dementia being ≥ 65 years old and having disrupted sleep. Diagnoses of dementia and sleep problems were based on participants' report of a medical diagnosis, membership of Alzheimers Wellington, and experiencing sleep disturbances.

Based on the homogeneity of the groups with regards to age, having a dementia diagnosis, and sleep disturbances, this sample was considered sufficient to reach theoretical saturation (Krueger, 1994). The type and stage of dementia was not recorded. However as participants with dementia were invited to be actively involved in the focus group discussions, they or their family member had deemed them cognitively able to participate. Therefore the PWD were also considered to have the capacity to consent for themselves, which they all did by signing a consent form prior to the focus group. Given that the PWD were taking part and consenting alongside their family caregiver to participate in a non-invasive protocol, the project was considered as a low risk observational study by the Health and Disabilities Ethics Committee (National Ethics Advisory Committee, 2012b, New Zealand Central Regional Ethics Committee, number CEN/10/EXP/33). Therefore no further assessment of mental capacity was undertaken.

Eight of the PWD were male, four were female. Caregivers were family members, 11 of whom were spouses (≥65 years old) and one was a daughter (< 65 years old). Three of the caregivers were male and nine were female. No other demographic information was collected.

Materials.

Prior to each focus group, participants were sent a brief focus group schedule. This approach was requested by Alzheimers Wellington to ensure that participants had time to discuss what was involved and were fully informed prior to giving written consent. This is considered particularly important when recruiting participants with dementia (National Ethics Advisory Committee, 2012b; Qureshi & Johri, 2008). Having the schedule prior to the focus group also gave the opportunity for participants to consider the topics they would be discussing and to bring written notes to the focus group meeting if they wished.

Discussions were semi-structured in order for groups to be as comparable as possible. Questions were designed to reveal the practices and beliefs of PWD and their family caregivers regarding sleep, and moved from general to more specific topics:

a) sleep of the PWD (getting to sleep, sleeping in the day, and sleep disruptions);

b) sleep of the family caregiver (getting to sleep, sleeping in the day, and sleep disruptions);

c) strategies for managing sleep (what they are doing already, what they might find useful).

The feasibility of using scheduled light exposure and physical activity as an intervention to help improve sleep of the PWD was also discussed. This was to help inform the design of a subsequent pilot study and is not presented here.

Procedure.

A letter was distributed to members of Alzheimers Wellington inviting them to take part. Those who were interested contacted the researcher via a free phone number to discuss the study in more detail. Potential participants were then posted a full information pack. This included a consent form to be signed by both the PWD and their family caregiver.

Participants took part in one of three focus groups in a community space used by Alzheimers Wellington during November 2010. Groups included between three and five pairs and lasted 60-90 minutes. A focus group methodology was chosen in order gain PWD's insights in a supportive and

empowering environment with less pressure to participate and more cues for memories (Bamford & Bruce, 2002).

Focus groups were facilitated by two members of the research team and a community worker from Alzheimers Wellington who offered familiar support (as recommended by Krueger, 1994). One facilitator led the focus group, with the second person actively listening, seeking clarification and making notes (as recommended by D. Morgan, 1997). A brief introduction was given at the beginning of each focus group to reaffirm the reason for being there and what would be discussed. This gave participants the opportunity to ask questions prior to the audio recorder being switched on. Participants were reminded that they were free to decline to answer particular questions, to leave the group at any time, or to withdraw their consent at any time. The main topics for discussion, the timeframe, and the participants' rights were written on a whiteboard and were also given as a handout for those who had not brought the schedule with them. Those who brought notes with them used these as a prompt during the discussions.

All participants were encouraged to contribute and to answer each question, and groups were conducted in a sensitive and supportive manner to allow participants time to voice their concerns while also monitoring for degrees of competency and signs of distress. This structure was deemed necessary to allow the PWD, who may have compromised comprehension and verbal abilities, the opportunity to contribute to the conversation (Bartlett & Martin, 2002).The focus groups were audio-recorded with the permission of the participants, and transcribed verbatim.

Data Analysis.

The data collected represented the experience, meanings, and reality of the participants. A thematic analysis of the transcripts was conducted, which was data-driven allowing themes to emerge from the transcripts (Braun & Clarke, 2006). Two of the authors read, re-read, and coded the transcripts independently with reference to the audio recordings and researcher notes as needed. Basic Themes were identified, and illustrative quotes noted. These were then compared and reviewed to identify broader patterns and then rearranged into Organizing Themes (which were further discussed, compared and revised where necessary). Finally all themes were categorized into even broader Global Themes, together forming a thematic network (Attride-Stirling, 2001).

Global and Organizing Themes concerning the sleep of the PWD and of the caregiver were grouped and quantified by whether they were identified by the individual or by their partner. Thematic networks concerning *strategies for managing sleep* and *beliefs and attitudes towards sleep* are reported purely qualitatively because comments on these topics were not directly solicited during the discussions.

2.5.4 Findings

Sleep of participants with dementia

Frequency of Global and Organizing Themes concerning the sleep of the PWD are summarised in *Figure 2.6* and presented below with illustrative quotes from both caregivers (C 1-12) and PWD (P 1-12). The PWD appeared to have less insight into the confused awakenings and dementia-related behaviours (DRB's). Some caregivers found this made coping at night more difficult: "She doesn't realise what I pick up at night. She has beautiful sleeps she says, which no doubt she does, but she doesn't realise during the time she is up and down" (C4). The PWD who were more aware of their night time behaviours excused them in relation to past experiences or routines.

Nightmares were an issue raised by five of the PWD. Some PWD recalled their nightmares in great detail, noting that the content was often vivid and recurring. Furthermore, morning dream residue (e.g., dream imagery or emotions remaining after waking) would contribute to poor daytime mood, sleepiness, or confused behaviours: "It stays with you for quite a bit during the day, doesn't it? (C3) "Yes" (P3), "until you find something else to replace it" (C3). For others, the content of the nightmare was not remembered but the emotional distress remained: "I can't remember the nightmares but I know I am absolutely terrified when he wakes me up" (P1). Some caregivers noted that nightmares were often accompanied by shouting or sudden disruptive movements.

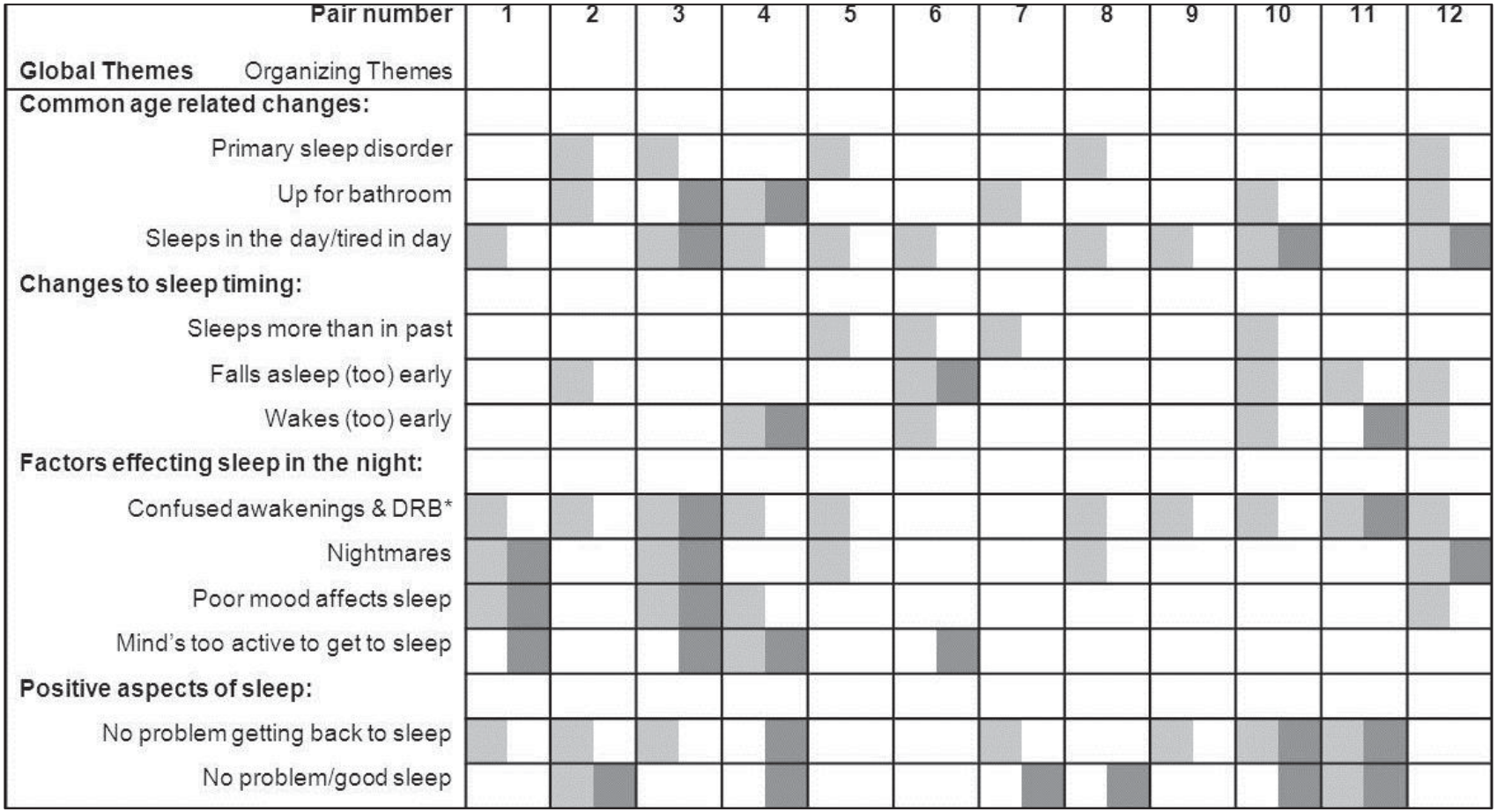


Figure 2.6. Global and organising themes concerning the sleep of those with dementia.

Split by caregiver (light grey) and PWD (dark grey) comments.

Poor mood, for instance feelings of depression or anxiety, were reported to contribute to problems getting to sleep raised by two PWD. In other instances, caregivers noted that paranoia or fear were related to agitated behaviours during the night and made getting up in the morning more challenging: "(P8) can wake up and he is suspicious at the world and he just doesn't want to get out of bed" (C8). Poor mood or lacking enthusiasm were raised as factors contributing to daytime sleepiness as well as an explanation for variable sleep: "We used to get up early but he's shown no interest in doing anything" (C12). A third of the PWD reported that their mind was too active: "If I could switch my brain off and stop thinking I would be able to go to sleep" (P1). Some explained that they were worrying about their prognosis, family, or finances.

Positive aspects of sleep.

Two-thirds of the PWD were reported to have no problem getting back to sleep after a disturbance. Participants related this to the automatic nature of the night time behaviours: "I have to get up and look for the doors and make sure that everything is. . . I suppose I am walking in my sleep and go back and I sleep" (P3). This was viewed with relief by some caregivers: "But once he had re-hidden everything he goes back to bed, and then he does go back to sleep" (C2).

Comments such as "I don't really have any problems sleeping" or "my sleep is good" were made by half of the PWD. Such comments were typically raised during the initial stages of discussion. Furthermore, many would disagree with the claims of their caregiver with regards to their sleep disruptions, arguing that they slept well.

Sleep of caregivers

Frequency of Global and Organizing Themes concerning the sleep of the family caregivers are summarised in *Figure 2.7* and presented bellow with illustrative quotes.

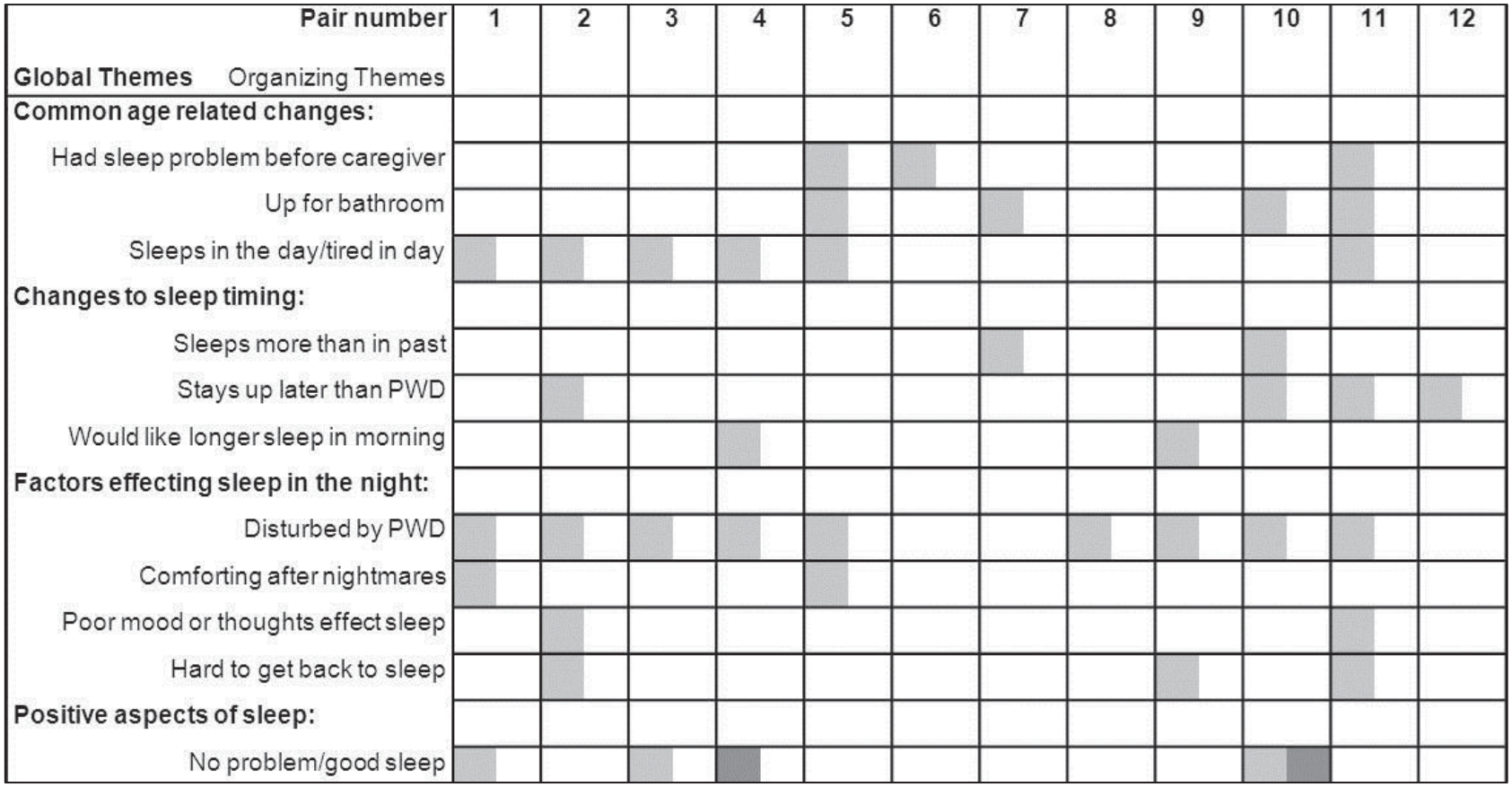


Figure 2.7. Global and organising themes concerning the carers.

Split by caregiver (light grey) and PWD (dark grey) comments, * DRBs (dementia-related behaviours).

Common age-related changes to sleep.

Sleep problems considered unrelated to (or beginning prior to) the care giving role included symptoms of insomnia, restlessness or daytime sleepiness. Night time trips to the bathroom were also common, however some were getting up simply because their partner was and thought they might as well. One caregiver noted that this pattern meant that she was "getting into a habit which I wouldn't have done" (C10).

Daytime sleepiness was welcomed with a regular afternoon "feet up" (C3) by some caregivers, however the opportunity to nap was not taken by all due to increased responsibilities making napping more difficult: "Just tired really yes, just tired all day. But you still have to do things because it is your responsibility really, you are the caregiver. I find sometimes it is a bit hard" (C9).

Changes to sleep timing.

Two-thirds of caregivers reported that they were often up later than their partner, who went to bed too early for them. Some caregivers used their additional evening time to relax or get chores done. However opportunities to sleep longer in the morning were reduced because they needed to provide morning care, suggesting that caregivers total sleep time was probably reduced.

Factors affecting sleep in the night.

Most caregivers had their sleep disrupted by the confused awakenings and DRBs of their partner. Instances included being physically disturbed: "He has been stretching out and kicking me and knocking me around, was terrible. He is terribly apologetic of course!" (C5), or providing comfort or support:

> When she is having had a nightmare I am usually woken up. I can hear screaming, you know. So I wake up and I will hold her for a while and then she goes off to sleep. But sometimes it happens three or four times a night (C1).

After such instances, some caregivers found it hard to get back to sleep: "I find that sometimes (P9) takes so long [with the light switch] that I get quite churned up and that its extremely hard to go back to sleep" (C9); "Yeah, it takes me a while. He always keeps putting his hand out to make sure I'm there" (C12).

Poor mood including worry and depression was raised by the caregivers, and in two instances this affected their sleep. However others noted that they felt too physically and/or mentally exhausted by bed time to lay awake.

Positive aspects of sleep.

Three caregivers noted that their sleep was not problematic or actually quite good. The only comment PWD had concerning their partner's sleep was that they had no problem with sleep, for example: "She shouldn't have any problems, no problems at all. She sleeps more than I do!" (P4).

Strategies to improve sleep

General discussions as to how the participants were managing their sleep lead to three Global Themes: a) strategies related to the sleeping environment; b) safety issues surrounding sleep and night time awakenings; and c) techniques to relax at night and stay awake in the day. These themes and their subthemes are presented below.

Strategies related to the sleep environment.

Many of the pairs were sleeping in separate beds or bedrooms. For some, this was to reduce the amount that they disturbed one another. For others this was related to safety and anxiety concerning caregivers being "knocked around" (C5). The amount of light in the bedroom was also discussed in all of the groups, with some PWD requiring the bedroom to be dark to help them sleep (using thick curtains and so forth), whereas others needed a little light to reduce fears associated with darkness as well as for safety (see below). Noise was also discussed, with some pairs enjoying the lulling effects of having a television, radio or music on in the bedroom to help them sleep.

Safety at night.

The risk of falls (typically concerning the PWD) was mentioned across all groups, and was mostly related to night time trips to the bathroom, but also to anxiety related to past experiences with falling. Strategies being used to reduce this risk included lighting in corridors, stairways and bathrooms; having a bedside torch; and the use of a bed pan. Other strategies to increase safety at night included a bell for the

PWD to alert their partner if they needed assistance, keeping doors and windows locked at night, and caregivers reading and increasing their knowledge about dementia-related support strategies.

Strategies to help get to sleep at night.

Having a routine during the day and around bed time was acknowledged by many pairs as important to help get to sleep. Specific routine activities included having regular meal and bed times, enforcing a reasonable or practical bed time, and taking planned naps:

> We try and keep it routine at night, with a similar timing and a hot shower, and I leave the light on the bathroom so if he gets up in the night he can see the way and see the way back to bed. He has got a bell so then he can ring me if he needs to in the middle of the night, and hopefully the cat obliges and goes to sleep. Those are things that we have tried to set in place that will enable him to get to sleep at least, regardless of what happens later on in the night (C3).

However it seems that implementing a routine is not always easy. Some PWD commented that they were not aware of their routines, and caregivers noted that DRB's can often override any planned schedule:

> He'll be tired and from half past seven he wants to go to bed and will not go to bed unless I go, and I am not going to bed at half past seven because I don't want to be awake from half past seven to god only knows when. Because I was probably an eleven or twelve o'clock at night person and so was he, so I say nine thirty, now we have got it down to nine then we go to bed and he quietens down (C2).

For the PWD, techniques to get to sleep included not consuming caffeine or alcohol too late in the day, calming teas before bed, a warm shower before bed, winding down in the evening with television or a film, having a pet on the bed for comfort, and using relaxation or hypnotic techniques to reduce thoughts at night: "So you can shut the brain off it goes 'boom boom', and I forget how do it, but inside I can do it still" (P3). Sedating medications were only used by a few pairs, with some actively avoiding such medications.

Some caregivers purposefully stayed up later than their partner to relax, whereas others found that they had to go to bed at the same time as their partner to comfort them. When caregivers had difficulty getting back to sleep, their techniques included getting up and making a hot drink, doing chores, writing down thoughts, or simply staying calmly in bed - waiting for sleep.

Many pairs raised the importance of keeping physically, socially and mentally active during the day to make themselves "good and tired" (C7) before bed. Alternatively, some were actively reading or doing puzzles to keep the mind active and prevent falling asleep during the day. Others took a routine daytime nap to reduce the risk of unintentional dozing later in the day, which they believed may subsequently contribute to some sleep problems at night.

Beliefs and attitudes towards sleep

Discussions revealed several Global Themes concerning participants beliefs and attitudes surrounding sleep, these were: a) the importance of sleep, b) the normalisation of sleep problems, c) feelings of guilt, d) belief of sleeping well, and e) hopeful and positive attitudes. These are presented in more detail below.

The importance of sleep.

Most of the participants (especially caregivers) acknowledged the importance of sleep and were aware of the implications of sleep deprivation on their daytime functioning: "I find if I have a decent night's sleep I have got more energy, more patience, I can cope with (P3) having a bad day much better" (C3). Sleep was therefore viewed with respect, with many referring to past experiences which caused sleep deprivation (e.g., as new parents).

The normalisation of sleep problems.

Comments such as "Isn't the theory that you need less sleep when you're older?" (P10), revealed a common belief that changes to sleep and sleeping problems were a normal part of aging. The attitude related to this belief was that little could be done to help and that they would have to "just live with it, [because] you can't really change it" (C8). Others also implied that they expected their sleep to

deteriorate: "I will probably get to that point, will I?" (P4). A common belief of caregivers was that their partner's daytime sleepiness was probably related to boredom or physical or mental limitations.

Feelings of guilt.

Some caregivers commented on their feelings of guilt associated with enforcing routines or sleeping practices with their partner. These feelings were partly related to the knowledge that their partner might not understand or remember why the changes had been made, or feelings of selfishness with regards to getting a good night's sleep at the expense of their partner's comfort.

Belief of sleeping well.

Beliefs and attitudes expressed by the PWD typically surrounded the belief that they had no problem with sleep or that their sleep was good. Through further discussion, this belief was translated by some caregivers as more of a lack of awareness to what was happening in the night or not remembering that they had slept during the day.

Hopeful and positive attitudes.

An overarching attitude across the groups was one of coping and hope for continuing to manage in the future: "I hope I manage to stay like this for a long time" (C4), "We are getting there slowly, these things can't be changed immediately or nothing" (C7). There were also frequent references to having a good sense of humour and the belief that living at home together was important: "Yes, we work pretty well together as a team, we always have done" (C7).

2.5.5 Discussion

This study used focus groups to generate themes concerning sleep disturbances experienced by PWD and their family caregivers. Understanding the type of sleep problems being experienced, as well as how they are viewed and being coped with, is a unique and essential step towards developing interventions to help improve dementia-related sleep problems.

All but one of the participants in the focus groups were aged 65 or over, and so some age-related sleep problems and daytime sleepiness were to be expected (Foley, et al., 1995). The PWD had

exacerbated age-related sleep problems compared to the caregivers. This has been reported in past studies and related to neurophysiological changes affecting sleep architecture, as well as degeneration of particular neural pathways associated with controlling sleep regulation, respiration, and movement during sleep (Bliwise, 1993; Vitiello & Borson, 2001).

Daytime sleepiness and variable sleep timing are commonly reported dementia-related symptoms (Bliwise, 1993). Participants related these symptoms to boredom or having less awareness of time of day. Past studies explain these symptoms by dementia-related degeneration of activity within the circadian body clock and the sleep and arousal systems (Volicer, et al., 2001; Wu & Swaab, 2007). Changes to sleep timing can have a significant impact on daytime functioning, be it psychosocial or performance-related (Kuhn, Edelman, & Fulton, 2005; T. M. Monk, 2010). For PWD and their caregivers, compromised physical and social activities might further contribute to weakened sleep regulation (McCurry, et al., 2007).

For both PWD and caregivers, the greatest disruption to sleep at night was related to confused awakenings with DRBs. The restlessness, disorientation, and automatic nature of the behaviours described are typical symptoms of 'sundowning', with symptoms increasing from around dusk and continuing through the night (Vitiello & Borson, 2001). Sundowning has been related to the effects of sleep deprivation as well as neurophysiological signs of sleep during states of wakefulness (Klaffke & Staedt, 2006; Volicer, et al., 2001), thereby explaining the performance of the behaviours seemingly half asleep or 'automatically'. This, as well as the typical cognitive impairment caused by dementia, is also likely to explain the reduced memory of such behaviours (Malamut & Ryan, 2008). Some of these behaviours could also be explained by parasomnias un associated with dementia (e.g., walking, talking, or eating during sleep, Mahowald & Schenck, 2000). However objective sleep monitoring would be required to differentiate between these behaviours.

Previous research shows an age-related reduction in recall for dreams and nightmares (Funkhouser, Hirsbrunner, Cornu, & Bahro, 1999). However in the present study, an exacerbation of nightmares for PWD was reported. This might be explained by dementia-related degeneration of the limbic system causing increased negative or repetitive dreams. Alternatively, damage to the frontal lobes

might prevent dream content from being remembered, but leave the feelings of fear described by some participants (Domhoff, 2001). Degeneration of the networks linking these key brain areas is also likely to contribute to the dream residue which some of the PWD experienced (Solms, 1997). The cholinergic system is considered responsible for controlling levels of consciousness and REM sleep. Changes in this system associated with dementia may increase the likelihood of dream-like hallucinations during wakefulness (E. Perry, Walker, Grace, & Perry, 1999). Increased repetitive dreams may also be explained by the repetitive nature of many waking symptoms of dementia continuing on into sleep (Altshuler, Barad, & Golfarb, 1963). In addition, commonly used medications such as Donepezil stimulate the cholinergic nervous system, increasing the risk of disturbed dreams (Kitabayashi, et al., 2006).

Poor mood has been highlighted as an issue for both PWD and caregivers (Crespo, López, & Zarit, 2005; Korczyn & Halperin, 2009). An increased risk of anxiety has been related to the diagnosis of dementia, changes within relationships to others, and a loss of skills (Qazi, Spector, & Orrell, 2010). Excessive thoughts at night and poor mood have also been identified as contributing to symptoms of insomnia (Foley, et al., 1995). In the present study, poor mood and uncontrollable thoughts, as well as nightmares appeared to contribute to an 'internal struggle' against sleep which was identified as the main sleep problem by some PWD. Whereas, caregivers were more likely to comment on their partner's behavioural sleep problems.

Many strategies were being used by the participants to help with sleep at night and also to reduce the risk of falls and improve safety in general. This was not surprising as these topics have been identified as key for healthy aging, a consequence of disturbed sleep, as well as factors contributing to moving family members into long term care (Rowe & Fehrenbach, 2004; Stone, et al., 2008). Environmental and relaxation techniques were more varied and personal in nature, however many participants were attempting the type of techniques found in standard recommendations for improving sleep (e.g., National Sleep Foundation, 2011) and used in institutionalised settings (Day, Carreon, & Stump, 2000).

There was an overarching belief that sleep changes were a normal part of aging and/or dementia. Many PWD were unaware of their sleep problems, commenting that their sleep was in fact good. This might be because of participants giving idealised accounts (Bamford & Bruce, 2002). However healthy

adults are often unaware or deny their own sleep problems making it more difficult to recognise and diagnose disordered sleep (Dagan, 2007). Furthermore denial, normalisation, or lack of insight are all common reactions among PWD concerning their symptoms, making the disease particularly difficult to manage (Feher, Mahurin, Inbody, Crook, & Pirozzolo, 1991). The PWD had much greater insight into their own sleep than that of their caregiver, with only two PWD having any comment on their partner's sleep.

This study provides some information into the commonality of dementia-related sleep problems and also illustrates the degrees of consensus between family members, and personal insight of particular sleep disturbances. The normalisation or lack of insight of age and dementia-related sleep problems is likely to have implications for recruiting "problem sleepers" into research studies. The use of objective sleep measures, as well as subjective and proxy reports, is particularly important for future studies concerning the sleep of PWD, because of the tendency of PWD to have a distorted view of their sleep as well as impaired memory (Feher, et al., 1991; Vitiello & Borson, 2001).

Using focus groups to understand the experiences of PWD raises some challenges because of the nature of dementia (Lindstorm et al., 2006). It is likely that some PWD might have found it difficult to follow the train of conversation or struggled to hold onto their thoughts until their turn in the discussion (however many appeared to manage). Impaired language and memory for words may have meant that one-on-one interviews might have been easier for some. To help counter these challenges, a brief focus group schedule was sent to participants to facilitate prior discussion and enable the PWD to make notes of things they might like to say. This strategy was successful in not only providing participants the opportunity to bring their own prompts for discussion, but also having a reminder of the schedule helped keep the discussions focused, as well as supplying all the information necessary for participants to make an informed decision prior to consenting to take part. Encouraging participants who hadn't already offered any comments prior to moving on to the next topic, was a useful strategy that gave them permission to speak if they had been waiting or had missed the conversational cues.

The discussions of the PWD often went off topic, with common tangents including the process of being diagnosed with dementia, memory problems, and television programs. Caregivers were helpful in

bringing these diversions back to the topic. In our experience, it was important to provide enough time to allow PWD to express themselves, but also necessary to be able to refocus the conversation when it became repetitive or off topic, without upsetting the participant or the flow of the group (having the schedule available as handouts and on the whiteboard helped counter this challenge). Some participants also told stories of past sleeping problems. In some cases, such stories served an important role in identifying the background and personality of the participant and in providing some explanation for current experiences (such as excusing seemingly odd behaviours in the night). For focus group moderators, it is important to try and understand the meaning behind such stories rather than immediately treating them as tangents (Bamford & Bruce, 2002).

Future studies might consider recruiting a larger and more diverse group reflecting the range of factors that have been identified to play a role in sleep disturbances (Gander, et al., 2005a; Mihaere, et al., 2009; Paine, et al., 2005; Tractenberg, Singer, & Kaye, 2006) and dementia-related care (Connell & Gibson, 1997; Stephens, Franks, & Atienza, 1997; Vetter et al., 1999).

This study adds to our understanding of the types of sleep problems being experienced by PWD and their caregivers living in the community, and how they view and cope with these. The next step is to attempt to improve sleep. Previous studies have shown that non-pharmaceutical interventions (such as timed light exposure and exercise therapy) used in institutionalised settings can have positive effects on the sleep of PWD (Ancoli-Israel, Martin, et al., 2003; Brown et al., 2013; J. L. Martin, Marler, Harker, Josephson, & Alessi, 2007). Attempts to use such interventions in the community have had mixed results (McCurry et al., 2011; Most, Scheltens, & Van Someren, 2010). Mood, daytime functioning, and quality of life are often affected by sleep disruption (Foley, et al., 1995; Naismith, et al., 2010). With dementia, such factors typically deteriorate at around the same rate as sleep (Moe, et al., 1995). Therefore effective sleep interventions might also improve the waking symptoms associated with dementia, as well as the sleep and coping of family caregivers.

Conclusion.

Sleep disturbances are a key issue for people living with dementia and for those providing care. Disturbances vary from exacerbated age-related sleep problems to irregular sleep timing and disruptive

behaviours during the night. Sleep problems of PWD originate from a variety of factors, including neurophysiological changes related to aging and/or dementia, behavioural influences of activities surrounding sleep, as well as beliefs and attitudes towards sleep. The sleep of family caregivers is also at considerable risk due to having to provide physical and mental support throughout the 24-hour day. Interventions are needed to improve the sleep of community-dwelling PWD that might in turn improve the sleep of their caregivers and have positive effects on the daytime functioning and quality of life of this older population.

Acknowledgements.

We would like to thank all the people who gave their time to take part in this research, and the community workers of Alzheimers Wellington for supporting and making this research possible. This work was supported by the Massey University Research Fund (grant number 09/0114); and the Maurice and Phyllis Paykel Trust (project grant 2010).

2.6 Personal Reflection

Conducting the focus groups for Study 3 was a fascinating experience. I was delighted with the interest and response to the proposal from the staff and members of Alzheimers Wellington. Some of the key points I took from conducting and analysing these groups were how diverse the reports of sleep problems were, the differences between reports from the PWD and their carer, and the lack of awareness that some of the PWD had concerning routines and carers' motives or own sleep. It was of interest that many avoided using medications for sleep. Although many had some strategies in place, it was clear that sleep needed to be more formally addressed in order to try and relieve some of the symptoms and behaviours of dementia as well as carer stress.

3 TREATING SLEEP DISTURBANCES OF OLDER PEOPLE AND PEOPLE WITH DEMENTIA

The literature reviewed in Chapter 2 and results of Study 3 clearly indicate that disturbed sleep is an important feature of dementia. When sleep of both a PWD and their family carer is disrupted, institutionalisation is considered more likely (Hope, et al., 1998; Pollak & Perlick, 1991; Strain, et al., 2003). There is increased drive to enable PWD to remain in their homes for as long as possible. This is related to the personal wishes of the individual and family regarding maintaining independence and quality of life, as well as reducing the economic impact of dementia-related care on society (Alzheimers New Zealand Incorporated, 2010). Thus it is important to manage or treat dementia-related sleep problems *before* institutionalisation.

This chapter reviews the treatment options for sleep disturbances among older people, particularly PWD. The focus is on treatments for symptoms of insomnia and circadian dysthymia rather than sleep problems attributable to other clinical sleep disorders. This is because these are the most common and disruptive sleep problems for PWD and carers, and because other sleep disorders (e.g. OSA and RLS) already have more well-defined treatment pathways (Hening, et al., 2004; Loewen, et al., 2010; McCurry & Ancoli-Israel, 2003; Vitiello & Borson, 2001).

Pharmacological options are often used for problem sleep and these are briefly reviewed below. However, sleeping medications and exogenous melatonin have limited efficacy and potential safety issues, and are not recommended for routine prescription or long-term use by older people. Therefore the majority of this chapter focuses on non-pharmacological therapies for improving sleep. Due to the variable nature and experience of dementia, interventions that offer a combination of therapies could be useful, particularly for PWD living in the community, for whom the delivery of such therapies is dependent on their cooperation and their informal carer. The literature review identifies a lack of community-based and person-centred therapeutic trials for improving the sleep of PWD and their family carers, which leads to the aims of Study 4, a feasibility trial of such therapies in a Wellington-based community sample.

3.1 Pharmacological Treatments

3.1.1 Pharmacological Approaches for Improving Sleep Quality or Duration

As noted in Chapters 1 and 2, many older people, including PWD and their carers' report symptoms of insomnia. Unsurprisingly then, there is also an increased use of sleeping medications by older people (both prescribed and over-the-counter; Vitiello & Borson, 2001). Benzodiazepines (BZD), such as temazepam) have been widely used. They bind to the GABA-BZD receptor complex, which has general sedating effects that contribute to sleep onset, as well as actions on muscle relaxation, anti-seizure and anti-anxiety effects. Non-benzodiazepines (NBZD such as zolpidem, or zopiclone) are a newer form of sedative that also binds to the GABA-BZD receptor complex but they tend to bind more selectively to the receptor responsible for sleep-related sedation. Therefore NBZD are typically more effective and have less adverse side effects. Many BZD and BNDZ are approved by the Food and Drug Administration (FDA) for short-term treatment of transient insomnia. There are also many non-prescriptive hypnotics available which are available over-the-counter and are self-administered. These include antihistamines, valerian (a herbal compound), and alcohol (Lee-Chiong & Harrington, 2008; Vitiello & Borson, 2001).

Sleep medication use within older people.

In a cross-sectional study of 9,393 Canadians aged over 60 years, 17% reported using sleeping medications and 9.2% were using BZD or NBZD (Neutel & Patton, 2010). Those aged over 80 years were 2.6 times more likely to be taking these compared to those aged 60-69 years ($p<0.05$). Those with comorbid physical or mental health problems also had increased likelihood of using BZD or NBZD ($p<0.05$). In a smaller study of older Canadians (N = 176, mean age = 74 years), 27% reported using over-the-counter medications for their sleep (Sproule, Busto, Buckle, Herrmann, & Bowles, 1999).

The use of sleeping medications is less well known in NZ. However, in 1982 it was identified that older people were more likely to be prescribed such medications (Reinkes, Sparrow, & Campbell, 1982), and that about half of those who were, were taking these medications in smaller doses, or less often than prescribed. A more recent study of over 1,000 British adults aged over 65 years, identified that despite the prevalence of sleeping problems being high (between 40-60%, based on the PSQI), only 13%

were using a medicine (prescribed or over-the-counter) to help with their sleep, and only 7% were using such medications three or more times a week (Venn & Arber, 2009). In subsequent interviews with a subsample of 62 participants from this survey, Venn and Arber (2012) identified that older people were reluctant to talk to their doctor about sleep problems. Many presumed that they would be prescribed sleeping medications, which they considered undesirable. Reasons for this resistance to medicate included: participants being reluctant to medicate what they considered a normal part of ageing; a desire to maintain control over their health and use of medications; beliefs surrounding the unnatural or inappropriate nature of sleeping medications; as well as concerns around the harmful nature and/or becoming reliant on such medications.

The number of randomised controlled trials (RCTs) incorporating older adults and objective sleep monitoring concerning BZD and BNZD are far fewer than in younger adults (Krystal, 2009). However, McCall et al. (2006) showed that they could be effective. In this multicentre, placebo RCT including 264 Americans aged 64-86 years with insomnia, they found that sleep onset latency improved by 33.5 minutes ($p<0.001$); time awake after sleep onset reduced by 25.3 minutes ($p=0.013$); and overall sleep duration increased by almost an hour ($p<0.001$) after a two-week trial of eszopiclone. Participants' reports also indicated significant improvements to sleep. Studies of younger adults indicate that NBZDS have the potential to also improve sleep architecture, for example, increase SWS and REM (Bastien, LeBlanc, Carrier, & Morin, 2003). However, McCall et al. did not find this to be the case in their sample of older people.

Other studies show less promising results. A meta-analysis of 24 studies including insomniacs aged over 60 years ($N = 2,417$) indicated that, on average, NBZDs increased participant-reported sleep duration at night by only 25 minutes and the number of awakenings reduced by only 0.6 across the night (Glass, Lanctôt, Herrmann, Sproule, & Busto, 2005). While these changes were significant ($p<0.001$), overall the studies reported an effect size of just 0.14 (range = 0.05 - 0.21) for improved sleep. This indicates generally low efficacy of these newer forms of sedatives (with an effect size of 0.3 considered to indicate a medium effect and 0.5 a large effect).

Both McCall et al.'s and Glass et al.'s studies were conducted with relatively healthy older people. Trials considering the efficacy and safety for sleeping medications with older people who have comorbid medical or psychiatric disorders are lacking (Krystal, 2009). The side effects of medications should be closely monitored by a clinician. While some individuals do not report any adverse effects to sleeping medications, many risks have been identified. Side effects include drowsiness and fatigue during the day, headaches, nausea, dizziness, nightmares, as well as cognitive and psychomotor impairment (Glass, et al., 2005; Monti & Monti, 2010; Sproule, et al., 1999). Such side effects translate to increased risk of falls, hip fractures, and road traffic accidents. For these reasons, sedating medications may be particularly risky for use amongst carers if they are required to provide care during the night. Medications with sedating effect on the central nervous system can also exacerbate respiratory disorders including sleep apnoea (Ebly, Hogan, & Fung, 1997; Kripke, 2000; Krystal, 2009).

Older people typically experience increased side effects because they metabolise drugs more slowly and therefore the effects are cumulative (Krystal, 2009; Lader, 1986; Salzman, Shader, Greenblatt, & Harmatz, 1983). Because of these side effects, sleeping medications are not recommended for long-term use. However, Neutel (2005) found that half of older Canadians who use them appear to do so for years. Misuse is likely due to a tolerance that many develop and the aggressive rebound of symptoms once such medications are withdrawn (Kim, et al., 2009; Reynolds, et al., 1985). Studies concerning the effects of sleeping medications over a period longer than two months are lacking in older people, so this is still an area of concern (Krystal, 2009; Lee-Chiong & Harrington, 2008).

Sleep medication use within PWD.

Case reports indicate that some PWD have improved sleep after using sleeping medications once the individual's optimal dose was reached (Shelton, Hocking, Guzman, & Demers, 1997). However, larger RCTs of sleeping medications for older PWD and nursing home residents in general show negligible or non-significant effects (McCarten, Kovera, Maddox, & Cleary, 1995; Monane, Glynn, & Avorn, 1996). This suggests that for PWD, sleeping medications, particularly at the standard geriatric doses, could be less useful than in healthier older people. Despite this, it appears that sleeping medications are over-prescribed and over-used for PWD, and there is evidence that their use increases with severity of disease and

institutionalisation (Elmståhl, Stenberg, Annerstedt, & Ingvad, 1998). A survey of 106 psychiatrists for older people across Australia and NZ in 2005 revealed that 64% rated BZD or other sedatives within their top three treatments of choice for PWD with insomnia (Greve & O'Connor, 2005). Also, 56% considered them as appropriate for dementia-related anxiety. In an American sample of 36 PWD/carer pairs living in the community, McCurry et al. (2005) found that approximately 30% of the PWD and 25% of the carers, were using a sleeping medication.

Antidepressants and antipsychotics are sometimes prescribed as an alternative treatment for sleep complaints, especially for older patients with comorbid conditions such as dementia and depression (Khachiyants, et al., 2011; Neutel & Patton, 2010; Staner, Demazieres, & Luthringer, 2010). In addition to sedating effects, such medications have been found to relieve some sundowning symptoms, including wandering and aggressiveness during wakefulness (Falsetti, 2000; Meguro et al., 2004; Vitiello & Borson, 2001). In Greve and O'Connor's (2005) survey, 77% of the psychiatrics rated antipsychotics within their top three treatments for sundowning symptoms, and 22% rated mood stabilisers as such. Such medications are not FDA approved for treating sleep problems and are not recommended for treating sleep problems alone (Bliwise, 2004). This is due to the lack of data concerning the effectiveness of this off-label use, as well as potential negative side effects, including excessive sedation, cardiac arrhythmia, hypotension, and the potential to exacerbate sleep problems such as RLS and PLMS (McCurry, 2000; Raskind, 1998; Ray, Griffin, Schaffner, Baugh, & Melton, 1987; Schneider, et al., 2006). Anticholinergic antidepressants may be particularly detrimental due to the already diminished amount of cholinergic activity apparent with dementia. The use of such medications could exacerbate cognitive impairment or sundowning behaviours (Staedt & Stoppe, 2005).

The AChEIs used to facilitate cognitive functioning for PWD have been considered useful for improving sleep due to their stimulation of the cholinergic system and potential to increase REM sleep (Schredl, Weber, Braus, & Heuser, 2000). Previous studies in healthy older participants and PWD found that AChEIs can improve REM sleep duration and intensity which was considered to contribute to improved cognitive functioning (Dos Santos Moraes et al., 2006; Schredl, Weber, Leins, & Heuser, 2001). However, such improvements have not been found in all studies (Ancoli-Israel, Amatniek, Ascher,

Sadik, & Ramaswamy, 2005; Cooke, Loredo, et al., 2006). Furthermore, a large survey of informal carers in America indicated that PWD who are taking AChEIs were 2.5 times more likely to also use sleeping medications (Stahl, Markowitz, Gutterman, & Papadopoulos, 2003). This indicates that AChEIs could be associated with disrupted rather than improved sleep. As noted in section 2.2.3, side effects of AChEIs can include insomnia and nightmares, which could paradoxically exacerbate sundowning behaviours and actually impair memory processing during sleep, especially if taken at night (Kitabayashi, et al., 2006; Rogers et al., 1998; Ross & Shua-Haim, 1998; M. Singer, et al., 2005). For these reasons, AChEIs are not recommended for treating sleep problems alone. In Greve and O'Connor's (2005) survey, only 2% of the psychiatrics rated AChEIs within their top three treatments for insomnia, but 34% rated them as such for sundowning symptoms.

In general, the mechanisms associated with precipitating or perpetuating sleep problems are not addressed through using sleeping medications, or other psychotropic medications. They simply override or mask the symptoms. Due to the potential development of tolerance to such medications, symptoms of poor sleep are therefore likely to reappear and persist over time (Barter & Cormack, 1996; Neutel, 2005). The factors and behaviours that perpetuate sleep problems also need to be directly addressed (Spielman, et al., 1987).

3.1.2 Pharmacological Approach for Improving Circadian Dysrhythmia

Many of the sleep problems associated with ageing and dementia are associated with changes in the circadian timing system. A decrease in melatonin synthesis has been identified as one of the precipitating factors associated with fragmentation of the sleep/wake pattern (Liu, et al., 1999; Touitou & Haus, 2000; Wu & Swaab, 2007). Older adults with insomnia have also been noted to have lower than normal melatonin synthesis (Garfinkel, Laudon, Nof, & Zisapel, 1995). Trials using exogenous melatonin are becoming more common in an attempt to boost the amplitude and stability of the SCN pacemaker's intrinsic rhythmicity and, in turn, improve sleep.

Melatonin use within general population.

Well-controlled studies have also shown that exogenous melatonin taken in the evening can advance sleep onset within an hour or two of administration (De Jonghe, Korevaar, Van Munster, & De Rooij, 2010; Sack, Brandes, Kendall, & Lewy, 2000; Sack, Lewy, & Hughes, 1998). Meta-analyses of oral melatonin used across age groups affected by insomnia have found that there are typically significant improvements reported with regards to sleep onset latency, sleep efficiency and sleep duration. However the size of these "improvements" may not be clinically significant. For example, Brzezinski et al.'s (2005) meta-analyses of 17 studies concerning 284 adult insomniacs (who were otherwise healthy), found that the average reduction in sleep onset latency (typically measured by actigraphy or polysomnography) was only 4.0 minutes (95% CI = 2.5-5.4). Sleep efficiency increased by only 2.2% (95% CI = 0.2- 4.2) and sleep duration at night increased by just 12.8 minutes (95% CI = 2.9- 22.8). A smaller study (N=12) of older people (mean age 76 years, SD = 8) showed similar results. Those trialling 2mg of melatonin for three weeks had reduced wake time after sleep onset (measured using actigraphy) compared to controls (49 minutes vs. 73, $p<0.001$) and greater sleep efficiency at the end of the trial (83% vs. 73%, $p<0.001$). Such results are not consistent between studies, and participants' reports do not always indicate success (Garfinkel, et al., 1995; Olde Rikkert & Rigaud, 2001).

Compared to sleeping medications and psychotropics, melatonin has been deemed relatively safe to use for adults, including people aged over 60 years (Buscemi et al., 2005; Buscemi et al., 2006). Reported side effects have included nausea, dizziness and sleepiness, but these effects were not significantly greater than those reported by individuals taking a placebo, which is promising. However, the long-term effects of exogenous melatonin are unknown and there are concerns regarding its impact on insulin sensitivity and blood pressure due to the widespread presence of melatonin receptors throughout the vascular system. These effects would be of particular concern for older people (Lee-Chiong & Harrington, 2008; Wider & Pittler, 2010). For these reasons exogenous melatonin is not recommended for routine clinical use by the FDA, although melatonin supplements are readily available as an alternative over-the-counter "sleep-aid" in the USA (Vitiello & Borson, 2001). In New Zealand, melatonin is available on prescription only (DermNet NZ, 2014).

Melatonin use within PWD.

Trials of melatonin in groups of PWD have had mixed results. Some studies, including RCTs, show that melatonin improves the regularity of sleep timing, reduces daytime sleeping and sundowning symptoms, and results in some cognitive improvements (Brusco, Fainstein, Marquez, & Cardinali, 1999; Cardinali, Brusco, Liberczuk, & Furio, 2002; Cohen-Mansfield, Garfinkel, & Lipson, 2000; De Jonghe, et al., 2010; Jean-Louis, Von Gizycki, & Zizi, 1998). However other studies, including larger, multicentre RCTs, indicate that the effects of melatonin on objective sleep measures or dementia-related behaviours are non-significant (Cardinali & Karasek, 2010; Gehrman et al., 2009; Serfaty, Kennell-Webb, Warner, Blizard, & Raven, 2002; C. Singer, et al., 2003). Mixed results are likely due to variations in study design, dosages of melatonin, and the heterogeneous nature of research participants with dementia. The variable and fluctuating nature of sleep disturbances and behaviours exhibited by PWD also make it more difficult to objectively measure the efficacy of treatments.

For people with MCI, melatonin appears more likely to improve subjective sleep ratings as well as waking performance and cognitive functioning (Furio, Brusco, & Cardinali, 2007). However, as dementia progresses, the efficacy of melatonin is considered likely to decrease (Wider & Pittler, 2010). This is due to the dementia-related deterioration of the pathway between the pineal and the SCN, as well as deterioration of the melatonin receptors in the SCN, particularly in Alzheimer's disease (Liu, et al., 1999; Wu, et al., 2007; Wulff, et al., 2010).

Comorbidities and other medications also need to be considered. The increased risks of cerebrovascular events associated with melatonin use mean that additional caution should be taken for people with VaD (Eeles, 2006). Commonly used drugs such as beta blockers may inhibit the release of exogenous melatonin and counter its reception, or create adverse effects (Perri et al., 2005).

Recent studies trialling a combination of melatonin with non-pharmacological therapies have shown promising results (Dowling et al., 2008; Riemersma-van Der Lek et al., 2008). For example, in the Dowling et al. (2008) RCT of melatonin combined with bright light therapy (BLT), a 116 minute reduction in daytime sleeping was recorded with actigraphy (from a mean baseline of 315 minutes, SD = 129 minutes). A non-significant increase in night time sleep was also reported (from a mean baseline of

459 minutes, SD = 109 minutes). However further research is required to develop guidelines with regards to the safe and efficient use of melatonin for older people and PWD.

3.2 Non-Pharmacological Therapies

3.2.1 Cognitive and Behavioural Strategies for Problem Sleep

Cognitive behaviour therapy for insomnia (CBT-I) comprises a set of strategies which target and address the perpetuating factors and processes contributing to poor sleep and insomnia. The strategies include: sleep hygiene education (behavioural recommendations with regards to lifestyle and environmental changes); stimulus control (conditioning the individual to associate the bed and bedroom with sleep, by eliminating non-sleep related activities in bed); sleep compression (restricting time in bed in order to reduce time spent awake); relaxation techniques (e.g., progressive muscle relaxation or meditation); challenging dysfunctional beliefs about sleep; and practising mindfulness (in order for individuals to cope with their current situation and reduce the impact that over-thinking or sensationalising sleep problems can have; Gehrman & Goonerante, 2010; P. Montgomery & Dennis, 2004). Formal CBT-I is implemented over several individual or group sessions with a trained provider. Such therapies have been identified as effective in 70-80% of younger adults with insomnia, with regards to subjective reports of sleep onset latency, sleep duration and wake after sleep onset (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995). A recent meta-analysis of RCTs of CBT-I used for 282 highly screened healthy insomniacs aged over 60 years, reported modest effects (P. Montgomery & Dennis, 2003). Self-reported sleep onset latency improved by 3 minutes, sleep duration improved by 15 minutes, and wake after sleep onset improved by 22 minutes. Some studies using polysomnography support these subjective findings, particularly concerning improved wake after sleep onset (Morin, et al., 1999; Morin, Kowatch, Barry, & Walton, 1993). A concern is that the effects of CBT-I may not be long lasting and follow up may be required (P. Montgomery & Dennis, 2004). However Morin et al. (1999) reported that the effects could last up to two years for participants using CBT-I, whereas those trialling a BZD or placebo had significantly poorer sleep after two years. Furthermore, greater subject, clinician and family satisfaction was reported for those on CBT-I treatment, compared to BZD treatment. These results indicate that CBT-

I is a preferable treatment for older people and it has a much greater benefit-to-risk ratio than the pharmacological options outlined above.

For older PWD and carers, not all aspects of the traditional clinical process of CBT-I may be possible or safe, due to cognitive impairment (e.g., restricting sleep opportunities, cognitively challenging dysfunctional beliefs, and actively relaxing). However a strength of CBT-I is the flexibility to tailor the treatment, using suitable strategies. This makes it a promising, person-centred option. Aspects of CBT-I, particularly sleep hygiene education and modification, can be delivered informally and successfully via reading material (Mimeault & Morin, 1999). Sleep hygiene education typically includes information on napping (e.g., not napping too close to bedtime or for too long), maintaining a regular sleep schedule throughout the week, sleeping in a suitable environment (i.e. minimising light and noise in the bedroom and maintaining a comfortable temperature at night), avoiding caffeine and alcohol after lunch, using relaxation techniques prior to bed, as well as information about environmental time cues that affect sleep (Gehrman & Goonerante, 2010; National Institute of Aging, 2009; National Sleep Foundation, 2011). Sleep hygiene education, as well as information about "normal" sleep and common sleep disorders, allows PWD and their carers to be empowered to change their own sleep rather than use a medication to mask the problem (Eeles, 2006; McCurry, et al., 2005).

McCurry and colleagues are at the forefront with regards to trialling non-pharmacological interventions for PWD and their carers living in the community (McCurry, et al., 2005; McCurry, Logsdon, Vitiello, & Teri, 1998). In a controlled study of dementia carers with sleep problems, McCurry et al. (1998) trialled a multicomponent behavioural intervention. This included sleep hygiene, as well as sleep compression, stimulus control and carer-related support. They found that 60% of carers had improved sleep. For example, diary recorded sleep efficiency improved from an average of 73% (SD = 1%) at baseline to 82% (SD = 9%) after four weeks of treatment. Compared to controls, the treatment group had significant improvements in their PSQI scores, which averaged 10.8 (SD = 3.4) at baseline compared to 7.8 (SD = 3.3) post treatment and 6.2 (SD = 3.6) at three-month follow up (whereas the control groups scores remained between 10-12; McCurry, et al., 1998). Similar results have been reported from a recent Australian study, which found that a sleep education package for dementia carers

significantly improved their self-reported sleep onset latency (average decrease=10 minutes) and sleep duration (average increase=31 minutes) at night, as well as reducing their PSQI scores (Sacre, 2010). These studies suggest that older carers can benefit from simply having more knowledge about their sleep and about possible techniques to improving it. Making tailored information easily available is considered vital for engaging dementia carers (who are typically busy individuals) and successfully improving their sleep and that of the person in their care (McCurry, et al., 2005).

Using CBT-I techniques for PWD is complicated by the severity and symptoms of dementia as well as carer motivation (Petit, et al., 2011). However McCurry et al. (2003) found that carers of PWD could successfully set up and implement strategies to actively promote better sleep hygiene for PWD. Carers in an active CBT-I treatment programme were encouraged to set goals regarding the PWD's sleep timing and the sleeping environment, to be implemented and monitored over several weeks. Compared to a control group who received hand-outs only, the active treatment group were more likely to maintain consistent bed and rise times (83% and 96% vs. 38% and 59% respectively), reduce daytime napping (70% vs. 28%) and increase activity in the day (86% vs. 7%). These were all significant differences ($p<0.01$). The results emphasise the importance of carer effort as well as the external support required for successfully changing a PWD's sleep behaviour.

In summary, a review of the current literature indicates that sleep hygiene education and behaviour modification may be preferable to pharmacological approaches for improving sleep of many PWD. These non-pharmacological approaches address some of the factors that perpetuate symptoms of insomnia and sleep disruption, have much lower risk of side effects, and are more likely to be effective for longer periods of time than pharmacological approaches. Furthermore, non-pharmacological approaches can also improve the sleep of the carer.

3.2.2 Chronotherapeutics for Circadian Dysrhythmia

The neurodegenerative changes in the SCN circadian pacemaker that underlie common sleep disruptions in dementia also present potential targets for therapeutic strategies. Chronotherapeutics is "the controlled exposure to environmental stimuli that act on biological rhythms, in order to achieve

therapeutic effects in the treatment of psychiatric conditions" (Benedetti, Barbini, Colombo, & Smeraldi, 2007, p. 509). Successfully implemented chronotherapies can enhance the strength of the SCN pacemaker signals necessary for sleep timing and help restore internal synchrony between the sleep/wake cycle with other circadian rhythms, as well as external synchrony with the day/night cycle (Wirz-Justice, et al., 2008). As noted in section 1.2.1, the rhythmicity of the SCN can be enhanced by reinforcing the pattern of the individual's exposure to the environmental time cues to which the SCN is sensitive, notably bright light and physical activity. Interest has increased in the use of chronotherapies for PWD, given the questions around the efficacy and safety of pharmaceutical approaches, and the well-established efficacy of chronotherapies for people with circadian rhythm disorders and seasonal affective disorder (Lack & Wright, 1993; Wirz-Justice, et al., 2008).

Timed exercise as chronotherapy.

With age, there is typically a reduction in physical activity, as well as general activities of daily living (Sirven & Mancall, 2008). Martin et al. (2006) found that older people living in residential homes in America (N = 492) were observed to be spending a quarter of their daytimes in bed. Reduced physical activity is somewhat related to increased likelihood of physical ailments and frailty, however physical activity is still considered important for improved sleep and health in general (Youngstedt & Freelove-Charton, 2005). Reduced amounts of exercise have been independently associated with self-reported sleep problems (Bazargan, 1996; Sherrill, Kotchou, & Quan, 1998). Conversely, regular exercise may reduce the risk of insomnia, increase sleep duration, and improve sleep architecture (increasing the proportions of REM and SWS), as well as act as a stimulus to stabilise rhythmicity of the SCN pacemaker (Driver & Taylor, 2000; K. Morgan, 2003; Youngstedt, 2005). In a meta-analysis conducted by Kubitz, Landers, Petruzzello and Han (1996), it was found that older men (60-72 years) who were defined as fit and active had greater sleep efficiency and a greater proportion of SWS compared to sedentary controls.

Research addressing the therapeutic effects of exercise in ageing and dementia is somewhat limited. Many trials have taken place in institutionalised settings and do not include sleep as a primary outcome (Heyn, Abreu, & Ottenbacher, 2004). However those that have directly measured sleep have found that exercise regimes for older people, including PWD, can have a positive effect (King, et al.,

1997; Li et al., 2004; McCurry, et al., 2011; Namazi, Zadorozny, & Gwinnup, 1995; Van Someren, Lijzenga, Mirmiran, & Swaab, 1997). The results of these trials vary, as do trials in younger populations. This is likely to be related to variations in the timing, type, and intensity of exercise undertaken, as well as the variability within and between samples.

Different types and intensity of exercise have been investigated with regards to their effects on sleep, primarily with younger, healthier participants who were more accustomed to vigorous exercise (Driver & Taylor, 2000). However, it has been argued that people who are more sedentary and/or older may be more sensitive to the effects of exercise and therefore less strenuous protocols might be sufficient to achieve a positive impact on sleep (Driver & Taylor, 2000; Kubitz, et al., 1996). Low intensity exercises (e.g. slow walking, tai chi, light stretches or housework; Ministry of Health, 2010)) have been shown to improve subjective sleep quality and daytime sleepiness of older adults (Li, et al., 2004). Tanaka et al. (2001) found that, combined with a short intended nap after lunch, 30 minutes of low intensity exercises at around 5pm significantly improved actigraphically recorded sleep variables within a sample of six Japanese adults who had reported difficulties sleeping (average age of 72 years, SD = 5.2). After their four week trial, wake time after sleep onset improved from 83.2 minutes at baseline (SD = 23.4) to 28.2 minutes (SD = 4.31, $p<0.01$); night time sleep efficiency increased from a baseline average of 73.9 % (SD = 4.24%) to 87.7% post intervention (SD = 1.70, $p<0.01$); and instances of unintentionally dozing off in the daytime decreased from a baseline mean of 1.7 times (SD = 0.41) to 0.5 times (SD = 0.24, $p<0.05$). Alessi, Yoon, Schnelle, Al-Samanai ,and Cruise (1999) found that their intervention of a daily exercise programme, plus sleep hygiene modifications conducted by nursing staff over 14 weeks, also improved sleep of 29 incontinent nursing home residents (mean age = 88.3 years). sleep efficiency showed greater improvements across the trial (52% pre trial, to 63% post trial) compared to those with sleep hygiene modifications alone (pre-trial mean=67%, post-trial mean=66%, $p=0.045$). For some, nursing staff s' observations concerning agitated behaviour also indicated improvements.

Moderate exercise (e.g., brisk walking or cycling, swimming, heavy housework; Ministry of Health, 2010) and high intensity exercise (e.g. fast jogging or cycling, tennis or muscle strengthening exercises) also appear to improve sleep. For example, one RCT of moderate exercise (30-40 minutes, four

times a week for 16 weeks) targeted older people with sleep complaints (but were otherwise healthy; King, et al., 1997). Significant improvements were found in subjective estimates of sleep onset latency (mean reduction=11.5 minutes, p = 0.007), and sleep duration (mean increase=42.0 minutes, p =0.05) as measured by the PSQI. Subjective sleep ratings also improved in the exercise group (mean global PSQI scores reduced from 8.7 to 5.3, p<0.001), but remained unchanged in the control group (King, et al., 1997). Trials vary in methodology, exercise protocols, and sleep measures, but there does not appear to be a dose relationship with regards to intensity of exercise and effects on sleep. Even low intensity exercise can have positive effects on sleep (Enderlin & Richards, 2006). This is an encouraging prospect for older people who have sleep problems as well as physical or cognitive limitations.

The timing of physical activity in the circadian clock cycle can affect the expression of melatonin and sleep timing. *Figure 3.1* shows a phase response curve for the phase shifting effects of exercise on the circadian rhythm of melatonin of young healthy men (N = 38). On three consecutive days, participants undertook an hour of high intensity exercise at the same time relative to the individual's circadian melatonin rhythm. The three-day exposure was then repeated at different times in the melatonin rhythm (lighting conditions remained dim throughout the protocol; Buxton, et al., 2003).

The amplitude of the phase response curve for older people may not be the same as in *Figure 3.1*, however it is expected that phase delays would still occur in response to exercise in the morning and phase advances in response to exercise in the evening (Baehr et al., 2003). This is the opposite pattern to the phase shifting effects of light (see below). Independent of the phase advancing effects of evening exercise on the SCN pacemaker, sleep may be delayed by exercise too close to bedtime because it causes an increase in temperature, contrasting with the body's need to cool down in order to create a window for sleep onset (Gehrman & Goonerante, 2010). However for some, exercise close to bedtime appears to promote sleep onset or have little effect at all. Differing results may be due to how accustomed individuals are to physical activity and their homeostatic sleep drive (Youngstedt & Freelove-Charton, 2005). Older retired people may have more flexibility about when physical exercise can take place compared to younger working adults. This makes regular timed exercise a potentially useful therapy for improving their sleep.

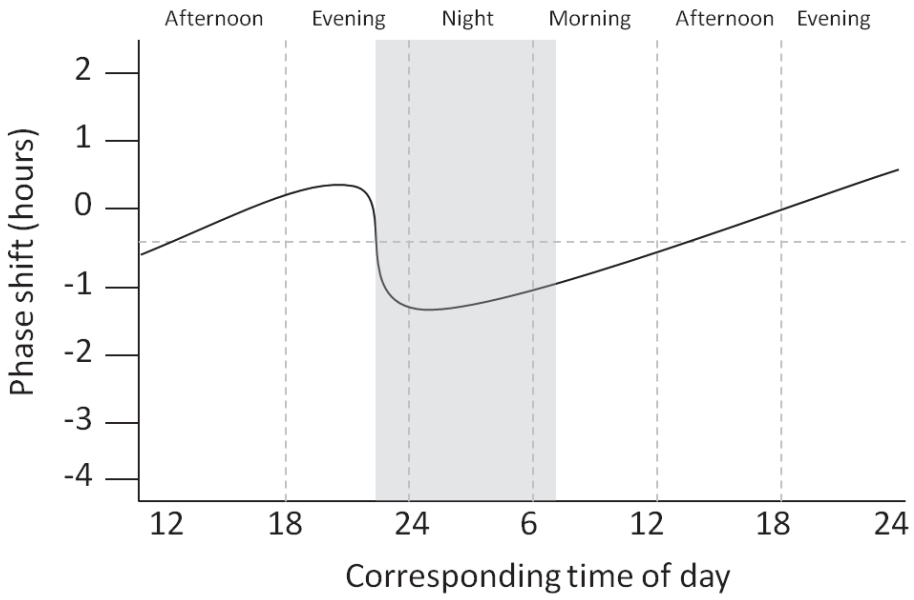


Figure 3.1. Phase response curve to 1 hour high intensity (stair climbing) exercise at different times in the SCN pacemaker cycle.

Grey shading represents typical night time sleep, redrawn from (Buxton, et al., 2003, p. 720).

As indicated in section 2.2.2, PWD may exhibit phase advances of sleep timing or, more typically, phase delays. The aetiology of these changes in sleep timing is not clear. For example, increased daytime sleep could prematurely lower the homeostatic pressure for sleep, leading to a delay in sleep onset at night. On the other hand, an unstimulating environment or depressed mood in the evening could promote an increased drive for sleep at an earlier time (Bliwise, 2004). Because of these differences, there are no standardised recommendations for the timing of therapeutic exercise as a sleep intervention for PWD. Van Someren, Lijzenga, et al. (1997) trialled a three month programme of therapeutic exercise for eight healthy older men that entailed bouts of moderate activity for 90 minutes, three times per week, at around noon (which would have minimal phase shifting effect on the SCN pacemaker - see *Figure 3.1*). They found significant reductions in the fragmentation of the participants' rest/activity timing, as indicated by reductions in intradaily variability (calculated from actigraphy data).

For PWD, the timing of exercise may be governed by when their more "difficult" times of day occur (with regards to dementia-related behaviours). Furthermore a time is required in which their caregiver is most available to support them and when there are no conflicts with other daily

commitments/activities. Low intensity exercise programmes have been trialled and authors report that such programmes are feasible for PWD. Such programmes have been found to be effective for improving the sleep, as well as physical health (strength, flexibility etc.) and psychosocial functioning of PWD living in both institutionalised and community settings (Alessi et al., 2005; Alessi, et al., 1999; Arkin, 1999; Brill, Drimmer, Morgan, & Gordon, 1995; Heyn, et al., 2004; J. L. Martin, et al., 2007; Naylor et al., 2000; Teri et al., 2003; Teri et al., 1998). Maintaining routine physical activity may also help manage symptoms of sundowning, if provided at a regular time of day on a consistent basis (Khachiyants, et al., 2011).

Among older people with increased likelihood of physical ailments, frailty, and reduced memory for places, low intensity exercises which can take place within the home (e.g. senior exercise videos or actively helping with the housework) or an accompanied local walk, are ideal (Barry & Eathorne, 1994; Heyn, et al., 2004). Naylor et al. (2000) conducted a 14-day trial of structured physical and social activities (e.g. walking and aerobic exercise followed by interactive games) for residents of a nursing home, some of whom had mild dementia. Activities took place over 90 minutes in the morning and evening. Sleep and circadian timing were measured using polysomnography and core body temperature. No significant results were found with regards to sleep timing and rhythmicity. However those participating in the active therapy had an increase in the percentage of SWS in the first half of the night from a mean of 11.8% (SD = 2.0) to a mean of 17.0% (SD = 3.1%, $p < 0.01$), as well as improved performance on a procedural memory task ($p = 0.03$). Control participants showed no significant changes. These findings highlight the benefits that reinforcing the environmental time cues can have on older people and PWD's sleep and memory. However, the sleep monitoring protocol used was rigorous and costly and would not always be well tolerated by PWD (Rose, et al., 2011; Yesavage, et al., 2003). For people with more severe dementia than those taking part in the study of Naylor et al. (2000), and for PWD living in the community, such a protocol could be too demanding.

McCurry and colleagues (2005; 2011) have trialled several non-pharmacological interventions for PWD living in the community. Their physical activity intervention involved 30 minutes of walking per day (no specific time) for as many days as possible over two months. This had a significant impact on reducing

the amount of time spent awake at night when exercise was prescribed alone (the mean decreased from 154 minutes at baseline to 128 minutes after the intervention, $p = 0.01$). However, an even greater improvement was seen when the exercise regime was combined with sleep hygiene modification and BLT (see below), with a decrease in the time awake at night from a mean of 121 minutes at baseline to 89 minutes after the intervention ($p = 0.01$; McCurry, et al., 2011). However, McCurry et al. (2010) also highlight the challenges of compliance concerning PWD's long-term adherence to a therapeutic exercise programme, regardless of the positive effects on health.

In summary, interventions involving therapeutic exercise can have positive effects on sleep as well as general health and wellbeing. Trials specifically involving PWD are limited, especially in the community setting. However results are promising with regards to such interventions being feasible, with the correct approach and support. An important factor is that exercise as a therapy "needs to be prescribed on an individual basis and approached in a safe way to make it rewarding to the participant and promote physical wellbeing" (Driver & Taylor, 2000, p. 400). For some older people and PWD, programmes involving physical activity may not be as easy and compliance is likely to become a problem. This might especially be the case for those living in the community who may have less external support. The literature reviewed indicates that a dyadic approach could be useful in which the informal carer is involved and supported with regards to the therapy targeted at the PWD in their care. One option is to offer a physical activity protocol as an option in a multicomponent approach, allowing participants to choose or combine therapies applicable to them in order to maintain a person-centred methodology that places less burden on PWD and carers.

Bright light therapy (BLT).

Sections 1.2.1, 1.2.3, and 2.2.2 described the mechanisms by which light entrains the SCN via the RHT, and its relationship to melatonin, and how this mechanism is impaired with ageing and dementia (Czeisler & Buxton, 2011; Swaab, et al., 1985). Timed BLT can be used to treat disrupted sleep/wake patterns by normalising their relationship to other external rhythms (Dowling & Mastick, 2010). It can achieve this through increasing the amplitude of the rhythmic output from the SCN to the pineal gland as well as stimulating the vasopressin-secreting neurons in the remaining areas of the SCN, thereby improving

circadian entrainment to the day/night cycle (Liu, et al., 2000; Swaab, Van Someren, Zhou, & Hofman, 1996).

Bright light therapy can be administered via timed natural light or exposure to a light therapy device (LTD). Types of LTDs include a light box placed within a metre of the individual, a light visor worn on the head, increasing the ambient light via special light fittings, or dawn-dusk simulation via computer-controlled lighting in the bedroom (Dowling & Mastick, 2010; Forbes, 2009; Wirz-Justice, et al., 2008). The effects of light on the SCN pacemaker are moderated by three factors: the spectral composition; intensity; and timing of exposure. These all need to be considered when using BLT, along with specific recommendations for older people and PWD.

Bright light therapy is considered to have much lower risk than pharmacological treatments. However there are some contraindications which are of particular concern for older people and PWD. As eye conditions such as cataracts and glaucoma are much more likely in older people and PWD, bright light can cause increased sensitivity and discomfort (Bradford, 2004; Wirz-Justice, et al., 2008). Some of the medications used by older people and PWD (e.g. antidepressants and anti-rheumatic medications) can also increase photosensitivity (Cloyd & Conway, 2008). Side effects related to these contraindications are more likely to occur when using an artificial LTD rather than natural sunlight due to the nature of the exposure. Although natural light is potentially more intense, the sensation of brightness (luminance) and glare can be much greater when using an LTD, due to the decreased distance and different angle between the light source and the eye compared to natural light which is broader spectrum and further away (Wibom, 1993). Despite this, previous research typically uses LTDs. This is likely due to the ability to more rigorously control the spectral composition and intensity of exposure and therefore produce more reliable results.

Traditionally LTDs use broad spectrum white light (Wirz-Justice, et al., 2008), however some recent studies highlight the increased sensitivity of the retinal ganglion cells that project to the SCN to light of shorter wavelengths within the visual spectrum (Brainard et al., 2001). In younger adults, blue light alone has been shown to produce a faster response, a greater suppression of melatonin, and increased feelings of alertness, compared to lights of longer or mixed wavelengths (Brainard, et al., 2001; Lockley et al., 2006). Small studies of older people and PWD using blue light also show some promise for supressing

melatonin (Figueiro, 2008; Figueiro, Lesniak, & Rea, 2011). However, blue wavelength light is generally not recommended for use with older people. The age-related thickening and yellowing of the eye's lens filters out shorter wavelength light and can create increased sensation or glare during exposure. Furthermore, short wavelength light could contribute to macular degeneration of older people's eyes and seem obscure or confusing to PWD. Therefore broad spectrum white light has been recommended (Dowling & Mastick, 2010; Wirz-Justice, et al., 2008).

The intensity of the light exposure is important with regards its effects on the SCN pacemaker. As noted previously (section 2.2.2), older people, particularly PWD, are seldom exposed to light that is more intense than 2000 lux (Ancoli-Israel, Klauber, et al., 1997; Campbell, et al., 1988; Shochat, et al., 2000). This impacts the activity within the retinal ganglion cells and RHT, and subsequently the SCN (Van Someren, et al., 2005). A dose of 10,000 lux is recommended for efficient light therapy (the equivalent of skyoutdoor light 40 minutes after sunrise; Wirz-Justice, et al., 2008). However light at half this intensity can still have an effect, and the degrees of effect are considered somewhat dependent on the intensity that the individual is accustomed to (Duffy & Wright, 2005). The sensitivity of the SCN begins to saturate in response above an exposure of 1000 lux (Zeitzer, et al., 2000) and much of the research involving PWD has used ambient light of 2000-4000 lux.

Timed light exposures can be an effective therapy for adults with circadian rhythm disorders, depression, or seasonal affective disorder (Chesson, 1999; Lack, Wright, Kemp, & Gibbon, 2005; Sumaya, Rienzi, Deegan Ii, & Moss, 2001). *Figure 3.2* shows a phase response curve for single exposures to 6.7 hours of bright light (10,000 lux) at different times in the melatonin circadian rhythm of young healthy adults (N = 21; Khalsa, et al., 2003). Exposure to light in the morning causes phase advances of the SCN pacemaker, while exposure to light in the evening causes phase delays, and light in the middle of the day has minimal effect on phase. To achieve similar phase shifts in a clinical/real-world setting, LTDs are used for several weeks for around 30 minutes at day at an intensity of 10,000 lux. Longer periods of exposure are needed to achieve the same effect with less intense light, for example, 2-3 hours of exposure would be necessary at an intensity of 2000-3000 lux (Chesson, 1999; Duffy & Czeisler, 2009; Jewett et al., 1997; Wirz-Justice, 1998; Wirz-Justice, et al., 2008).

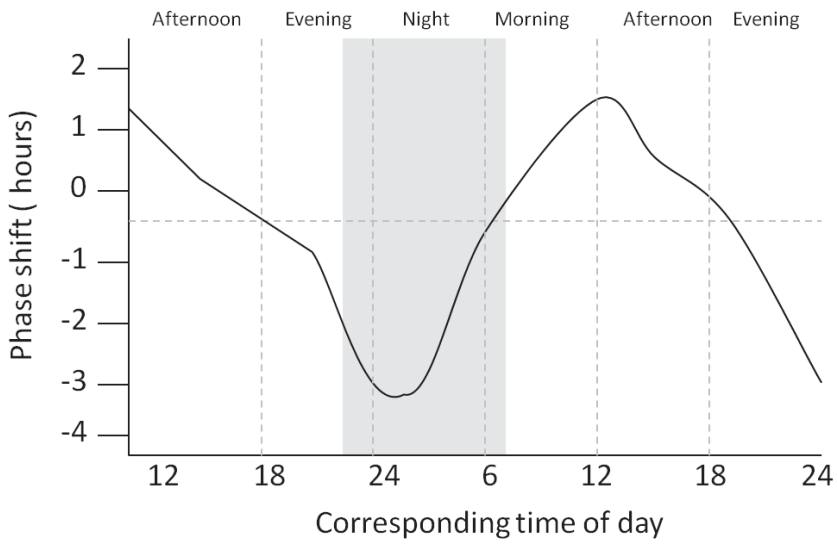


Figure 3.2. Phase response curve to bright light pulses (10,000 lux, for 6.7 hours) administered at different times in the SCN pacemaker cycle.

Grey shading represents typical night time sleep episode (redrawn from Khalsa, Jewett, Cajochen, & Czeisler, 2003, p. 948).

For older people in general, it seems logical to use BLT in the evening to help treat the commonly advanced sleep timing (Campbell et al., 1995). However, there are limited studies of older people trialling BLT. Because of the variability of changes in sleep/wake timing among PWD and challenges with accurately measuring circadian timing, there are no standard recommendations for the timing of light exposure for BLT for this population. There is also a risk of inadvertently increasing manic episodes by delivering BLT at an inappropriate time in the SCN pacemaker cycle (Chesson, 1999; Dowling & Mastick, 2010; P. Montgomery & Dennis, 2002; Song, et al., 2010).

A Cochrane review, of ten randomised and highly controlled trials which took place in institutionalised settings, found inconclusive results regarding the usefulness of BLT for dementia-related sleep problems (Forbes, 2009). Modest improvements to sleep and daytime functioning were reported at best, irrespective of the time of day participants were exposed to light. However the trend in the results suggested that BLT could improve sleep for PWD. Quasi-experimental research using crossover trials, single-case designs, and case reports showed that some institutionalised PWD had a positive response to BLT with regards to sleep, circadian regulation, and dementia-related symptoms (Dowling, Mastick,

Hubbard, Luxenberg, & Burr, 2005; Lovell, 1995; Okumoto, 1998; Satlin, Volicer, Ross, Herz, & Campbell, 1992; Sloane, et al., 2007). Mixed results could be due to the variable nature of dementia, the small sample sizes used (and therefore lack of statistical power to identify significant changes), as well as the use of different methods, intensities of BLT, and sleep measures.

Previous studies in institutionalised settings have found that BLT in the evening can have a positive impact on the sleep quality and the symptoms of sundowning of PWD (Satlin, et al., 1992). However exposure to bright light at this time is also more likely to delay sleep timing (Jewett, et al., 1997; Sloane, et al., 2007), which may be undesirable in cases when sleep timing is already very late. In addition, the evening is often a more challenging time for carers because of the increase in dementia-related behaviours and carer work load during this time, and use of a LTD could be a distraction from usual evening activities (Bachman & Rabins, 2006; Wirz-Justice, et al., 2008). Morning light exposure has also been shown to be effective for reducing daytime naps and consolidating the night time sleep of PWD (Sloane, et al., 2007). Improvements in circadian rhythmicity and sundowning behaviours have also been reported (Ancoli-Israel, Martin, Kripke, Marler, & Klauber, 2002; Mishima et al., 1994). However early morning light can also advance sleep phase which may be undesirable for those PWD who go to bed or wake unusually early.

As with therapeutic exercise, it appears that for older people and PWD, BLT could be more useful to reinforce rhythmicity in the SCN rather than shift its timing. Increased light in general seems to be effective for reinforcing the regularity of circadian timing and improving circadian stability (Ancoli-Israel, et al., 2002; Dowling, et al., 2005; Van Someren et al., 1999). With ageing and dementia, the endogenous rhythmicity of the SCN reduces in amplitude and robustness (as reviewed in section 2.2.2). A study with 10 older insomniacs in a Japanese nursing home (without dementia, average age 74.2 years), found that increasing the ambient lighting in a common room area to 2500 lux significantly increased participants' melatonin levels ($p<0.05$), and decreased their wake time at night ($p<0.05$; Mishima, et al., 2001). Studies comparing timing of BLT typically find that increasing the intensity of morning and/or all-day light exposure for PWD living in institutionalised care, have greater improvements to sleep duration at night and/or reduced daytime napping compared to evening light (Mishima, Hishikawa, & Okawa, 1998;

Sloane, et al., 2007; Van Someren, Kessler, et al., 1997; Yamadera et al., 2000). For example, Sloane et al.(2007) also installed ambient lighting delivering 2500 lux to living areas and compared the effects of light exposure at different times in three institutions for PWD. Actigraphic sleep recordings were compared between baseline and after three weeks of BLT for groups who had either 2.5 - 3 hours of exposure to 2500 lux in the morning, or in the evening, or all day (average 8.4 hours), versus a control group exposed to 500 lux. In this crossover trial, Sloane et al. found that those with more severe dementia who were exposed to light in the morning or all-day had the greatest increase to sleep duration at night (16 minutes, $p = 0.008$, and 14 minutes, $p=0.01$, respectively). These conditions appeared to improve the fragmentation of sleep. People with severe dementia had a significant reduction in number of sleep intervals at night after morning BLT (estimated mean change= -1.62 (95%CI= -2.87--0.36, $p = 0.01$). Others have found that dawn-to-dusk light simulation can have similar effects for some PWD living in institutions (Fontana Gasio et al., 2003).

Previous studies have typically used standardised sleep measures derived from actigraphic recordings. Van Someren et al. (1999) propose that the effects of BLT could be more reliably captured by applying their non-parametric methods to calculate and compare the interdaily stability, intradaily variability, and amplitude of the circadian activity rhythm. This is because of the variable nature of PWD's sleep/wake timing both between and within subjects. Van Someren, Kessler, et al. (1997). modelled and compared the rhythms of 22 PWD before and after increasing ambient lighting levels in living areas of a geriatric ward from a mean of 436 lux (range = 930 - 1417 lux), to a mean of 1136 lux, (range = 790-2190 lux). They found significant improvements in interdaily stability (more robust/predictable timing of sleep and wake, $p=0.002$), and in intradaily variability (reduced fragmentation of sleep across the 24-hour period, $p<0.01$) in people with severe dementia without visual impairments. The amplitude of the circadian activity rhythm did not change significantly ($p=0.18$) but the rhythm became smoother and less variable due to the improvements in the other factors

There are few community-based trials of BLT for PWD. The cost and feasibility of installing ambient LTDs in private living/dining areas is generally prohibitive, compared to institutionalised settings. Therefore light visors, boxes or natural light are used, the exposure to which typically requires active

involvement and encouragement by carers (McCurry, 2000). People with dementia undertaking BLT need to be able to understand and cooperate with the intervention in an ethical manner, i.e. without coercion or restraint. Light visors seem promising because of their simplicity of use and have been trialled in a sample of community-dwelling people with AD (Colenda, Cohen, McCall, & Rosenquist, 1997). Two hours of morning light of 2000 lux was reported to have no significant effect on actigraphic measures of sleep or circadian amplitude, although reanalysis of the data by Van Someren et al. (1999) suggested that there were improvements to intradaily stability ($p=0.02$). It should be noted that the Colenda et al. trial was relatively short (10 days) and light visors might not be the most effective and comfortable means for delivering light due to the angle and intensity of light delivered (Wirz-Justice, et al., 2008).

More recently, McCurry and colleagues (2005; 2011) conducted two RCT's for improving the sleep of PWD living in the community, using evening light delivered by light boxes. The first was a feasibility study ($N = 36$ PWD/carer community-dwelling dyads) and included a multimodal intervention of light, exercise, and sleep hygiene modification. The intervention was deemed successful in that the number of awakenings and time spent awake at night significantly reduced for the treatment group compared to the control group after the two-month trial (all $p<0.05$). However the individual impact that light had on sleep could not be determined due to the study design. The following trial included 132 community-dwelling pairs and compared a group using a light box alone to a group using therapeutic exercise (walking), or a combination of BLT and exercise plus active sleep hygiene modification (as per their 2005 study), or a control group. After two months of trialling the interventions, PWD using a LTD had a significant improvement in the amount of wake time at night, from a baseline mean of 141.8 minutes ($SD = 14.1$ minutes) to a mean of 110.2 minutes at follow up ($SD = 13.9$, $p = 0.04$). The effects of light were greater than those of exercise alone (mean improvement = 25.8 minutes, $SD = 11.0$, $p = 0.05$). When BLT was combined with the exercise and sleep hygiene modification, an even greater improvement was recorded (mean improvement = 32.4 minutes, $SD = 9.9$, $p = 0.01$). However, this difference is considered negligible given the amount of work added by the additional interventions. Despite the objective improvements, carers' reports of disturbed sleep of their family member with dementia (as indicated by their SDI scores; Tractenberg, et al., 2003) did not significantly improve, nor did their ratings

of depression. McCurry and colleagues (2005; 2011) used a light box which delivered 2500 lux, which participants were required to use for an hour to get any estimated effect. Light exposure was in the evening (within two hours of usual bedtime). Some carers (19%) reported that using the light box was difficult for them or required too much effort (McCurry, et al., 2011). The non-significant change in subjective improvement and issues with intervention burden could have been related to the difficult time of day and the length of required light exposure.

A more recent study examined the effects of light therapy delivered at a higher intensity, for a shorter time in the early morning. Friedman et al. (2012) used 4220 lux for 30 minutes, starting within 30 minutes of rising from bed. They also incorporated sleep hygiene modification but this was delivered more passively via conversation and a manual, rather than the structured and negotiated plans used in the studies of McCurry et al. This protocol was considered by the authors as less likely to increase carer burden. The actigraphic data and subjective assessments of insomnia symptoms of PWD and their carers sleep were compared to a group who received dim red light and the sleep hygiene information. The protocol had no positive effects for the PWD, whose time in bed and sleep efficiency actually decreased. It may be that the delivery of BLT was still not sufficiently intense considering the length of exposure (Wirz-Justice, et al., 2008). Despite the lack of improvement for PWD's sleep, Friedman et al. found that carers' sleep efficiency and sleep quality ratings improved, as did their self-reported symptoms of depression. This was evident for carers in both the bright light as well as the comparison group, suggesting a positive impact of sleep hygiene education alone on the sleep and mood of informal carers.

Although objective improvements to the sleep of PWD as a result of BLT appear modest, these changes are comparable to the effects of the pharmacological treatments reviewed above. Key points are that BLT does not have such adverse side effects, and BLT can be continued long-term by permanently changing the light fittings in living areas or establishing a routine whereby sufficient exposure to natural sunlight is achieved. One study trialling the long-term effects (average e= 15 months, SD =12 months) of ambient light changes showed positive effects for mood and cognitive functioning of PWD (Riemersma-van Der Lek, et al., 2008). Given the heterogeneity among PWD, it should be noted that some people do appear to benefit from BLT despite the lack of statistically significant findings for study groups. Rigorous

research design and analysis may well be masking the personal effects. Case studies of PWD trialling LTDs illustrate that individual benefits can be achieved. Okumoto (1998) describes a PWD living in an institution who trialled morning bright light (9:30-11:00) via a light box, intermittently over six months (54 days first baseline, followed by 34 days of BLT of 4000 lux, followed by 44 days of second baseline, and a further 46 days of BLT). The participant's sleep diary records (maintained by nursing staff) clearly showed more consolidated night time sleep during periods of BLT, which returned to their fragmented state when the LTD was removed (see *Figure 3.3*). These results are similar to a case of a participant with AD presented in the study of Van Someren, Kessker et al. (1997). Observations from nursing staff also suggested an improvement in sundowning type behaviours during periods of light exposure (Okumoto, 1998).

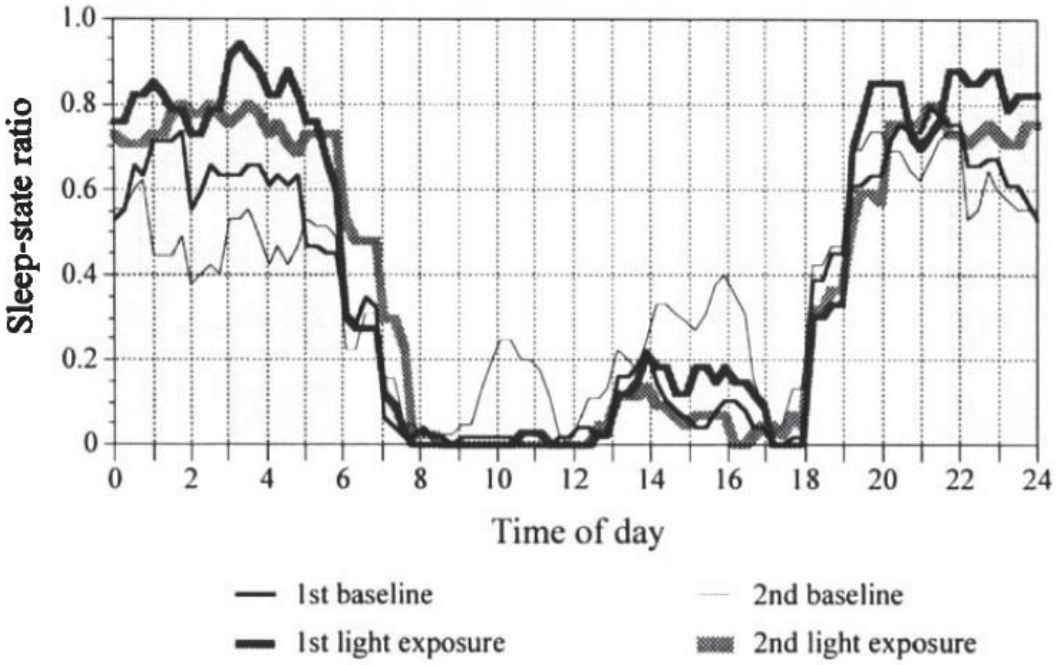


Figure 3.3. The sleep state ratio of a PWD trailing BLT.

The sleep state ratio was calculated from 15 minute epochs of sleep diary data being defined as "sleep" (scoring 1) versus "wake" (scoring 0; Okumoto, 1998, p. 196) .

In Summary, BLT is considered a promising option for treating the sleep problems of PWD because:

- the mechanism for light entrainment of the SCN is well understood;
- older people and PWD have been found to have reduced and/or inappropriate exposure to light;
- studies with younger adults and older adults without dementia offer conclusive evidence that BLT can be used safely and effectively to shift the timing of sleep, reinforce circadian rhythmicity, and improve mood and functioning; and
- trials of BLT for PWD seldom report *adverse* effects.

While the results of highly controlled trials might not offer conclusive support for BLT, it seems that a proportion of individuals do experience benefits. McCurry et al. (2011) found that adherence to light therapy over time could be an issue for long-term treatment, but those using the light box four or more times per week were significantly more likely to have positive effects regarding wake time at night ($p = 0.006$) and sleep efficiency at night ($p=0.005$).

3.2.3 A Person-Centred, Dyadic Approach to Treating Dementia-Related Sleep Problems

The consensus opinion of the National Institute of Health of America and others is that treatments for the sleep disorders of older people should be patient-led and target any underlying causes of sleep problems before considering medications (Bliwise, 2004; Dauvilliers, 2007; National Institute of Health Consensus Group, 1991; Small et al., 1997). However, Vitiello and Borson (2001) raise the important point that the increased use of sedating medications for PWD could be carer-driven rather than person-centred. The administration of sedating medications to PWD to make the job of caring for them less challenging is considered unethical. This could be a particular risk for PWD in advanced stages of dementia, in institutionalised settings, or among those who are unable to consent for themselves. One study in a nursing home found that more medications were given for agitation in the late afternoon and early hours of the morning than at other times of day. This appeared to reflect the temporal pattern of

sundowning behaviour as well as the shift patterns of nursing staff (Exum, Phelps, Nabers, & Osborne, 1993). However some medications were also administered throughout the day and the concern is that a "major ethical dilemma arises when sedative drugs are substituted for adequate day-time stimulation, activity and other nonpharmacological interventions with the potential to promote more normal sleep patterns." (Vitiello & Borson, 2001, p. 790).

Salami, Lyketsos, and Rao (2011) argue that non-pharmacological interventions should be considered "first line treatment strategies due to the relatively low risk of adverse effects to patients following administration of BLT or application of behavioural and multifaceted intervention" (p. 779). Vitiello and Borson (2001) highlight the need for more RCTs and longitudinal trials to assess the efficacy of non-pharmacological interventions for dementia-related sleep problems. While it is clear that more quantitative trials of this type are required and will help to develop standardised procedures and guidelines, another clear gap is the lack of community-based trials evaluating treatments and research methods for PWD on a case-by-case basis. In reviewing the literature, it became clear that the subjective opinions and sleep ratings of PWD and their carer are generally omitted. The subjective perceptions of sleep and health from the PWD/carer dyad are considered vital to dementia care and quality of life (Gibson, et al., 2014).

As informal carers are responsible for managing many of the activities and behaviours of PWD, they must be actively involved, not only in identifying the sleep problems of the PWD in their care, but also in assisting with therapies to improve their sleep. Caregivers need to actively promote non-pharmacological therapies by participating themselves and providing support to the PWD to maintain a routine which is conducive to good sleep. At the same time, clinicians and researchers need to be mindful that carers are also likely to have sleep disruptions which could impact on their capacity to take on additional tasks with regards to dementia-care. Therefore, especially when embarking on new therapies and routines, the dyad might require additional support from clinicians, researchers, or community workers to promote long-term compliance (McCurry, et al., 2003; Molano & Vaughn, 2014).

Previous studies in institutions, as well as the community, have found that the combination of BLT with physical or social activities as well as sleep hygiene education or behavioural modification, has led to the greatest improvements in sleep and other psychosocial factors (Alessi, et al., 1999; J. L. Martin, et

al., 2007; McCurry, et al., 2005; McCurry, et al., 2011). However, the workload for the carer needs to be considered, particularly for long-term compliance. One approach is to offer non-pharmacological treatment *options* rather than requiring combined treatments. It is also important that non-pharmacological interventions to improve the sleep of PWD do not inadvertently disrupt the sleep of their carer, who must generally assist and may participate in the interventions.

3.3 Rationale for Study 4

The 2010-2015 National Dementia Strategy (Alzheimers New Zealand Incorporated, 2010) identifies the need for research to improve the quality of dementia care practices. Psychosocial approaches to delaying institutionalisation have been highlighted as key for reducing the economic and social impact of dementia (Alzheimers New Zealand Incorporated, 2008). Effective strategies that improve the sleep and psychosocial factors for both those with dementia and their carers could also relieve some of the other dementia symptoms and carers' experience of burden, enabling people to continue living together longer in their own homes.

At the conclusion of each of the focus groups conducted for Study 3, participants were presented with the concept of non-pharmacological sleep interventions. Participants were given photographs of a potential light box and the procedure for using this, and therapeutic exercise and sleep hygiene were discussed. The light box was met with some confusion at first but once initial questions were answered (e.g., that it does not expose individuals to ultraviolet rays, that it works via a physiological mechanism rather than just psychological impact, and people with cataracts may find it uncomfortable to use), it was met with positivity. Across the groups, the main consensus was that there was no harm in trying BLT: "If it is a dull day and there is no sun is that [referring to the light box] going to make me feel as good as if we were out in the sun? And if it does, well I will [be] in for it!"(C5). Some carers expressed concern that their partner with dementia would not sit still long enough for the bright light exposure, however still felt it was worth attempting, especially during the winter when they were less likely to go outside. Other carers had concerns that introducing a LTD could create paranoia: "I think he would just think I am tricking him" (C8), and that compliance would depend on the mood of their partner on any given day.

One carer also raised concern that introducing the routine of using BLT on a daily basis might interfere with their normal routine and going outside.

The concept of routine exercise was also met with a positive response from both PWD and carers: "I think that's something we need to get into" (P10). It also seemed to be something many of the pairs had discussed doing or already did: "P3 is pretty regular at having an hour's walk and it sets him up for during the day" (C3). Some expressed wariness due to being less physically able, but the concept of having a light exercise routine that could be performed at home seemed appealing. As presented in Study 3, many of the pairs already had some sleep hygiene knowledge and practice, but they agreed that having a formal manual could be interesting and useful for them.

Study 4 was a pilot study assessing the feasibility of using non-pharmacological interventions to help improve the sleep of PWD and their informal carers living in the NZ community. It was designed to address some of the gaps highlighted throughout the literature review, particularly:

- the lack of community-based sleep data from PWD and their carers;
- the lack of community-based trials of non-pharmacological interventions;
- the lack of person-centred research methods for understanding and treating dementia-related sleep problems; and
- the lack of NZ-based research concerning sleep of older people and people affected by dementia.

This study was designed to answer the question: "Are non-pharmacological treatment options feasible and effective for community-dwelling PWD and their carers?" The aims of Study 4 were:

1. To gather objective and subjective data on the sleep of older PWD and their carers in NZ;
2. To identify factors that affect the sleep of older PWD and their carers;
3. To design and evaluate practical non-pharmacological interventions to improve the sleep and waking function of PWD being cared for in their own homes; and

4. To pilot the methodology required to conduct dementia-related sleep research in the NZ community.

Based on the literature review the following hypotheses were proposed:

1. Enhancing the rhythmicity of the SCN of PWD by timed light exposure and exercise will consolidate night time sleep and reduce daytime sleepiness of some PWD;

2. Sleep hygiene education will promote behaviour modification and improve symptoms of sleep disturbance for some PWD and their carer;

3. Improved sleep of PWD will have a positive effect on waking symptoms and quality of life;
4. Providing a sleep intervention package will also improve the sleep of some dementia carers; and
5. Providing a sleep intervention package will have a positive effect on carers' mood and burden as well as perceptions of dementia-related sleep disturbances.

Methods and results are presented in Chapters 4 and 5.

3.4 Ethical and Methodological Considerations for Dementia-Related Research

It is clear that PWD should be actively included in research (as outlined in section 2.3). However there are considerations and challenges which need to be addressed when designing and conducting research involving PWD (Bartlett & Martin, 2002; Goldsmith, 1996). The literature and guidelines that informed the design and conduct of Study 4 are reviewed here.

3.4.1 Risks and Benefits for Participants

Much dementia-related research aims to improve scientific knowledge, rather than to provide immediate benefit to the participants (Berghmans & Ter Meulen, 1995). Careful consideration by an independent ethics committee is required, particularly for intervention studies (National Ethics Advisory Committee, 2012b). To ethically be able to include PWD in intervention studies, Berghmams and Ter Meulen (1995) consider that there needs to be a real chance that the individual could benefit from taking

part, not just a theoretical possibility. Any research involving human participants needs to consider the benefits versus risks to the participants (National Ethics Advisory Committee, 2012a; The National Archives, 2005). Participants' motives for taking part typically include gaining direct benefit for themselves (e.g. therapeutic relief or cure), or potentially benefitting other individuals in the future. For PWD, direct benefit with regards to improving their personal degenerative process is considered unlikely. Furthermore, future benefits cannot typically be included as personal benefits, due to the nature of dementia occurring later in life with such a poor prognosis. However, as outlined above, secondary symptoms such as sleep quality, depression, or sundowning behaviours may be improved through taking part in intervention studies. Altruistic motives might also be included in estimating the risks and benefits. Such benefits might include joy or interest in taking part, contributing to knowledge and community, and benefit sharing and reciprocity. For older people and PWD, the diversion from routine, meeting new people, and feeling generally useful might also be classed as benefits. However these are person-specific so cannot be overestimated to justify the research (Patel, Renvoize, Higham, Crawford, & Suriya, 2005; Slaughter, Cole, Jennings, & Reimer, 2007). In fact, Iliffe (2008) argues that the typical Hawthorne effects observed in therapeutic research (Wickstrom & Bendix, 2000) may not be as great for PWD compared to other illnesses, because knowing that there is no cure for dementia could possibly affect participants' optimism about a positive outcome. Therefore participating in dementia-related research carries the potential risk of highlighting and exacerbating participants' impairments and rate of decline.

While it is important that the benefits outweigh the risks of taking part (or at least be proportional), how much risk is acceptable is primarily dependent on the content of the research and is based on the decision of the potential participants (National Ethics Advisory Committee, 2012a; Slaughter, et al., 2007). The risks of taking part in intervention studies can be great, for example, if trialling medicines or care plans that are in conflict to established practice. This is the reason that informed consent is so important. However, for PWD, the process of informed consent is in itself a challenge (see below). Therefore it is the responsibility of the researcher and ethics committees to assess whether there is more than minimal risk to the participants (e.g., no more risk than could be expected from discomforts

experienced through everyday life), and if there is, to conduct the research with greater care and with proxy supervision (Post, 2003).

3.4.2 Recruitment

There are many hurdles to overcome to recruit research participants, but there are particular issues when including older people, PWD, and their caregivers. These include *gatekeeping*, methods of advertising, and avoiding coercion. While the opportunity to take part in research may be empowering to PWD, their rights and privacy need to be safeguarded. To access PWD and offer a research opportunity, the study not only requires approval from a local ethics committee. Permission and assistance is often also required from those responsible for the care of the PWD, such as social workers, residential home management and family carers. These people and institutions can have valuable inputs in research planning to ensure it is appropriate and accessible to PWD. However they also play an important, protective role for safeguarding those with dementia and in some situations this can hinder recruitment if they are over protective, thereby gatekeeping access to the potential research participants (Bartlett & Martin, 2002; Clarke & Keady, 2002; Hellström, Nolan, Nordenfelt, & Lundh, 2007; McKeown, et al., 2010).

Fisk and Wigley (2000) found this to be the case when recruiting participants from institutions for a survey concerning services received. They found that around a quarter of care home management stated that their residents would not be interested and therefore refused to offer the research to their residents at all. Although gatekeepers can be viewed as a barrier to dementia-related research, care providers are also very useful in that they have the knowledge and relationship with potential participants which, if they endorse the project, can make the situation less anxiety provoking. They can also be useful when it comes to disseminating results because they remain in contact with the people beyond research (Pratt, 2002).

Community-based dementia research often requires family carers to participate and support the study. These are also a hard-to-reach group due to the nature of their responsibilities and time commitments, age, and obligation to protect the privacy and sense of self of their family member with dementia (Murphy et al., 2007). There is also the risk of the whole experience being overwhelming for an already burdened carer (Whitebird et al., 2011).

Previous studies have found that traditional methods such as paid advertising and media are not as successful for recruiting PWD or their family carers as they are for other groups. A more person-centred approach is required, using face-to-face contact to present the research and ask the potential participant's permission to take part (Dewing, 2002; Downs, 1997; Innes, 2009; Patel, et al., 2005). Outreach programmes incorporating healthcare professionals, community workers, and those with dementia-related experience have higher success rates (Dyall, et al., 2013; McKeown, et al., 2010; Whitebird, et al., 2011).

3.4.3 Informed Consent and Assent

The risk of coercion can be higher in dementia-related research if potential participants are less able to receive or understand the information required to make an informed choice to take part (Berghmans & Ter Meulen, 1995). To avoid this, researchers need to carefully judge potential participants' capacity for consent and whether or not to use proxies.

The National Ethics Advisory Committee (NEAC) of New Zealand emphasises the importance of giving vulnerable participants the opportunity to take part in research which is of relevance to them, and to respect that the label *vulnerable* "may not apply to all individuals in such groups, and even where it does apply, it may do so only intermittently" (2012a, p. 14). Likewise, due to the heterogeneous nature of dementia with regards to type and severity, individual vulnerability as well as cognitive ability, lucidity, and therefore capacity to understand, will vary between and within participants (Qureshi & Johri, 2008). This is important as PWD should be treated respectfully from a person-centred approach in order to protect their sense of self (Downs, 1997; Kitwood & Bredin, 1992; Patel, et al., 2005). Fellows (1998) reminds us that "values and decision-making skills that served these patients for their entire lives may persist at some level, even if no longer expressed coherently" (p. 923). Therefore researchers working with PWD need to be stringent with regards to communication and the judgement of competence and capacity to consent, as well as assent and dissent of their participants whilst taking part.

Informed consent is defined as a decision informed by an understanding of the relevant information, and that the decision is entirely voluntary (Massey University, 2010; National Ethics Advisory

Committee, 2012a). The decision needs to be based on communication rather than simply transferring information (Manson & O'Neill, 2007). Therefore the researcher must make a judgement on whether the participant has the competence to provide informed consent. This can be complicated because competence, and therefore the capacity to give consent, is a continuum so needs to be judged on a case-by-case basis. Slaughter et al. (2007) surmise that "competent decision making involves more than being cognitively capable. It involves the ability to understand and appreciate the context and implications of the decision, and also involves the capacity to translate the decision into action" (p. 30). Some of the specific factors that affect people with cognitive impairment and dementia being able to provide informed consent and participate in research include: a reduced vocabulary, understanding of complex or abstract words or ideas, or difficulty following long sentences; reduced short-term memory and attention span; reduced ability to answer time-related questions; as well as possibly having an emotional disposition, and a reluctance to say if they do not understand (Bray, 1998; Hubbard, Downs, & Tester, 2003). When a participant has diminished competence, they still retain the right to make informed choices and give informed consent, to the extent appropriate to their level of competence (Health and Disability Commission's Code of Rights right 7.3, Health and Disability Commissioner, 1996). Therefore, for research with more than minimal risk, careful judgements need to be made to avoid exploitation of those who might not have understood the nature of the research and the potential risks (Qureshi & Johri, 2008). The NEAC (2012a) recommends that information provided to PWD is not overly detailed or complex as this can be confusing or frustrating, which develops a barrier rather than assisting free and informed consent.

In research with increased risks it may be necessary to formally assess the participants' level of competence. This can be done through cognitive testing prior to the consent process, or giving a short quiz on what the research involves and their risks and rights (Black, Kass, Fogarty, & Rabins, 2007; Buckles et al., 2003). While repeating the information might be useful for potential participants, researchers need to appreciate the effects that these methods may have on the participant's self-esteem and dignity (Buckles, et al., 2003; T. Moore & Hollett, 2003). The consent process might be simpler if PWD made advanced directives concerning their interests in taking part in research to assist proxy decision making (Stocking et

al., 2006). However, legal and care directives take priority over what type of hypothetical research PWD might be interested in taking part in in the future (T. Moore & Hollett, 2003; Slaughter, et al., 2007). Furthermore, due to possible changes in personality and decision making with ageing and dementia, it might be in the PWD's best interest for proxies to focus on the present circumstances rather than past preferences in order to avoid decisions being made which are contrary to a vulnerable adult's current wishes (Slaughter, et al., 2007).

Local ethical guidelines state that when the level of competence is unclear "it may be appropriate for an investigator to seek both the informed consent of that person and the informed agreement of another person who is interested in, or has responsibilities for, that person's welfare" (National Ethics Advisory Committee, 2012a, p. 15). A dual consent process keeps the research person-centred. The PWD retains their dignity and right to be involved with the consent process whilst their carer supervises the situation and acts as an advocate for the PWD, thereby adding a layer of protection for vulnerable participants (Dewing, 2002). However there are no set international guidelines specific to including PWD in research, and so protocols for consent vary between studies (Black, et al., 2007).

In circumstances when it is clear that the participant does not have the capacity to consent, ideally a legal representative for the participant should be invited to decide on their behalf. However many PWD, carers and ethical review boards believe that a legally-appointed decision-maker is unnecessary for low risk research (Bravo, Paquet, & Dubois, 2003). Furthermore, many PWD living at home have not allocated someone to be a lasting power of attorney. In such cases lack of capacity to consent should not be a reason to exclude PWD from socially responsible research (Medical Research Council, 1991). The research should continue so long as it is approved by a local research ethics committee, is in concordance with the views of those responsible for the PWD's welfare, is considered relevant to the participant, and they assent to participating (see below; Medical Research Council, 1991; National Ethics Advisory Committee, 2012a; Slaughter, et al., 2007). Families make a huge range of care-related and day-to-day decisions for their family member with dementia, so we should also trust them to give proxy consent. However researchers need to respect that being a proxy could be burdensome if the research has more than minimal risk to the participant.

In the planning stages of research, contemplating the barriers and guidelines regarding consent make conducting dementia-related research a daunting prospect. However, Berghmans and Ter Meulen (Berghmans & Ter Meulen, 1995) emphasise that:

> If one holds the view (which we do not) that the obligation to obtain informed consent outweighs all other obligations, no research with persons suffering from dementia is ethically justified… Are we then forced to halt our efforts to improve our knowledge of this devastating disease, which imposes a great burden not only on those who suffer from it, but on caregivers and society as a whole as well? (p. 648).

This statement reminds us that the question is not *if* we should be conducting research including people with dementia; it is a matter of carefully considering *how*.

In any study involving human participants, the NEAC recommends an ongoing dialogue between researcher and participant throughout consent and research process, in order to give the participants the opportunity to raise questions or concerns (National Ethics Advisory Committee, 2012a). For PWD this oral dialogue is essential and it should be conducted in a way that the participant does not feel coerced or manipulated. This is important as there might be a tendency for PWD to comply with the perceived demands of an authority figure (Bray, 1998). A verbal discussion of the project may be more appropriate than providing lots of written material as this can overcomplicate the consent processes and be overwhelming (Black, et al., 2007; McKeown, et al., 2010). For some, recording verbal consent may also be more suitable than gaining a written signature, however this needs to be justified to an ethics committee as it is important that a permanent record of consent is obtained (National Ethics Advisory Committee, 2012a).

Due to the nature of cognitive impairments and memory difficulties that some PWD may be experiencing, continued consent and assent need to be carefully monitored throughout the research process rather than just at the outset. This is especially the case in longitudinal research when dementia is likely to have progressed (Qureshi & Johri, 2008). Regardless of consent being formally obtained from the individual or a proxy, the participant still needs to assent to taking part (T. Moore & Hollett, 2003;

Slaughter, et al., 2007). Assent is verbally or non-verbally showing agreement to take part and can be judged through developing a good rapport with the participant prior to data collection. Signs of dissent include a participant showing verbally or non-verbally that they are uncomfortable or distressed, by way of facial grimacing, agitation, perseverance on troubling subjects, or behavioural outbursts (Slaughter, et al., 2007). If a participant clearly dissents then they should be removed from the study. Moore and Hollett (2003) recommend that when it appears that the participant is become uncomfortable, the researcher should offer the participant a break, support and listen to them, and give the choice to stop the research process, rather than simply ceasing the data collection based on a personal judgement of a behavioural outburst. Offering the choice to continue respects the rights of the participant who might be happy to take part despite feeling emotionally distressed (Cohen-Mansfield, 2003; as cited in Slaughter, 2007, p. 33).

Engaging potential participants and retaining them in research have been highlighted as key issues for conducting dementia-related research. In the most recent study of McCurry et al. (2011) 43% of the dyads who were initially interested in taking part (n=535) did not meet their eligibility criteria and 32% declined to participate, leaving them with a sample of 132. A further 13% of the sample also dropped out before post-testing. This is to be expected due to the nature of dementia and older age. The severity of symptoms could rapidly decline, making the PWD or carer more likely to dissent to research activities, and increased severity of dementia could also lead to institutionalisation or death. Alternatively, due to the complexity and routine nature of the caregiving situation, enrolment and compliance in research are less likely to be at the forefront of a carer's agenda. For these reasons, it is necessary that research assistants have skills in dementia care, to avoid unnecessary loss of participants and to manage withdrawals appropriately.

3.4.4 Collecting Dementia-Related Data

"Data collection requires creativity and a positive approach to managing the challenges of researching with people with dementia" (Clarke & Keady, 2002, p. 40). Researchers experienced in dementia-related studies as well as the NEAC have made methodological recommendations for research design involving people with cognitive impairment and dementia (Black, et al., 2007; Booth & Booth, 1996; Clarke & Keady, 2002; Cotrell & Schulz, 1993; Dewing, 2007; Downs, 1997; Goldsmith, 1996;

McKeown, et al., 2010; National Ethics Advisory Committee, 2012b; L. Nygård, 2006; Pratt, 2002; D. Reid, Ryan, & Enderby, 2001; Sherratt, et al., 2007; Whitebird, et al., 2011). Common themes in this literature include maintaining a person-centred approach to the research, being open and aware of the opportunities and the effect of taking part in the research on the PWD as well as those around them, appropriate and effective communication, and allowing for additional time when conducting research. Figure 3.4 provides a summary of the common recommendations within these four themes. When including these suggestions, the researcher also needs to consider the additional costs to the research with regards to extra time requirements for recruitment and data collection, additional travel expenses, and individual care requirements. It may be that smaller sample sizes are required, and this is deemed more appropriate (Cotrell & Schulz, 1993). However, hopefully following these recommendations makes it likely that participants will be more compliant with the research protocol and less likely to withdraw.

3.4.5 Considerations for Study 4

The design of Study 4 was informed by the literature and recommendations outlined above and in *Figure 3.4*. This was aided by consultation with local community workers as well as my personal experiences of working with PWD. An important challenge is that whilst incorporating special methodological approaches, research quality needs to be maintained. The data being collected should be reliable and valid in order to do justice to those contributing (T. Moore & Hollett, 2003). This balance is considered particularly challenging in community-based psychosocial research, where the protocol may be less controlled than in research taking place in an institutionalised setting or trialling medications.

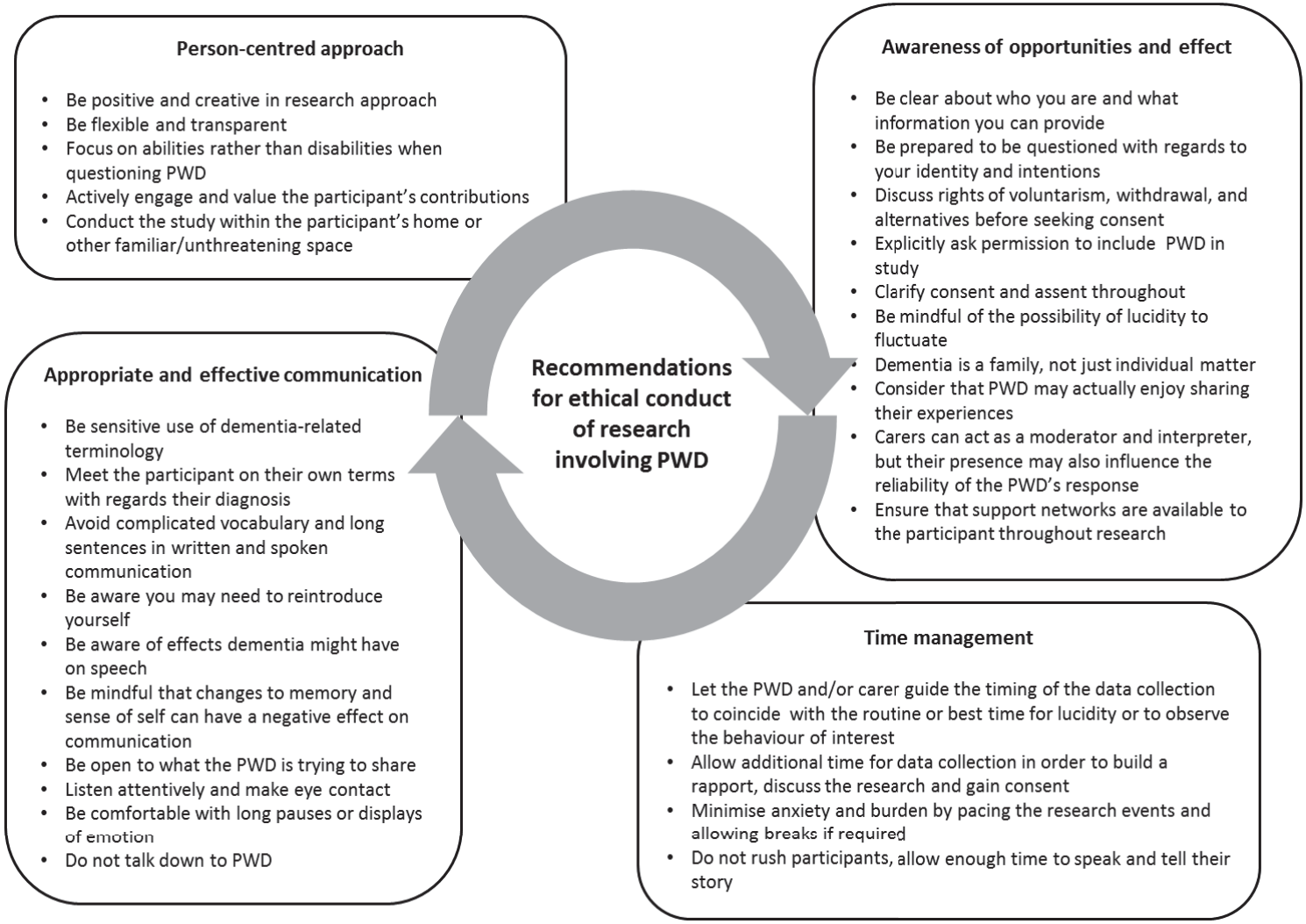


Figure 3.4. Considerations for the appropriate and ethical conduct of dementia-related research, based on the reviewed literature

4 STUDY 4 METHODS

4.1 Design

Study 4 was a within-subjects study that trialled non-pharmacological interventions targeted at improving the sleep of PWD and subsequently that of their family carer. Sleep was measured using both objective and subjective measures. Tools and materials were selected because they had either been validated in prior research with the older people, or were deemed appropriate and practical to use with this population. The intervention period lasted five weeks and involved introducing timed BLT and physical exercise, as well as sleep hygiene education.

Based on the literature review and the potential challenges to dementia-related research, this was designed as a pilot study in order to explore the most appropriate ways to conduct the research. Each participant was expected to have specific needs and it was deemed important to evaluate these needs and the research situation in order to conduct the project in an ethically responsible, yet scientifically rigorous manner.

4.2 Ethics

Approval for Study 4 was obtained from the Central Health and Disability Ethics Committee (CHDEC, application number CEN/11/01). A copy of the letter of ethical approval can be found in Appendix 3. Participating in the study was voluntary and based on informed consent.

Non-invasive methods were used for monitoring sleep (actigraphy, see below). Side effects to BLT are minimal and include headaches and nausea, often caused by sitting too close or staring directly into the light. As noted in section 3.2.2, chances of side effects are increased for those with pre-existing eye conditions or taking medications which could cause photosensitivity. Participants were screened for these factors at recruitment.

Measures to support and protect the PWD during the research process were considered in the study design (as noted in section 3.4). These measures included:

- Face-to-face contact with participants;
- allowing additional time for describing the study and answering queries;
- the option of proxy consent;
- recording verbal assent at the beginning and throughout the study;
- using questionnaires and interventions which could be administered by the carer or researcher if/when necessary;
- free phone contact and an email address were made available for participants to contact the researcher directly; and
- Participants were telephoned weekly to offer study support.

With their permission, participants' General Practitioners (GPs) were also informed that they were taking part in the study. This gave the GP the opportunity to support their patient during the study process. The GPs also received a summary of results at the conclusion of the study (see below for more detail).

4.3 Recruitment of Participants

Power calculations were undertaken to estimate the sample size needed to detect clinically relevant changes to the sleep of PWD. They were based on a previous intervention study involving 15 nursing home residents with AD who were exposed to 10 weeks of one hour of morning BLT and evening melatonin administration (Dowling, et al., 2008). Using the same actigraphy devices as in the present study, the average baseline daytime sleep for this group was 315 minutes (SD =129 minutes). The average reduction in daytime sleep following treatment was -116 minutes (there was a non-significant average increase in night time sleep of 30 minutes). On this basis, to detect a change of 75 minutes with 80% power (two-tailed $p<0.05$), the present study would require 26 participants. The average baseline daytime/night time sleep ratio was 0.70 (SD = 0.27). After treatment, the average reduction in the daytime/night time sleep ratio was 17%. On this basis, with 26 participants the present study would have

80% power (two-tailed $p<0.05$) to detect a reduction of 15.4%. Based on these considerations, the study was designed to include 30 PWD/carer dyads in order to conduct reliable statistical analyses. As previously noted, recruitment and retention rates in dementia-related research can be low. Therefore it was proposed that up to 45 pairs would be sought.

Due to poor response rates, pairs were recruited into the study in four phases via different methods, inclusion/exclusion criteria, and organisations of people assisting with recruitment. Participants could volunteer their interest to the researcher by phone, email, via a healthcare professional, or directly at a public presentation. The four phases of recruitment are outlined in detail below to offer insight into this complex process.

4.3.1 Recruitment Phase One

Initial recruitment took place through two locations, Alzheimers NZ Incorporated and Alzheimers Wellington. Alzheimers NZ Incorporated is a not-for-profit organisation with 21 Alzheimers member organisations located throughout the country that provide support, information, education programmes and services appropriate to their local community. In 2011-2012, Alzheimers NZ had approximately 4000 people subscribed to receive newsletters. Alzheimers Wellington was the local member organisation where the majority of active recruitment was conducted (as per Study 3). In 2011-2012, Alzheimers Wellington had approximately 390 members (however these members could include health care workers, students and alumni, as well as families affected by dementia).

The inclusion/exclusion criteria for taking part in the study during phase one are outlined in Table 4.1. These were the most rigid and ideal criteria. In order to recruit a homogeneous group, it was desirable to have participants over a specified age, with definite sleep disturbances, one with a definite diagnosis of dementia and the other a spousal carer. In this phase, any contraindications to using the components of the intervention meant complete study exclusion. A summary of the recruitment strategies used together with the outcomes are given in Table 4.2. Some examples of materials used during recruitment can be found in Appendix 4.

Table 4.1

Inclusion / exclusion Criteria Used in Recruitment Phase One

Inclusion Criteria	
People with Dementia	Carers
Diagnosis of dementia Live with their spouse/partner who cares for them Aged ≥65 years Have disrupted sleep 3 or more times a week	Live with and provide care for a spouse/partner with dementia Aged ≥65 years Identify sleep as a problem for their partner with dementia
Exclusion Criteria	
People with Dementia	Carers
Have a movement disorder (e.g.,Parkinson's disease) which may interfere with sleep measurement by actigraphy Vision problems, or medical contradictions to bright light exposure Inability to walk across room (for exercise intervention)	Have a movement disorder (e.g.,Parkinson's disease) which may interfere with sleep measurement by actigraphy

Due to the decreasing response rates through Alzheimers New Zealand and Alzheimers Wellington, the study was advertised more widely through other community organisations. Advertisements, informative articles, and oral presentations were delivered to senior groups. A summary of these additional recruitment strategies together with the outcomes are given in Table 4.3.

Table 4.2

Recruitment Strategies Used in Phase One, Alzheimers NZ & Alzheimers Wellington

Date	Strategy	Target	Approximate targeted	Responded	Recruited
May 2011	Mail out	Past focus group participants, Alzheimers Wellington	12	6	1
June-August 2011	Mail out	Members of Alzheimers Wellington (fitting inclusion criteria)	120	12	5
September 2011	Article and advertisement	Alzheimers Wellington newsletter	400	1	0
September 2011	Web article and call for participants	Alzheimers NZ national website	Open access	0	0
October-November 2011	Presentation on sleep in ageing and dementia plus promotion of study	Three groups of Alzheimers Wellington members	53	0	0
December 2011	Article describing focus group findings and call for further participants	Alzheimers Wellington newsletter	390	0	0
December 2011	Article, detailing importance of study with call for further participants	Alzheimers NZ Newsletter	4,000	0	0
Totals			>4,975	19	6

Table 4.3

Recruitment Strategies Used in Phase One, Other Senior Services

Date	Strategy	Target	Approximate targeted	Responded	Recruited
July 2011	Posters	Freemason Lodges across Wellington region	10 Lodges	0	0
August 2011	Flyer	Information packs at Hutt Carers Expo	200	0	0
September 2011	Article and call for participants	Carers NZ magazine and E-zine (Carers for all types of disability nationally)	34,000	2	0
August - December 2011	Presentation*	Residents of four retirement villages	170	0	0
August 2011 - March 2012	Presentation*	Nine senior social groups across Wellington region	280	0	0
August -December 2011	Presentation*	Two dementia-related support groups across Wellington region	50	1	1
October 2011	Article and call for participants	Age Concern Wellington newsletter	Unknown	0	0
December 2011	Article and call for participants	Senior Services newsletter, Ministry of Social Development	Unknown		
March 2012	Presentation*	Café Scientifique	40	0	0
June 2012	Radio interview	Grey Power radio	Wellington/Kapiti	0	0
April 2012	Flyers in conference packs	Brain Day (Neurological Foundation), Scientific and clinical audience	Open access	0	0
May 2012	Flyers in conference packs and posters	Alzheimers NZ conference, Scientific and clinical audience, plus PWD and carers	Open access	0	0
	Totals		>34,700	2	1

* Oral presentation with slides about sleep in ageing and dementia plus promotion of study (see slides in Appendix 4).

4.3.2 Recruitment Phase Two

Due to poor response rates from phase one, a new recruitment strategy was designed. Feedback from the public, those working with PWD, and from those who decided not to take part in the study, informed changes to the recruitment materials and criteria. The main amendments which were approved by the CHDEC included:

- Changes to the tone of the documents sent to potential participants (to be more considerate and sound less clinical).

- The PWD were no longer required to have "disrupted sleep three or more times a week" as defined in phase one. They were simply required to be "interested in improving their sleep". This amendment was made after considering the results of Studies 1 and 3 as well as through conversations with potential participants which revealed that sleeping problems were often normalised or simply not identified. Therefore having disturbed sleep as an inclusion criterion may have been hindering volunteering.

- Vision problems or contraindications to bright light exposure were no longer treated as exclusion criteria from the study as a whole, but simply as exclusion from using the light box. Bright light therapy could be delivered by natural light in such cases.

- The inability to walk across a room was no longer considered as an exclusion criterion from the study as a whole, but simply from the walking aspect of the exercise intervention. A programme of exercises which could be done from a chair using a DVD would be recommended for such cases.

The second phase of recruitment involved engaging selected GPs, health practitioners and geriatricians for their assistance. Those agreeing to assist with study recruitment signed a Locality Assessment Form (from the CHDEC) for their site. Health providers assisted with recruitment using a choice of three methods dependent on what they deemed appropriate for their site:

1. Identifying potential participants via their client database and posting them a letter of support together with the study advert and/or letter;

2. recognising that a client/patient might benefit from the sleep study and informing them of the study or handing them a flyer with the study details at a scheduled appointment; and/or

3. making posters and flyers for the study available in waiting room areas and offices.

For those actively recruiting participants (i.e. using method 1), an offer was made for staff to be compensated for their time by either reimbursement of administration costs for identifying and mailing out the information to patients (1-2 hours pay), or a gift hamper of equivalent cost to share with the practice.

A Continuing Medical Education (CME) endorsed lecture was conducted in February 2012. This allowed GPs and other healthcare professionals to meet the researcher, learn more about the changes to sleep with dementia, and also gain greater insight into the intentions of the research. This course offered compensation in the form of CME credits for health providers who were (or were considering becoming) involved with the sleep study. It also provided a time for members of organisations to introduce themselves and sign up. A summary of the locations that assisted with recruitment as well as the type of strategy used (coded 1-3 as above) and outcomes are given in Table 4.4.

4.3.3 Recruitment Phase Three

In this final phase, the inclusion criteria were changed to include family carers of any age or relationship to the PWD. This opened up the demographic to include alternative dementia service providers, as well as more members of Alzheimers Wellington.

Times were allocated for groups of participants to begin the research protocol. These were scheduled throughout April and May 2012 via Alzheimers Wellington. It was anticipated that providing a set date might encourage people to enrol more quickly rather than putting it off or forgetting to call. It was also considered that a group atmosphere might make taking part less intimidating. This strategy was also more cost effective than multiple home visits.

Table 4.4

Recruitment Strategies used in Phase Two, Healthcare Professionals

Recruitment site	Strategy*	Approximate targeted	Recruited
Psychogeriatric services, Kenepuru Hospital	2&3	Unknown	0
Island Bay Medical Centre	1&3	4	0
Karori Medical Centre (post CME event)	2	1	1
Older Adult, Rehabilitation and Allied Health	2	unknown	0
Capital and Coast Care Co-ordination Centre	2	unknown	2
Elder Family Matters	2	unknown	1
Upper Hutt Health centre	2 & 3	unknown	0
Elder service provider network	2	unknown	0
District Nurse teams	2	unknown	0
Total			4

* 1 = posts information, 2 = informing given during consultation, 3 = posters and flyers

Once the new inclusion criteria were approved by the CHDEC, invitations to one of three groups (Wellington, Hutt Valley or Kapiti) were sent out to members of Alzheimers Wellington. Those who had already taken part or actively opted out of the study were not included in the mail out. Morning tea, taxi or petrol vouchers were offered as compensation to those willing to take part. None of the groups went ahead due to minimal response. However the mail-out successfully engaged two new pairs. Another two pairs were enrolled via study promotion by Alzheimers Wellington support workers at this time.

After approximately one year of multiple recruitment efforts, 15 dyads were recruited (six via phase one, one via phase two, four via phase three, and four via phase four). During the recruitment process, 17 other couples contacted the researcher but did not take part in the research. Many of the carers were keen to try an alternative for treating their family member's sleep problems but could not take part for various reasons. Typically because they felt the study protocol would be too much for them to take on, or their family member did not know or accept their diagnosis, or their dementia was progressing too quickly for them to feel they could embark on a six-week trial (Table 4.5). All but two of these pairs wanted to be kept on a mailing list of updates and results related to the study.

Table 4.5

Reasons for Potential Participants not Taking Part in the Intervention Study

PWD-related reason	n	Carer-related reason	n
PWD does not know or accept their diagnosis and carer concerned about unnecessarily distressing them	3	Felt the study protocol would be too much for them	6
Cognitive functioning deteriorating very quickly	3	Carer exhausted or too busy	2
Neither participant had dementia	3	Study was more in depth than anticipated	1
PWD moved into institutionalised care before trial	1	Already have a lot of people conducting home visits with questionnaires and giving dementia-related advice, reluctant to add more by choice	1
Sleep not considered problematic enough to warrant taking part	2	Carer concerned that their partner would agree to the study but then not comply with the protocol properly, causing friction in their relationship	1
PWD denies having any sleep problems and so would not consent	1	Carer worried they would be making answers up for the questionnaire concerning their family member as they had severe cognitive impairment	1
Severe Parkinsonian tremor	1	Carer became unwell before trial	1
Physically handicapped	1	Felt too old to take part	1
PWD takes sleeping medication nightly and not prepared to try anything else	1		
PWD would not consent, no apparent reason	1		

4.4 Materials

An overview of the materials used in Study 4 can be found in Table 4.6, with more detail given below.

4.4.1 Questionnaires for People with Dementia

The full questionnaire for the PWD can be found in Appendix 5. This was limited in length and content due to the ethical and methodological considerations outlined in section 3.4, as well as a lack of standardised questionnaires for PWD to complete themselves. Questionnaires concerning the PWD were also included in the carer questionnaire.

Table 4.6

Overview of Materials Used in Study 4

Domain	Material	Purpose	Completed by: PWD	Carer
Sleep	Pittsburgh Sleep Quality Index (PSQI; Buysse, et al., 1989)	Measure of sleep disturbance	✓	✓
	Sleep Disorders Inventory (SDI; Tractenberg, et al., 2003)	Measure of dementia-related sleep disturbance		✓
	The Actiwatch-2™ (Mini mitter, Philips, Respironics)	Actigraphic recording of sleep timing	✓	✓
	Sleep diaries	Subjective records of sleep timing and quality	✓	✓
Cognition	Mini Mental Health Exam (MMSE; Folstein, et al., 1975)	Measure of cognitive status	✓	✓
Dementia symptoms	The Revised Memory and Behaviour Problems Checklist (RMBPC; Teri et al., 1992)	Measure of the frequency and severity of dementia-related symptoms		✓
Impact of dementia	The Quality of Life in Alzheimer's Disease: Patient and Caregiver Report (QOL-AD; Logsdon, Gibbons, McCurry, & Teri, 1999)	Measure of quality of life of community- dwelling PWD	✓	✓
	The Carers of Older People in Europe index (COPE; McKee et al., 2003)	Measure of carer burden		✓
Health	Self-Administered Comorbidity Questionnaire (SCQ; Sangha, Stucki, Liang, Fossel, & Katz, 2003)	Record of comorbid health conditions	✓	✓
	Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983)	Screening tool for depression and anxiety		✓
Intervention	The Day-Light (Uplift Technologies, 2007)	Bright light therapy	✓	
	Sit and Be Fit senior's exercise DVD (McLennan, 1999), Push Play Stretches pamphlet (Sport and Recreation New Zealand)	Therapeutic exercise programme	✓	✓
	Sleep Support: A Sleep Handbook for Older People Living with Dementia	Sleep hygiene education	✓	✓

Demographic information.

Demographic details included the participant's age and sex, what type of dementia they had been diagnosed with, and approximately when they were diagnosed.

Sleep.

The Pittsburgh Sleep Quality Index (PSQI; Buysse, et al., 1989) was used to gather subjective sleep data. As noted in section 1.3.3, the PSQI is a standardised measure of subjective sleep quantity and quality over the past month. It contains 18 self-report items with additional ratings of sleep disturbance to be reported by a partner if applicable. It gives an indication of self-satisfaction with sleep and is used to identify those with a sleep disturbance, as well as highlighting the types of sleep disturbance.

The PSQI includes questions concerning usual sleep timing and duration (time gone to bed, time it takes to get to sleep at night, time up in the morning, and hours of estimated sleep per night). Participants also rank the frequency that they have experienced any of 10 symptoms of sleep disturbance (*not during the last month, less than once a week, once or twice a week,* or *three or more times a week*). The symptoms include: "Not being able to get to sleep within 30 minutes, waking up in the middle of the night or early morning, having to get up to use the bathroom, not being able to breath comfortably, coughing or snoring loudly, feeling too hot, feeling too cold, having bad dreams, having pain", and "any other reasons". In the initial stages of data collection these questions were modified to include a fifth frequency option, *don't know*, as some PWD reported they simply couldn't remember. If their carer also did not know the answer, it was deemed more reliable to include this additional option rather than risk missing or misrepresenting data. Participants were then required to indicate how often they have taken sleeping medication, how often they have had trouble staying awake in the daytime, and how much of a problem it had been to "keep up enough enthusiasm to get things done". Finally participants rated their overall sleep quality (options: *Very good, fairly good, fairly bad,* or *very bad*).

An additional section of the PSQI required carers to help answer some questions concerning disturbances occurring during sleep that the PWD may have been less aware of. Together they rated how often (as above) the following symptoms had occurred in the previous month: "Loud snoring, long pauses

between breaths when asleep, legs twitching or jerking when asleep, episodes of disorientation or confusion while asleep", or "any other restlessness".

The PSQI is scored on seven components: *subjective sleep quality* (how the participant rates their sleep overall), *sleep latency* (based on their reports of sleep timing and ability to fall asleep within 30 minutes), *sleep duration* (participant's estimate of total sleep time), *sleep efficiency* (calculated using difference between the reported time spent in bed and sleep duration), *sleep disturbances* (frequency of symptoms over past month), *use of sleep medication* (over past month), and *daytime dysfunction* (trouble staying awake and enthusiasm in the daytime). A global PSQI score is then calculated (0-21 points), where 0 indicates no sleep difficulty and 21indicates severe sleep difficulty. As previously noted, a threshold of 5 is typically used to distinguish "good" from "poor" sleepers. This threshold is considered reliable with a diagnostic sensitivity of 89.6% and specificity of 86.5% (Kappa = 0.75, $p < 0.001$; Backhaus, et al., 2002; Buysse, et al., 1989).

The PSQI has been widely used in clinical and research settings (Barclay, Eley, Buysse, Rijsdijk, & Gregory, 2010; Hancock & Larner, 2009). It has also been successfully used with older people (Buysse, et al., 1991; King, et al., 1997) including PWD (Boddy, et al., 2007; B. Carpenter, et al., 1996; McCurry, et al., 1999; Tsai, et al., 2008), and caregivers (Castro, et al., 2009; McCurry, et al., 1998; R. Moore, et al., 2011; Sacre, 2010; Von Känel et al., 2012). The PSQI has a reliable test-retest consistency over several weeks (correlation coefficient = 0.85 – 0.87, $p < 0.001$; Backhaus, et al., 2002; Buysse, et al., 1989) and a high internal consistency (Cronbach's alpha coefficient = 0.80 – 0.83; Buysse, et al., 1989; J. Carpenter & Andrykowski, 1998). This makes it an appealing tool to measure changes related to treatment.

In addition to the PSQI, participants were asked if they napped during the day (options: *Yes, no,* or *sometimes*), and if they did, for approximately how long. Participants were also asked whether or not they had been diagnosed with a sleep disorder, and if so, what type and when they were diagnosed.

Health.

Questions concerning medications and comorbid conditions were included in order to help describe the sample and potentially control for some of the variation between participants. Medications were recorded as a list with dosages. The Self-Administered Comorbidity Questionnaire (SCQ; Sangha, et al., 2003) was used for participants to self-report whether they had any of the 13 common problems listed (heart disease, high blood pressure, lung disease, diabetes, ulcer or stomach disease, kidney disease, liver disease, anaemia or other types of blood disease, cancer, depression, degenerative arthritis, back pain, or rheumatoid arthritis), plus any additional problems which they could add. Participants also noted whether or not the medical conditions were currently being treated (giving an indication of disease severity), and whether or not they considered them to limit their functioning (giving an indication of burden of the condition). The SCQ was scored by each medical condition receiving 0-3 points based on the presence of the condition, whether receiving treatment, and whether the condition limits the participant's functioning.

The SCQ is quick to administer, has a high test-retest reliability (intraclass correlation coefficient = 0.94, 95% CI = 0.72-0.99), and has reasonable agreement with the medical record-based Charlson Index (78% - 90%; Charlson, Pompei, Ales, & MacKenzie, 1987; Sangha, et al., 2003). The SCQ is therefore a time and cost effective option for measuring coexisting health conditions without consulting medical records. Furthermore it has successfully been used in previous studies with older people and PWD (e.g., McCurry, et al., 2011; Van Nispen, Hoeijmakers, De Boer, Ringens, & Van Rens, 2008).

Cognitive status.

The Mini Mental Status Exam (MMSE, Folstein, et al., 1975) was used to evaluate cognitive functioning. This is a brief standardised tool containing 11 questions across five categories: *Orientation* (e.g., "Where are we?"), *registration* (e.g., remember and repeat the names of three objects), *attention and calculation* (e.g., subtract from 100 in sets of seven), *recall* (recall the three objects from earlier in the test), and *language* (e.g., repeat the phrase, "no ifs, ands or buts"). Scores for each question are combined to provide a global score of cognitive functioning (on a scale of 0-30). Scores were interpreted with regards to a set of thresholds: 23-30 representing "normal cognitive functioning", 19-22 representing "mild cognitive impairment", 10-18 representing "moderate cognitive impairment", and ≤9 representing "severe

cognitive impairment". While the MMSE is not a reliable tool for identifying and diagnosing specific disorders, it is sensitive for detecting cognitive impairment and dementia (87% sensitivity, 82% specificity; Anthony, LeResche, & Niaz, 1982).

Although the MMSE is not as comprehensive as other cognitive assessments (e.g., the Addenbrooke's Cognitive Eaxamination; Mioshi, Dawson, Mitchell, Arnold, & Hodges, 2006, or the Montreal Cognitive Assessment; Nasreddine et al., 2005), it was chosen due to its shorter length. This was considered appropriate given that it was not used for diagnostic purposes, but rather to ascertain participants' positions on a scale of cognitive functioning and to measure any change in cognitive function between the beginning and end of the trial.

Quality of life.

The Quality of Life in Alzheimer's Disease: Patient and Caregiver Report (QOL-AD; Logsdon & Albert, 1999) is a tool specifically designed to assess the quality of life of PWD living in the community. The QOL-AD is designed to be completed independently by both PWD and their carer. Participants rated their feelings about 13 aspects of life ("physical health, energy, mood, living situation, memory, family, marriage/closest relationship status, friends, self as a whole, ability to do chores around the house, ability to do things for fun, money", and "life as a whole") on a Likert scale ranging from 1 (*poor*) to 4 (*excellent*), giving a global score ranging 13-52.

The PWD completed the QOL-AD in the form of an interview, with the researcher showing the participant the categories and scales, reading the questions aloud, and asking the participant to verbally respond or point to their answer. The QOL-AD was discontinued in instances when a participant was unable to understand or respond to more than two items (Logsdon, et al., 1999). The carer completed the report in the form of a questionnaire, rating their partner's situation as they saw it (see below).

The QOL-AD was designed to reflect the four domains highlighted by Lawton's quality of life framework for older adults (as outlined in section 1.3.4; Lawton, 1983, 1991). Past research has shown that the QOL-AD correlates well with other measures representing Lawton's domains (Logsdon, Gibbons, McCurry, & Teri, 2002). It has good internal consistency (Cronbach's alpha coefficient = 0.84), and high

interrater reliability (intraclass correlation coefficients of ≥ 0.75), as well as good test-retest reliability (intraclass correlation coefficients of ≥ 0.6; Logsdon, et al., 1999, 2002; Thorgrimsen et al., 2003). Furthermore the scale is sensitive to changes across time or with therapy (Spector et al., 2003).

Notes.

At the end of the PWD's questionnaire there is space for them (or their carer, or the researcher) to note any additional comments they would like to make concerning their sleep or health.

4.4.2 Questionnaires for Carers, about Themselves

Questionnaires specifically for participants who were carers included questions concerning themselves (sleep, mood and coping) as well as questions addressing the PWD's sleep, dementia-related behaviours and quality of life (see section 1.4.3). The full carer questionnaire can be found in Appendix 6.

Demographic information.

Demographic details included the participant's current age and gender. Carers were also asked if they worked in paid employment outside of the home, and, if so, for how many hours per week.

Sleep.

Carers completed the PSQI (Buysse, et al., 1989) as described above, including the additional option of answering "don't know" to any questions in order to maintain consistency between participant groups. Questions concerning daytime napping and a diagnosis of a sleep disorder were also included as per the PWD questionnaire. Carers were also asked "where do you and your spouse/partner usually sleep?" (Options: *same bed, same room different beds,* or *separate rooms*).

Mood.

Carers completed a short questionnaire concerning anxiety and depression. The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) includes seven statements concerning anxiety (HADS-A) and seven statements concerning depression (HADS-D). Participants rated the statements based on one of the four possible responses. For example, "I feel cheerful", options: *Not at all, not often, sometimes,*

or *most of the time*. Participants were asked to try to give their most immediate response, to describe how they were currently feeling.

Responses were scored (0-3) on the basis that a higher score equals more anxiety or depression. Scores were then added for each domain giving a global score ranging from 0 to 21 (0-7 representing normal range, 8-10 being borderline abnormal, and 11-21 being an abnormally high score). Scores from the HADS are a measure of mood status rather than a diagnostic tool. Different thresholds have been used between studies, making it difficult to assess the validity of the HADS. However Lewis and Wessely (1990) reported acceptable sensitivity and specificity (of approximately 80%). Others have found poorer validity, however this is likely due to the specific patient/disease characteristics, weak external criteria, or high false negatives for patients diagnosed with major mood disorders (Herrmann, 1997). Despite debate over the clinical threshold, the HADS is widely used in research and clinical practice. It correlates well with other mood- related questionnaires (e.g., the Beck's Depression Inventory, The General Health Questionnaire-28, and Spielberger's State-Trait Anxiety Inventory; Bjelland, Dahl, Haug, & Neckelmann, 2002). Furthermore scores have been associated with sleep measures in studies of older people (Jimenez, et al., 1989).

The HADS is short and easy to administer, and so previous studies have had high response rates. It is a reliable measure with good internal consistency (mean Cronbach's alpha coefficients = 0.82 (HADS-D), and 0.81 (HADS-A); Bjelland, et al., 2002) and high retest reliability within 2 weeks (correlation coefficient = > 0.80, decreasing to 70 - 76 after 2 weeks). Furthermore, scores from the HADS have been shown to be sensitive to treatment and interventions (Herrmann, 1997).

Health.

Carers also completed the SCQ (Sangha, et al., 2003) and listed their medications in order to help describe the sample and potentially control for some of the variation between participants.

Carer status.

Questions addressing the caregiving relationship included:

- "How long have you been providing dementia-related support for your partner?" (*Years* and *months*);
- "On average how much time do you spend providing care each day?" (Options: *All day and night, all day, all night, several hours,* or *about an hour*);
- "Do you also provide care/support for others beside your partner?" (Options: *Yes* or *no*);
- "Do you have any support in terms of relief from care-giving, house work, daily chores?" (Options: *Yes* or *no*).

The Carers of Older People in Europe index (COPE; McKee, et al., 2003) was used to measure degrees of caregiver burden. Although many scales are available to measure such burden (e.g., R. Montgomery, Gonyea, & Hooyman, 1985; Vitaliano, et al., 1991; Zarit, Reever, & Bach-Peterson, 1980), the COPE index was selected as it not only measures the *negative impact*, but also the *positive value* of caring and the quality of support that carers receive, which can be important influences on mental health and quality of life (Nolan, Grant, & keady, 1996; Rapp & Chao, 2000).

The COPE index includes 15 questions/statements. Carers rated each regarding their current feelings, using the options: *Always, often, sometimes, never,* or *not applicable* (giving their most immediate response). The index has six questions concerning the negative impact of caring (e.g., "Do you feel trapped in your role as a caregiver?"), and five questions concerning the positive value of caring (e.g., "Do you have a good relationship with the person you care for?"). Ratings were summed to give a global negative and a global positive COPE score, ranging from 0-18 and 0-15 respectively. High scores on the negative scale and low scores of the positive scale indicate trouble coping or burden. Although there are no validated thresholds for the COPE index, Roud et al. (2006) used a score of more than 12 on the negative scale and less than 12 on the positive scale as indicators for trouble coping/burden. There are also four questions concerning the quality of support included in the COPE (e.g., "Do you feel well supported by health and social services?") however a global score is not typically calculated. Answers to the question "overall do

you feel well supported in your role of caregiver" were used to summarise the level of support the carer was getting.

Although the COPE index was developed in Europe, it has been used successfully in NZ with dementia carers (Roud, et al., 2006). Roud et al. found that the index had reasonable internal consistency for both the positive value and negative impact scores from the index (Cronbach's alpha coefficient = 0.59 and 0.69 respectively). Furthermore it has been validated against scales measuring general health (L. Goldberg & Williams, 1988), quality of life (Skevington, et al., 2004), as well as mood and burden (McKee, et al., 2003; Roud, et al., 2006; Zarit, et al., 1980).

Cognitive status.

Carers completed the MMSE (Folstein, et al., 1975) as a structured interview with the researcher. This was included to help describe the sample of carers in comparison to the PWD.

4.4.3 Questionnaires for Carers about the PWD

A set of questionnaires were included for carers to complete about their family member with dementia. Standardised questionnaires have been developed in previous studies in order to gather information about sleep disturbances and dementia-related behaviours which the PWD may be less aware of.

Sleep of people with dementia.

Carers answered the Sleep Disorders Inventory (SDI; Tractenberg, et al., 2003). As outlined in section 2.2.3, this seven-point questionnaire assessing symptoms of dementia-related sleep disturbances. The SDI was developed by expanding the sleep-related item in the Neuropsychiatric Inventory (Cummings et al., 1994), to give greater detail regarding sleep-related changes with dementia. The SDI has the advantage that it includes a measure of carer distress.

For the SDI, carers were required to rate their family member concerning the frequency and severity of the following behaviours: "difficulty falling asleep; getting up during the night; wandering, pacing, or getting involved in inappropriate activities at night; awakening others during the night; awakening at night, dressing, and planning to go out, thinking that it is morning and time to start the day;

awakening too early in the morning; and sleeping excessively in the day". They could also add "any other night time behaviours that are bothersome". Frequency of the behaviour was marked on a Likert scale from 0-4 (*not present in last two weeks, less than once per week, one to two times per week, several times per week but less than every day,* or *once or more per day / every night*). Severity was also rated on a scale from 0-3 (*not present, mild, moderate*, or *marked*). Carers then rated their own distress related to the particular behaviour on a scale of 0-5 (*not at all, minimally, mildly, moderately, severely,* or *very severely / extremely*). The option of *not sure / NA* was also available for each behaviour. This was used in instances such as when the carer was unsure whether behaviours were occurring due to sleeping in a separate room, or being a deep sleeper themselves. The global SDI score represents the average of the seven frequency ratings multiplied by the average of the seven severity ratings, giving a possible range of 0 (*no disturbance*) to 12 (*very severe sleep disturbance*).

Tractenberg et al. (2003) found that scores from the SDI were significantly correlated with actigraphic sleep measures of sleep duration at night (correlation coefficient -0.244, $p = 0.014$), sleep efficiency (correlation coefficient = -0.283, $p = 0.0004$), and wake after sleep onset (correlation coefficient = 0.243, $p = 0.014$). Poorer SDI scores were considered reliable for identifying problem sleepers (as defined by sleeping less than 6 hours at night). Scores were also associated with measures of cognitive functioning and other symptoms of dementia.

Memory and behaviour of PWD.

The Revised Memory and Behaviour Problems Checklist (RMBPC; Teri, et al., 1992) is a carer-completed report of PWD's observable memory and behavioural problems associated with dementia (excluding sleep-related symptoms). It is a 24-item list of behaviours on which carers rated the frequency of each behaviour on a Likert scale from 0-4 (*never occurred, not in the past week, 1-2 times in the past week, 3-6 times in the past week, daily,* or *more often*). They then rated their own reaction to that behaviour on a Likert scale from 0-4 (*not at all, a little, moderately, very much,* or *extremely*). Carers could also rate any behaviour as *don't know* or *not applicable* when necessary (scored as 0). The RMBPC was designed for use in both clinical and research settings. As with the SDI, the RMBPC has the strength that that it combines the occurrence of the symptoms as well as caregiver reaction on the same checklist.

Scores from the RMBPC are divided into three areas to describe dementia-related behaviours. The first subscale includes seven points on the checklist related to memory (e.g., "asking the same question over and over" range = 0-28); the second subscale refers to nine points on the checklist related to depression (e.g., "crying and tearfulness", range = 0-36), and the third subscale refers to eight points on the checklist related to disruptive behaviours (e.g., "destroying property", range = 0-32). Global frequency and reaction scores were also calculated as an indicator of general behavioural disturbance, with higher scores indicating greater frequency or caregiver reaction to the behaviour (range = 0-96).

The RMBPC subscales have been validated against well-established measures of depression (correlation coefficient = 0.44, $p < 0.01$; Hamilton, 1967) and cognitive impairment (correlation coefficient = -0.48, $p < 0.01$; Folstein, et al., 1975). Furthermore carers' reactions to the behaviours within each of the three subscales are consistent with measures of carer burden (correlation coefficients = 0.32, 0.42, and 0.41, respectively, all $p < 0.01$); Deimling & Bass, 1986) and depression (correlation coefficients = 0.29, 0.36 and 0.31 respectively, all $p < 0.01$; Radloff, 1977). Therefore, the RMBPC is considered a reliable all-round checklist of dementia-related symptoms and their effects on carers.

Quality of life of PWD.

Carers also completed the QOL-AD concerning their family member's current situation as they perceived it. This was in the form of a written questionnaire (PWD completed this as an interview with the researcher). As with the PWD's scores, the internal consistency has been shown to be good for carer scores (Cronbach's alpha coefficient = 0.86; Logsdon, et al., 2002).

Carer QOL-AD scores have also been shown to significantly correlate with measures of carer depression (correlation coefficient = -0.53, $p < 0.001$; Yesavage, 1988), and subjective and objective burden (correlation coefficient = -0.53, and -0.52` respectively, $p = < 0.001$; Vitaliano, et al., 1991). Furthermore, both PWD and carer QOL-AD scores have been shown to correlate with some aspects of the RMBPC (Logsdon, et al., 2002).

The scores of PWD and carers from the QOL-AD are less well correlated (correlation coefficient = -0.19, 95% CI = -0.02 - 0.37). However, this is not considered a lack of reliability of the measure, but

rather an indication of real difference in the way PWD and carers perceive the PWD's QOL. This strengthens the argument for using a dyadic measure such as this.

Notes.

At the end of the questionnaire there was space for carers to note any additional comments they would like to make. For example, concerning their own or partner's sleep, health, or the study procedures.

4.5 Materials: Sleep Monitoring

The methods and materials used for objective sleep monitoring were chosen based on their reliability, as well as how easy and flexible they were to use independently by older people and PWD. From the literature reviewed in Chapters 1-3, it was clear that polysomnography was not practical, due to its short recording span and the high demands it places on PWD and carers. Instead, sleep was objectively monitored using actigraphy supported by sleep diaries.

4.5.1 Actigraphy

Actigraphy recording has been used in previous studies with older people and PWD (e.g., D. Lee, et al., 2007; J. L. Martin, et al., 2007; Mishima, et al., 1994; Most, et al., 2010; Werth et al., 2002) and has been identified as a reliable tool for measuring sleep of this population (Ancoli-Israel, Clopton, Klauber, Fell, & Mason, 1997; Pollak & Stokes, 1997; Sivertsen et al., 2006; Van Someren, 2007).

Ease of use and comfort were primary considerations for the choice of actigraph and protocol design. The Actiwatch-2™ ("Actiwatch", Mini Mitter, Philips, Respironics, *Figure 4.1*) was selected. It is a light weight (16 grams) monitor worn on the non-dominant wrist continuously over the data collection period (except when at risk of being damaged). It automatically collects and scores activity counts from an accelerometer which is sensitive to 0.01 g. The sampling rate is 32 hertz, and the device has a non-volatile 1 megabyte memory. It also has a button serving as an event marker for participants to press at relevant times (e.g.,at bedtime), and a sensor for measuring the amount and duration of ambient white light

exposure in units of lux (from the wrist). Finally, it has a waterproof casing which limits how often it needs to be removed compared to other models, which was expected to improve compliance.

The PWD and carers both wore an Actiwatch for the week prior to the introduction of the intervention and throughout the fifth week of the intervention, to objectively monitor their sleep/wake timing (see section 4.8).

Figure 4.1. The Mini Mitter Actiwatch-2 ™

(source:http://www.healthcare.philips.com/main/homehealth/sleep/actiwatch/default.wpd).

4.5.2 Sleep Diaries

Sleep diaries for both PWD and carers were completed to help interpret the actigraphy data. They were also completed throughout the intervention period in relation to the PWD's sleep and compliance with the intervention.

Diaries were in the form of 24-hour timelines and were adapted from those previously used in the Sleep/Wake Research Centre (SWRC) with many population groups (e.g., Gander, et al., 2005a, 2005b; Gibson, Gander, et al.; Signal, et al., 2005). Participants recorded the times of sleep start and end (to the nearest 15 minutes) for any sleep that was 10 minutes or longer. Times when the Actiwatch was removed were also recorded so they could subsequently be excluded from actigraphy analysis. During the intervention period, participants marked the times that the PWD received bright light exposure (either natural or from the LTD) or took part in physical activities.

For each diary day, participants rated the quality of their most recent main (night) sleep on a Likert scale from 1 (*extremely good*) to 7 (*extremely poor*). They could also provide comments regarding any factors affecting their sleep at the end of each week. An example of the sleep diaries for actigraphy and intervention periods can be found in Appendix 7.

4.6 Interventions

The interventions and materials used were chosen based on their potential for effectiveness as well as ease of use and flexibility for older people and PWD. Chapter 3 concluded that a combination of non-pharmacological interventions was a favourable approach for attempting to improve sleep of community-dwelling PWD. It allows participants to use all or some of the options depending on their needs, symptoms and/or abilities. For this study, timed BLT and therapeutic exercise were chosen, along with sleep hygiene education. The light and exercise were prescribed for times of day that were considered favourable in reinforcing the activity of the SCN rather than shifting the phase of sleep timing (see *Figure 4.2*). More information regarding the materials for the intervention is given below (a description of the procedures is in section 4.8).

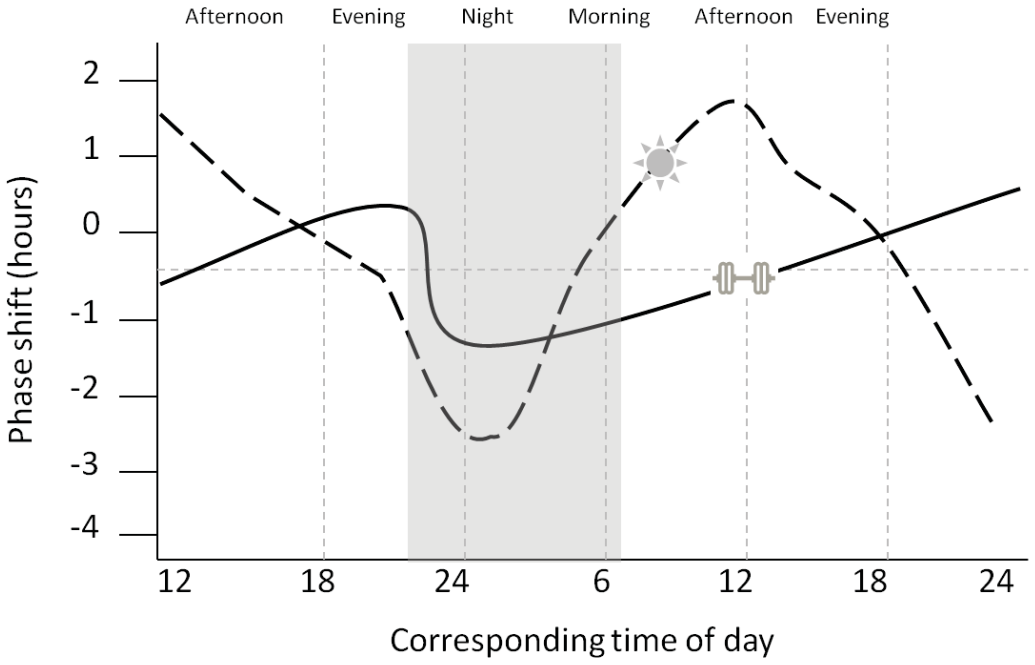


Figure 4.2. Timing of prescribed intervention plotted on the approximate circadian phase. Light exposure (dashed line) and exercise (solid line), grey shading represents typical night time sleep (redrawn from ; Buxton, et al., 2003; Khalsa, et al., 2003).

4.6.1 Bright Light Therapy

During the five week intervention period, the PWD were encouraged to get 30 minutes of bright light exposure every morning (or on as many mornings as possible) between 9-11am. A light box was provided for the intervention period (Day-LightsTM, Uplift Technologies Incorporated, *Figure 4.3*) as an alternative to natural light. However in cases when the participant or GP noted contraindications to artificial light, the PWD used natural light only (see section 4.8.4 for further information on screening).

The Day-Light device was chosen because it meets recommended best practice criteria (Wirz-Justice, et al., 2008). It provides up to 10,000 lux of illumination at a comfortable sitting distance (30-33 centimetres); uses soft, broad-band white light rather than coloured light; includes a diffusing screen to filter ultraviolet light; has three height adjustments so that participants' eyes are positioned towards the centre of the screen; has angle adjustments to project the light downwards and reduce glare; and it uses a large surface area in order to maximise therapeutic range whilst remaining portable. These last factors were considered particularly important for PWD, who may be more likely to move their head or body when in front of the light box, or find unnecessary glare more uncomfortable compared to people without dementia.

Figure 4.3. The Day-Light LTD (Uplift Technologies, 2007).

4.6.2 Therapeutic Exercise

During the intervention, PWD were asked to undertake low intensity exercise on as many days as possible (preferably every day) during the middle of the day (11am-2pm). Carers were encouraged to join in with physical activities or allocate appropriate supervision as they saw necessary. The proposed exercise regime was 30-40 minutes of walking or yoga style stretches and movement. Participants were loaned a senior's exercise DVD (Sit and Be Fit; McLennan, 1999) for the intervention period. Participants were also given a pamphlet in order to promote safe warm down after exercise (Push Play Stretches; Sport and Recreation New Zealand, 2003). These types of physical activity programmes have be introduced successfully in previous research with an emphasis on self-pacing and with use of less intense programmes for the more frail participants (Alessi, et al., 2005; King, et al., 1997; McCurry, et al., 2011).

4.6.3 Sleep Hygiene Education

Participants were provided with a booklet containing sleep hygiene guidelines as well as information on sleep with ageing and dementia. "Sleep Support: A Sleep Handbook for Older People Living with Dementia" (referred to as "sleep support handbook") was written and developed for Study 4. Content was based on the literature review and sleep hygiene models produced for older adults elsewhere (e.g., McCurry, et al., 2003; National Institute of Aging, 2009; National Sleep Foundation, 2011; Sacre, 2010; Song, et al., 2010). Drafts of the sleep support handbook were reviewed by the research team at the SWRC, staff at Alzheimers NZ, and Alzheimers Wellington, as well as a PWD and his spousal carer. Having a broad range of reviewers including the target audience allowed this document to be refined with more appropriate and accessible language whilst keeping the content of a scientific and evidence based nature. The document was targeted more towards the carer than the PWD. However larger spaced type with easy-to-follow icons and design were used in order to make it as appropriate as possible for both. A copy of the sleep support handbook can be found in Appendix 8.

Participants were encouraged to try some of the ideas/solutions offered in the sleep support handbook, and note in the sleep diaries if and when they had tried new things. Participants were also encouraged to give feedback regarding the content and design of the document, as a basis for future revisions.

4.7 Materials: Feedback Questionnaire

As Study 4 was a pilot, it was vital to seek participants' feedback concerning the methods and research processes. A short (10 question) feedback form was designed and sent to all participants at the end of their involvement (regardless of whether they completed the entire protocol, see Appendix 9).

Participants were asked whether they had sufficient information prior to beginning the study and whether any queries had been answered satisfactorily (*yes/no*). They were then asked to rate (options: *Easy, OK,* or *difficult*) and give any comments on:

- the use of the Actiwatch;
- completing the sleep diary; and
- completing the questionnaires

Participants were also asked whether they completed the full trial and if not, for what reason they were unable to complete. They were asked to rate each aspect of the intervention: the light and exercise regimes, and the sleep support handbook (*easy, OK, difficult,* or *not applicable*), and could add comments about each. Participants were also asked if they would recommend the interventions to others (options: *Yes, no,* or *maybe*) and why. Finally, they were given space to note any additional comments.

4.8 Procedure

An overview of the study procedure is provided in *Figure 4.4* and in detail below.

4.8.1 Screening

Potential participants contacted the researcher to show their interest. They were given some general information about the study over the phone, and questions were answered. They were also screened for suitability for the study. This initial screening checked for age, relationship status, and diagnostic status of dementia, as well as living within the Wellington region. If still interested, eligible participants were posted an information pack containing a letter introducing the study, an information

sheet, and consent form (see Appendices 10 and 11) with a free-post envelope to return it to the researcher (if appropriate, see consent processes below).

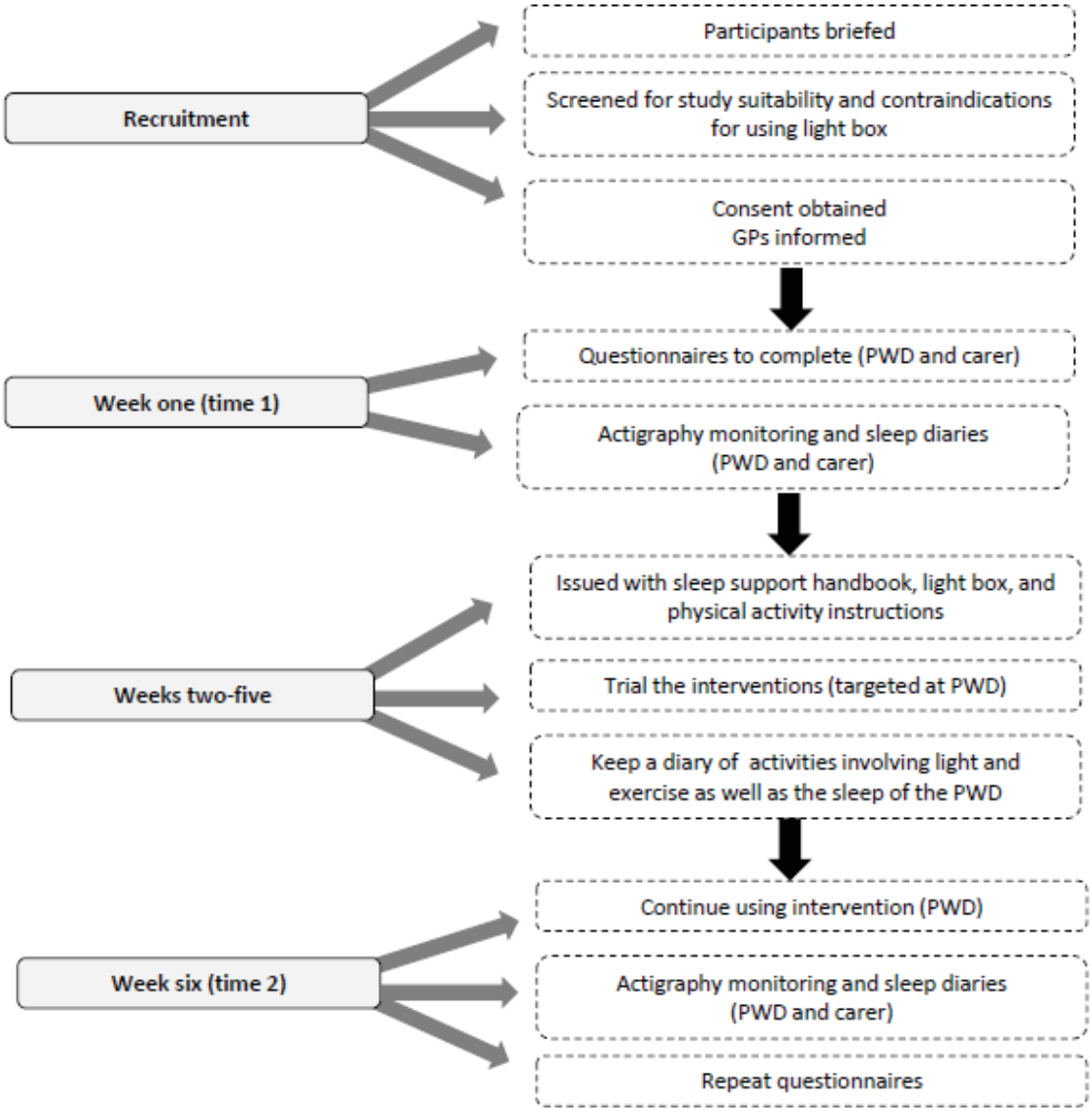


Figure 4.4. Overview of protocol for Study 4.

The PWD were screened for suitability to use the light box. Contraindications to using any LTD include pre-existing medical conditions of the eyes or skin that might cause photosensitivity reactions to bright visible light, or using any medications which may photosensitise their skin and/or retinal tissues (Terman, Remé, Rafferty, Gallin, & Terman, 1990; Uplift Technologies Inc, 2009; Wirz-Justice, et al., 2008). During the recruitment process participants were asked about any eye or skin conditions of the

PWD. They were also required to list the PWD's medications on the consent form for the researcher to review.

4.8.2 Consent

Participants in the earlier stages of dementia were often able to understand the study brief and were able to provide written informed consent. However for those who were in more advanced stages of dementia, or considered less lucid (by their family member) at the time of gaining consent, a written consent process was not considered appropriate or reliable. In these cases, a *Statement by Partner Form* was used, which allowed for the carer to act as a proxy and declare that the PWD would have chosen and consented to participate in the study if he/she had been able to fully understand the information received (Appendix 11).

Once the study was underway, it became clear that an ethical amendment was required with regards to the consent procedure. For some PWD, the consent process itself was considered a risk due to sensitivity surrounding their diagnosis. According to their carers', some PWD denied or forgot their diagnosis, and would become distressed by being asked to take part in a study about "dementia". In these instances the standard consent process was considered the largest study-related risk, causing unnecessary anxiety or hostility, and discouraging PWD or carers to engage with the study. These PWD were typically happy to take part in the protocol due to their interest in the subject of sleep and acceptance of completing questionnaires, using the equipment etc. However, given the sensitivity surrounding the diagnosis and use of the word "dementia" in the documents and consent process, an amendment was approved by the CHDEC allowing the Statement by Partner form to be used if the carer considered that addressing their diagnosis directly would be too distressing.

For all participants, regardless of the method of written consent, verbal assent was also collected. This was achieved though audio recording the primary discussion and briefing, including the participants agreeing to take part. These recordings acted as a dual source of consent and an audio reminder in case the participants requested such verification. Subsequent discussions during home visits were also recorded in order to signify continued consent, assent, and to support the researcher's field notes.

During the consent process, permission was sought from the participants to inform their GP about their participation and the nature of the study. Examples of letters to the GP for carers and PWD can be found in Appendix 12. This gave the GP authority to notify the researcher if they felt that using the light box could be harmful for the PWD (due to the contraindications mentioned above). It also permitted the researcher to notify the GP of any health-related issues identified through taking part in the study (e.g., signs of depression or a clinical sleep disorder). A free-call mobile telephone number and email address were available throughout the study for any questions and comments. Participants were reminded that their information would be kept confidential, and that they had the right to withdraw from the study at any time without having to provide explanation.

4.8.3 Week One (Time 1)

Week one was the baseline recording week (*Time 1*). The researcher visited participating dyads at their home at a convenient time for them. The initial home visit took 60-90 minutes. During this time the researcher introduced herself and reiterated what the study involved for both the PWD and carer. Then the Actiwatches were introduced, followed by the sleep diaries and questionnaires. This order was used as participants were typically intrigued about the concept of actigraphy and tended to have questions concerning this. So it was introduced first to allow them to be reassured and have time to wear the Actiwatch while the researcher was present.

Use of the Actiwatch.

Each Actiwatch was initialised at the SWRC and a log was created linking the participants ID number with the Actiwatch's serial number. Actiwatches were differentiated by colour stickers for PWD (blue) and carers (green) to avoid confusion. Participants were instructed to wear the Actiwatch for seven nights. A note reminding participants to "keep on" was written on the Actiwatch straps to help prevent participants from taking it off when preparing for bed as they might with jewellery. Finally the address of the SWRC was noted on the strap in case of misplacement.

Actiwatches were set to record activity and levels of illuminance in one minute epochs. This is the typical time frame used in actigraphy, unless being used alongside polysomnography recordings (in which

case, 30 second epochs are typically used for ease of comparison). This epoch length also allows for lengthier continuous monitoring whilst maintaining a high level of resolution (Ancoli-Israel, Cole, et al., 2003; Littner, et al., 2003; Van Someren, 2007).

Participants were assured that the Actiwatch only measured movement and light and were shown the event marker. They were asked to wear their Actiwatch on the non-dominant wrist, which is the validated position for adult actigraphy (Sivertsen, et al., 2006). As the Actiwatch-2™ has a waterproof case, participants were instructed to keep it on the for the duration of the monitoring periods accept for any times they were in water for long periods of time or at significant depth (e.g., swimming), or if doing strenuous activities which might damage the Actiwatch.

Participants were told to press the event marker on their Actiwatch to mark times of "starting trying to sleep" and again when "finishing trying to sleep" for any sleep of 10 minutes or longer throughout the day and night. The event marker was also to be pressed at the beginning and end of a period when the monitor was removed to enable exclusion of these periods from analyses.

Based on a review of methodological issues related to using actigraphy in the field (Trost, McIver, & Pate, 2005), the following strategies were put in place in attempt to increase the reliability and compliance of the recordings:

- A supplementary instruction sheet was given reminding participants of the actigraphy and diary protocol (Appendix 13).
- An example of the actigraphy output ("actogram") was shown in the information sheet. To encourage compliance, participants were offered a copy of their actogram at the end of the study.
- Halfway through the study week, the researcher telephoned participants. This contact served as a reminder for participants to be wearing the Actiwatch and to use the event marker. This time was also used to discuss any general questions or issues regarding the study.
- Participants had access to a free phone number on which they could contact the researcher at any time to ask questions concerning the study protocol.

Use of the daily diary.

The diary was explained using an example page. During week one, sleep diaries were required for both the carer and the PWD. Diaries were differentiated by colour for PWD (blue) and carers (green) to avoid any confusion.

The importance of the diary data for interpreting the actigraphy data was explained. Participants were asked to mark the start and end times of day and night time sleeps on the 24-hour time lines. They were advised that they did not have to mark the exact times they fell asleep, rather the times that they were in bed *intending* to sleep, i.e. when they turned the light out to try and start sleeping until when they woke up and stopped trying to sleep in the morning. These times were used to help define *rest intervals* within the actigraphy data (see section 4.9.3). If the participants woke in the night (e.g., to go to the toilet or general restlessness) they were not required to mark the awakening unless they were up for more than 10 minutes. Participants were reassured that it was not compulsory to mark these longer awakenings during the night as it may inadvertently make them feel more alert, but they could mark the approximate times in the morning with the assurance that the Actiwatch would verify the awakening. Carers typically maintained the diary for the PWD, or supported them to complete it themselves.

Each morning, participants were asked to rate the quality of their sleep from the night before on the Likert scale. For participants who had difficulty remembering how well they slept, it was recommended that they discussed the topic together (perhaps over breakfast), in order to come to a decision together. At the end of the diary week participants were encouraged to include notes of factors which might have affected their sleep (e.g., if they were unwell, or had visitors). Magnetic clips were provided for participants to display their diaries (e.g., on the fridge) for easy access and to act as a reminder to complete them.

Use of the questionnaires.

The questionnaires were also colour coded (blue for PWD and green for carers). The PWD's questionnaire was completed with the researcher present for support. The method of completion varied dependent on the cognitive ability of the PWD, this was participant-led. Some read, understood and completed the questionnaire alone, with a little clarification from the carer; others completed the

questionnaire more in an interview style, with the researcher asking questions and completing the paperwork while they read along; and in instances of severe cognitive impairment, the carer would answer the majority of the questions on behalf of the PWD, or complete the paperwork for them. The time taken to complete the questionnaire of the PWD varied between 10-30 minutes.

In the interests of sensitivity, the subject of the type and diagnosis of dementia was often left to be raised naturally in conversation, rather than being asked casually amongst the other questions. This method was used after feedback that some PWD and carers have sensitivity surrounding the language used to describe dementia, as well some PWD having issues with disbelief or not knowing/acknowledging their diagnosis.

Carers were also given their questionnaire at the home visit. They were asked to check that they understood the questions and clarify any points while the researcher was there. They were encouraged to begin completing the questionnaire during the home visit, but as their questionnaire was longer than the PWD's, they were typically left to complete it in their own time. This protocol was used to prevent the home visit exceeding two hours and becoming tiring for the participants.

The MMSE and the QOL-AD interviews were conducted one-on-one between the researcher and the PWD. Carers were asked to leave the room if they felt comfortable to do so. The carer typically sat in an adjacent room in the house (giving them a chance to begin their questionnaire). This procedure gave the PWD privacy when completing their quality of life questions and also meant that the carers were unable to hear the answers to the MMSE before they were also tested. Once the interviews were complete (approximately 10 minutes) the carer then returned to the room and completed their own MMSE.

Completion of the first home visit.

Once all areas of the research and particularly the first week's tasks were completed, participants were free to ask any further questions and the comfort of the Actiwatches was checked. They were left with a folder each (blue for the PWD and green for the carer) to store their sleep diaries and study information. The MMSE and QOL-AD interview paperwork was taken directly back to the SWRC,

however the questionnaires were left with the couple during the first week for the carer to have time to complete theirs and for any additional comments to be added.

4.8.4 Weeks Two-Five

At the beginning of week two the researcher visited the participants again. This home visit was for collection of the sleep monitoring materials and delivery of the intervention. At the beginning of the visit, participants removed their Actiwatches and were asked for feedback on how they managed with wearing them and maintaining the sleep diaries. The questionnaires and diaries from week one were collected and briefly checked for any missing data or areas which needed clarification (e.g., hard to decipher handwriting).

Introduction of intervention.

Participants were introduced to the intervention materials and given an information sheet covering each aspect of the intervention (light, exercise and education, Appendix 14). It was stressed that the crux of the intervention was maintaining a more regular and timed exposure to the environmental cues that help keep the body clock synchronised to the day/night cycle. If they also gained light exposure or exercise outside of the recommended routine, this was not problematic, but they were requested to keep to their new routine on as many days as possible.

Participants were lent the Day-Light light box for the 5 week intervention. They were instructed to use the light box (or get equivalent natural light exposure) for 30 minutes around the same time each day within the time-frame of 9-11am. Participants were shown the light box and the researcher set it up for them, making sure the light was at the correct height and angle for the PWD to use. Participants were required to sit upright approximately 30 centimetres away from the light, which was positioned just above eye level. They were informed not to stare directly at the light, and that they could perform sedentary activities such as reading, eating, or talking whilst using it (as per manufacturers user guide; Uplift Technologies Inc, 2009). Reminders of how to use the light were attached to the frame with a 30-centimetre piece of string as a guide of where to sit. Safety instructions were also included in the information sheet. At the home visit, the PWD was asked to sit in front of the light to "give it a go" while

the researcher was present. This gave participants the opportunity to give immediate feedback, and for the researcher to assess the positioning and their response to the bright light.

The researcher then introduced the physical exercise aspect of the intervention, again stressing the importance of activity for maintaining the timing of the body clock. Participants were asked to take part in 30-40 minutes of exercise around the middle of the day (between 11am-2pm), on as many days as possible. They could maintain whatever physical activities they were already doing (e.g., walking to the shops or swimming) or begin new activities based on the intervention, but were asked to perform these activities daily if possible and within the specified time frame. Participants were lent the senior fitness DVD (McLennan, 1999). This was recommended for those who were less physically able, as well as for days with bad weather or when considered less easy to leave the house.

Carers were encouraged to join in with the exercise program in order to support the PWD, especially in instances when the PWD was less cognitively able and at greater risk of getting lost whilst on a walk or injuring themselves during exercise. Although 30-40 minutes of exercise was the recommendation, participants were advised to do as much activity as they felt able. The DVD contained a 45 minute programme, but if this was deemed too long or strenuous for the individual, participants were advised to stop when necessary. Participants were issued with the guide to stretching and were encouraged to consult with their doctor if they felt they had specific medical conditions which may affect their ability to undertake the physical activity aspect of the intervention.

Finally the participants were given the sleep support handbook. The researcher showed and described the two sections of the handbook (firstly the overview of sleep, ageing and dementia; and secondly the advice and tips for healthy sleep). Participants, particularly carers, were encouraged to read the book in order to gain a better understanding of how and why sleep may be changing for them and their family member. Participants were encouraged to try and identify the factors that could be associated with any of their sleep problems and to try some of the sleep recommendations.

Continued diaries.

During week's two to five, diaries were kept only for the PWD. Participants were requested to continue logging sleep start and end times as per week one. In addition they were asked to log the times that they used the interventions. These times were marked in diary and coded as *physical activity* (PA), *natural light exposure* (NL), or *using the light box* (LB). At the end of each week's diary, participants also had a space to write any comments as well as indicate whether they had tried any new sleep strategies from the sleep support handbook. These diaries acted as compliance monitoring for the intervention and also allowed for subjective sleep monitoring of the PWD over the entire intervention period.

At the completion of the home visit, participants had the opportunity to ask any further questions. To help improve compliance, participants were given a fridge magnet with reminders to use the light, get their exercise and complete the diary. This also had the contact details for the researcher who they could contact at any time (Appendix 15). The researcher telephoned participants once a week to check on their compliance with the intervention and diaries, as well as to answer any of their questions and to offer general support for their continued participation in the study.

4.8.5 Week Six (Time 2)

Repeat sleep monitoring.

Week six was the final week of the intervention trial (*Time 2*). Participants continued to use the sleep intervention as per weeks two to five. However they also repeated the sleep monitoring. At the end of week five, participants were reissued with Actiwatches and sleep diaries to complete as per Time 1. These were either delivered by the researcher or couriered, depending on participant distance and availability. The researcher reminded the participants about how to use the Actiwatches, referring them back to the instruction sheet. Participants were reminded to continue using the sleep interventions and logging the times in the PWD's diary. The carers also completed a sleep diary for week six, logging their sleep timing only.

Repeat questionnaires.

At the end of week six, the researcher visited the participants for a final time. After collecting the Actiwatches and checking the diary data, the researcher gave participants the Time 2 questionnaires. These questionnaires were identical to Time 1 except that the demographic questions were omitted. Participants were asked to list any changes in medications or medical conditions since Time 1. Finally the MMSE and QOL-AD interviews were administered (as per Time 1).

In order to control for any time-of-day effects on questionnaire responses or cognitive ability, the researcher scheduled the Time 2 home visit at approximately the same time of day as at Time 1. To maintain consistency within subjects, the PWD's questionnaire was administered in the same way as they had completed at Time 1 (i.e. by the PWD, as an interview, or by the carer).

Completion of the study.

Questionnaires, diaries and equipment were returned to the SWRC at the end of week six. On successful download of the Actiwatches, participants were posted copies of their actograms with a letter briefly annotating the findings (see example in Appendix 16). The feedback questionnaire and a freepost envelope were also sent. Participants were encouraged to complete the feedback form with frankness in order to help design future studies, and were reminded that their feedback would be kept anonymous.

On review of the questionnaire data, GPs were informed of any unusual results concerning sleep or cognitive impairment of PWD; and/or sleep, mood, or issues coping for the carers, in case these areas required clinical follow up (example letter Appendix 17).

4.8.6 Withdrawal

The study protocol was to be terminated in the following circumstances.

- The participant experienced adverse effects due to the interventions.
- The participant's medical condition deteriorated, preventing them from being able to take part or significantly altering their sleep (e.g., suffering an injury, or starting or stopping sedating medications).

- The participant actively decided to withdraw from or refused to continue the study protocol (e.g., for personal reasons, no longer consented, not interested, agitated or distressed during data collection).

  Of the 15 pairs who enrolled into the study, 6 withdrew for one or more of these reasons.

4.9 Data Analysis

Initial applications to funding bodies and the CHDEC proposed that inferential statistical analyses (or example linear mixed models, paired t-tests, and McNemar tests) would be used to interpret the data with regards to effects of time (pre vs. post intervention), role (PWD vs. carer) and completion status (those who completed the trial vs. those who withdrew). However the final sample was too diverse and underpowered for reliable statistical analyses of this kind. Questionnaire, diary, and actigraphy data were processed and compared as intended but are presented in a descriptive form and as case studies. This avoided overanalysing the results and allowed for a more holistic approach to each dyad's data.

Table 4.7 provides references for interpreting the variables derived from the questionnaires, diaries and actigraphy, and more detail is provided below. This chapter concludes with an overview of the case-study design.

Table 4.7

Reference for Interpreting Questionnaire, Diary and Actigraphy Variables

Measure	Representing	Range	Interpretation
Pittsburgh Sleep Quality Index (PSQI)*	Sleep disturbance	0-21	>5 = disturbed sleep
Sleep Disturbance Inventory (SDI)*	Dementia-related sleep disturbance	0-12	Continuous scale: 0 (no disturbance) - 12 (very severe disturbance)
Rating of night's sleep	Sleep quality, median score from daily diary	1-7	Continuous scale 1 (extremely good) - 7 (extremely poor)
Mini Mental Status Exam (MMSE)	Cognitive functioning	30-0	23-30 = normal, 19-23 = mild cognitive impairment, 10-18 = moderate cognitive impairment, ≤ 9 = severe cognitive impairment
Quality Of Life in Alzheimer's Disease (QOL-AD)	Quality of life of PWD living in community	52-13	Continuous scale completed by both PWD and carers: 52 (excellent)-13 (poor)
Revised Memory and Behaviour Problem Checklist (RMBPC)	Dementia-related symptoms		
	Memory	0-28	Continuous scale of the sum of behaviours that occurred in the
	Depression	0-36	previous week, and carer reactions, 0= not occurred- 28/36/or 32
	Disruptive behaviours	0-32	= occurred and carer extremely bothered/upset by the behaviour
Hospital Anxiety and Depression Scale (HADS)	Mood of carers	0-21	risk for anxiety or depression , 0-7 = normal, 8-10 = borderline, 11-21 = heightened
Carers of Older People in Europe index (COPE)	Carer coping/burden		
	Positive impact of caring	15-0	<12 = reduced positive impact of caring
	Negative impact of caring	0-18	>12= increased negative impact of caring
Total sleep per night (actigraphy)*	Sleep duration	Minutes	<360 (6 hours) = problematic
Sleep efficiency/ % sleep night (actigraphy)*	Sleep quality	percentage	<85% = problematic
Number of wake bouts night (actigraphy)*	Sleep quality	Counts	Continuous scale

* = key variable

4.9.1 Analysis and Presentation of Questionnaire Data

Management of questionnaire data.

The first two dyads recommended that the option of *don't know* should added to the PSQI. This was included so as to avoid participants feeling they had to guess how often behaviours occurred or not answering the question at all. Where participants marked *don't know* this was scored as for the behaviour *not occurring during the last month* (0). Likewise, in other scales, if the participant marked or overwrote *don't know* or *not applicable,* the item was scored as 0. In instances when participants marked two responses or in-between two responses, the average of these responses was used.

Where possible, participants were contacted to see if they could complete missed questions. However some felt they were unable to answer the questions retrospectively, or were unable to be reached before analysis took place. In such instances, the following methods of item imputation were used:

- If the missing score was related to a carer severity or reaction score within the RMBPC or SDI and the frequency score was 0, then the missing score was also marked as 0, based on the assumption that one cannot have a reaction to a behaviour that does not occur.

- If missing data affected three or more of the seven PSQI components, then a global score was not calculated and was considered missing. In instances when one or two components were affected by missing data, an average score from the completed components was substituted for the missing component/s in order to estimate a global score.

- For all other scales which had a global score to calculate, an 80% threshold was used. If less than 80% of the scale was complete, the entire scale was considered missing. If 20% or less of the items were missing, these were substituted with an average score from the complete items within the scale. This allowed for global scores to still be estimated.

Due to an error with the initial questionnaire collation, the first five carers had a section of the RMBPC missing. This included four items of the depression checklist and one item from the disruptive behaviours checklist. For these participants, scores concerning depression frequency and severity are

unavailable, and global RMBPC scores could not be calculated due to greater than 20% of the items being missing.

Presentation of questionnaire data.

Demographic data were summarised for each pair. These factors could not be controlled for statistically due to the small sample size. Global scores were calculated for each validated questionnaire and its subscales. For scales with established threshold values, participants were categorised according to whether they scored above or below the thresholds. Descriptive statistics were calculated for the entire group at Time 1 in order to understand how the sample compared to previous research samples of this type. Individual scores were plotted on bar charts and scatter graphs to give a visual indication of where each participant was placed with regards to the group median. These also allowed for comparisons to be made within participants between Times 1 and 2 and to visually compare the distribution of results between participants who completed the full study versus those who did not.

Within each case study, questionnaire scores were tabulated for the PWD and carer. The difference between Times 1 and 2 was calculated to give the points of improvement or deterioration within the scale. Note "improvement" does not necessarily relate to an increased score, nor does "deterioration" necessarily relate to a decreased score, as this is dependent on the design of the scale. The SDI and PSQI scores were considered key with regard to trouble sleeping. Elements within each of these scales, together with participants' comments, are described to give a more complete portrayal of the factors disturbing the participant's sleep.

4.9.2 Analysis and Presentation of Diary data

Sleep timing from the diary was used for the analysis of the actigraphy data only. Sleep quality ratings regarding the most recent night's sleep were averaged for each participant. The average score was used as participants expressed some confusion over which night's sleep they were rating on the scale.

Compliance with the intervention was estimated from the diaries. The number of days with light or activity logged within the allotted time frames was calculated to complement the objective recordings

from the Actiwatch. Use of the LTD and exercise outside of the allotted times was also identified and described.

4.9.3 Analysis and Presentation of Actigraphy Data

Determination of sleep and wake.

The Actiwatches were downloaded using Actiware® software (2005, Version 5, Bend, OR: Mini Mitter, Respironics). The data is initially presented as an actogram. The actogram example in *Figure 4.5* runs from midnight to midnight. Black vertical lines represent activity (underscored by a horizontal red line) on a scale of 0-200 counts per minute. The yellow lines represent ambient white light on a scale of 0.1-50,000.0 lux, as recorded from the participant's wrist.

The Actiware® software includes a validated algorithm that categorises each epoch of the recording as either *sleep* or *wake*. The software considers not only the particular epoch, but the weighted fractions from the two epochs before and after (*Figure 4.6*). The sum of the activity counts from the epoch of interest plus fractions of the activity counts from the surrounding epochs is compared to a set threshold for wake (low [20], medium [40], or high [80]). If the sum of activity counts exceeds the threshold, then the epoch is scored as wake. If the sum is below the threshold, then it is scored as sleep.

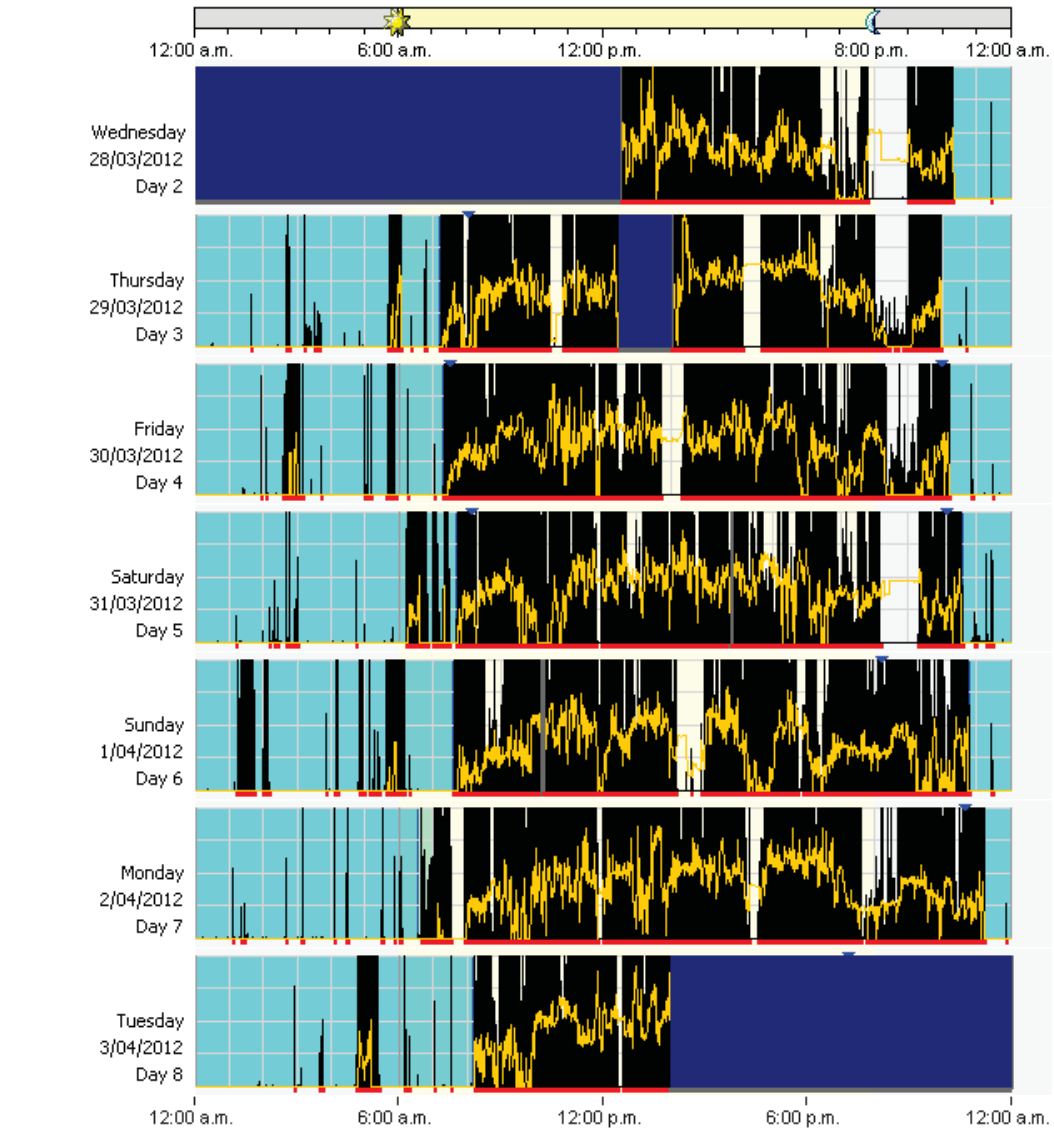


Figure 4.5. An example of 1 week of actigraphy recording from a PWD.

Activity scale: 0-200 counts, white light scale: 0.01-50,000.0 lux.

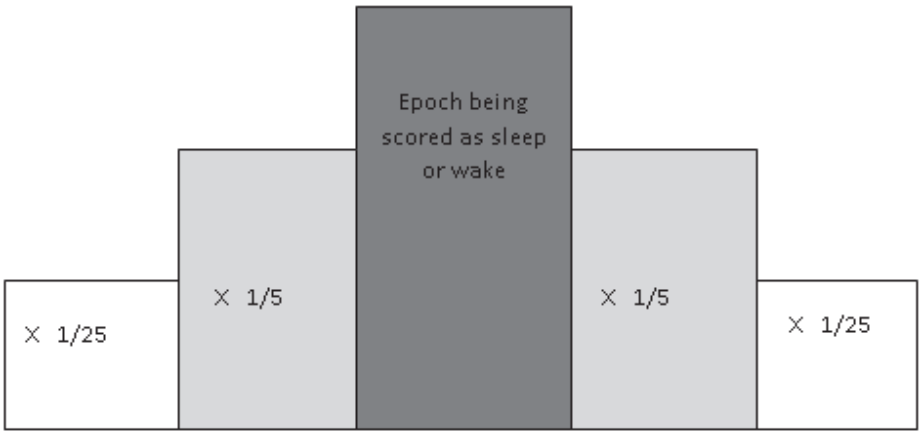


Figure 4.6. Actiware® software *algorithm for calculating the sum of activity counts for a 1 minute sampling epoch in order to score sleep or wake.*

Some previous research using actigraphy with older people suggests using a lower threshold setting, based on the assumption that older people move less so less movement should be required to define an epoch as wake (Kushida et al., 2001; Van Someren, 2007). However, this is not standardised for PWD and many have sleep disturbances related to excessive activity, so the lower setting may not be justified. In previous studies of healthy males aged 30-50 years, actigraphic estimates of sleep duration were shown to correlate highly with polysomnography regardless of the threshold setting (range = 0.84-0.95; Signal, et al., 2005). The medium setting is typically used for adults aged less than 65 years. As participants who were carers could include the PWDs' family members who were younger than 65 years, using the lower setting was deemed inappropriate. For consistency, the threshold for defining wake was set to medium (40 activity counts per minute) for analyses of all of the actigraphy data in the present study.

The actigraphic software provides statistics related to periods of time in bed, sleep, and wake. However the software cannot distinguish between sleep and still wakefulness or the Actiwatch not being worn. Therefore manual scoring of times in bed (or *rest intervals*) is necessary, as is manually excluding any periods of invalid data. For this purpose, diary data are essential (Sadeh, Hauri, Kripke, & Lavie, 1995).

Manual scoring of actigraphy intervals.

Two different types of intervals were identified by manual scoring. Rest intervals were defined as the time the participant spent in bed. The software can then directly apply an algorithm which defines a

sleep interval within the rest interval (see below). Rest intervals were identified based on three sets of information:

1. The sleep diary kept by participants indicating times in bed and times the monitor was off;
2. the event marker should have been pressed at the beginning and end of bedtimes and removed times, which results in an arrow-like marker being plotted in the actogram;
3. a change in the amount of light around the times of rest indicating *lights out*; and
4. a change in activity around times of rest.

Excluded intervals were defined as any time that the participant was not wearing the Actiwatch (e.g., while swimming, if it fell off, or was removed) and were not analysed. Times when the Actiwatch came off were not always noted in diaries. For consistency, the following rules for excluding periods of time were created:

1. Any periods of time with no activity for 1 hour or more and not already noted as a rest interval;
2. times prior to the study beginning and when the Actiwatch was in transit back to the SWRC;
3. any rest intervals containing an excluded interval (e.g., if the Actiwatch slipped off in the night) were fully excluded; and
4. days when the participants were noted as being in respite or hospital.

Reliability of manual actigraphy scoring.

There were 24 sets of data available for the PWD, of which 12 (50.0%) were double scored to check inter-rater reliability. A total of 187 rest intervals were re-scored and any discrepancies of more than 15 minutes for either start or end times were flagged and re-analysed. Of the total time points (374), there were 57 discrepancies which gave an agreement rate of 84.8%.

There were 21 sets of data available for the carers, 11 of which (52.3%) were double scored. A total of 85 rest intervals were rescored as above. Of the total time points (190), there were 15 discrepancies which gave an agreement rate of 92.1%.

Software defined intervals.

Once rest and excluded intervals have been identified manually, the Actiware® Software (2005) was used to define sleep intervals (from initial sleep onset to final wakeup) within the manually identified rest intervals. All other times are defined as *active*. Sleep onset was defined as the first of 10 consecutive minutes within the rest interval, with all but one epoch being defined as immobile. Final wakeup was defined as the last epoch of the last 10 consecutive minutes within the rest interval, with all but one defined as immobile. The time from initial sleep onset to final wakeup is known as *assumed sleep*. Sleep intervals were used to produce sleep propensity curves for each participant (see below). Note that sometimes the participants would go to bed or intend to have a nap but not fall asleep according to the algorithm. Therefore not all days have the same number of sleep intervals and rest intervals.

Defining actigraphy variables

The rest intervals were further categorised as occurring during either the night or day. *Night* was defined as the time the participant initially went to bed intending to sleep (*bedtime*) until the final morning rising (*rise time*). Note if participants had reported periods of wakefulness during the night, these periods were still scored as within the night sleep interval. The remaining time constituted *day* intervals. This method was based on previous research of PWD living in the community (McCurry, et al., 2011). It is consistent and reliable given that some participants did not record or were unaware of periods of wakefulness in the night.

For each participant, for each day of recording, descriptive statistics were calculated for actigraphic variables concerning sleep timing, duration and efficiency. These variables were all calculated from the rest interval (i.e. the time in bed) and are described in Table 4.8. Appendix 18 contains an example using 48-hours of actigraphy recording to step through how the software defines sleep intervals and how the variables of interest are calculated from the data.

Table 4.8

Descriptions of the Variables Calculated from the Actigraphy Records

Variable	Description
Bedtime (hours : minutes)	The time that the participant went to or was put to bed (the beginning of the rest interval)
Risetime (hours : minutes)	The time that the participant got up or stopped trying to sleep (the end of the rest interval)
Time in bed (minutes)	Total time in bed/duration of the rest interval (manually scored)
Total sleep per night (minutes)	Total time within the night rest interval defined as sleep (defined by the software algorithm)
Sleep efficiency (% sleep night)	The proportion of the night time rest interval spent asleep (defined by the software algorithm)
Wake time night (minutes)	Total time within the night rest interval defined as wake (defined by the software algorithm)
Number of night wake bouts	Total number of continuous blocks, one or more epochs in duration, with each epoch of each block scored as wake, within the night time rest interval (defined by the software algorithm)
Total sleep per day (minutes)	Total time within the day rest interval defined as sleep (defined by the software algorithm)
Total light exposure (lux-minutes)	The sum of all valid light data in lux, for all epochs from the beginning to end of the interval
Average activity (counts/minute)	The average of all valid physical activity counts for all epochs from the beginning to end of the interval

The Actiware® software was also used to estimate the PWD's compliance with using the interventions. Rest intervals set at 9-11am and 11am-2pm were defined during all days without any excluded intervals within these timeframes. These were used to calculate the total white light exposure (within the scale of 0.01-50,000.0 lux), and the average activity counts per minute (within the scale of 0-200 counts). These were used to describe how much light and activity the PWD experienced at baseline. Data was also compared between Time 1 and 2 to assess any changes in light and activity exposure during the allotted timeframes.

Presentation of actigraphy data.

Once invalid actigraphy days were excluded (as described above), actigraphy data were averaged for each day of monitoring and then for each week (Time 1 and Time 2) per participant. As the number of valid actigraphy days varied between participants, the averages for each variable per participant were calculated and then averaged across the whole sample to describe the sleep of the carer and PWD groups. Since many actigraphy variables were not normally distributed, the median and ranges are presented as well as means and standard deviations.

A purpose-built algorithm was used to calculate the percentage of monitored days on which a participant was asleep (defined by actigraphic sleep intervals) in each 10 minute interval across the 24-hour day. This method, known as a "sleep propensity curve", summarises an individual's sleep/wake pattern and can be visually compared between Times 1 and 2. *Figure 4.7* shows an example. In cases where the participant's sleep was particularly disrupted, an excerpt of their raw actigraphy data is also presented within their case study.

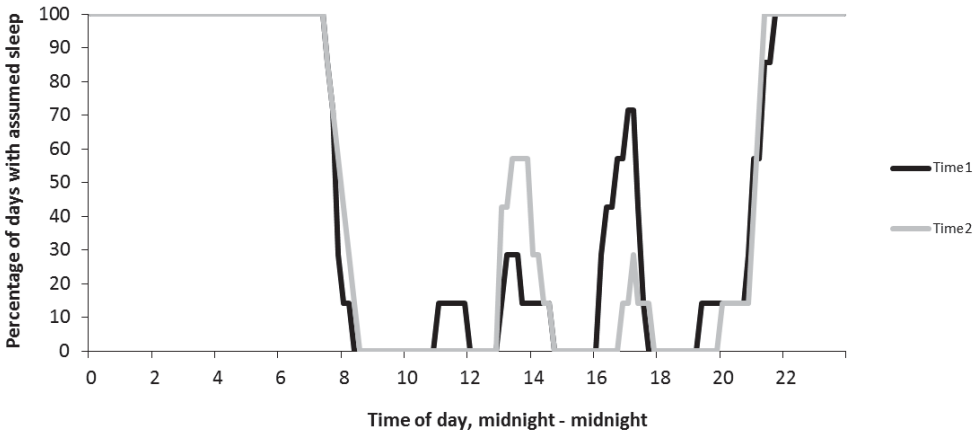


Figure 4.7. An example of the sleep propensity curves of a participant with dementia, based on seven 24-hour periods at Time 1 and at Time 2.

In order to include as many 24-hour days as possible for each participant, incomplete days (i.e. not running from midnight to midnight such as those at the beginning or end of a study) were pasted together to create a "complete" 24-hour period to be used by the sleep propensity algorithm. For participants who completed the full trial, Time 1 and Time 2 data were matched for number of days. If one week had more complete days of recording than the other, the studies were matched as far as possible by days of the week.

4.9.4 Analysis and Presentation of Field Notes and Feedback

Field notes were collected at the end of each home visit and after telephone conversations. Comments regarding study and intervention compliance were used to describe the participants' experience with the research. In some cases these notes also gave insight into the participant's background and experience with dementia.

Descriptive statistics from the nominal questions in the feedback form were calculated and are presented for those who completed versus those who did not. Comments within the feedback form were used to help describe each participant's experience with the research and are summarised as themes.

4.10 Approach to the Presentation of Results

The conceptual framework developed in section 2.2.4 (*Figure 2.5*) highlights the complex and variable nature of the factors affecting the sleep of PWD. Due to this complexity and the small sample size, a critical realist approach was used to present the results from the intervention study. Critical realism argues that we cannot isolate or control for factors, expecting to make reliable predictions about a phenomenon (Danermark, Ekström, Jakobsen, & Karlsson, 2002). Rather the mechanisms producing the phenomenon of interest are identified whilst considering the continuous changes likely within the participant. In the present research, dementia-related symptoms and caregiving requirements were observed to fluctuate throughout the trial. Therefore no attempts were made to statistically predict which factors contributed to healthy sleeping or the successful influence of the interventions. Instead a more holistic approach was taken, and the results are presented per dyad in the form of case studies.

Using case studies allowed for additional factors that could have affected sleep (other than the intervention) to be considered. This design is an ideal alternative to an extensive piece of empirical research with a larger sample size. Each case can be presented completely and with vigour, as well as within the framework of the study protocol (Blampied, 1999, 2001; Ragin, 1992; Thomas, 2011). In Study 4, this allowed for the questionnaire, diary and actigraphy results to be interpreted in their quantitative or categorical forms, whilst also providing a qualitative "story" concerning each dyad. This method gave an opportunity to present the results in a person-centred form, giving space for the voices of the PWD. Pseudonyms are used throughout the thesis to protect participant anonymity and no information that could identify them to any reader other than themselves is given. Information regarding the literal presentation of the case studies is given in the introduction to the results chapter (section 5.1).

5 STUDY 4 RESULTS

This results chapter is organised into three sections. The first gives an overview of the full sample's data at Time 1 (N=15). Results summarising the questionnaire, diary and actigraphy data (including light and activity) are presented in order to frame the sample with regards to previous studies of older people and PWD, as well as give a reference point whilst interpreting the individual case studies presented in the following section. Participants' data are plotted on a series of bar charts and scatter plots in order to visualise the baseline data, and to make comparisons between participants who completed the trial versus those who withdrew, as well as between PWD and carers.

Case studies concerning the pairs who did not complete the entire trial were produced to help develop a greater understanding concerning the feasibility of conducting community-based dementia research. These cases are summarised in section 5.2 and can be found in Appendix 19.

Section 5.3 is a set of case studies concerning the nine pairs who completed the full study (Times 1 and 2). Each case follows the same format to maintain consistency and to allow each pairs' data to be presented in its entirety.

Available data.

Of the 15 dyads, nine completed the full trial and Time 2 assessments (pairs coded 1-9). Time 1 questionnaires were completed concerning all 15 PWD and carers. Actigraphic data were available for all PWD and 14 of the carers at Time 1 (carer 6 refused to wear the Actiwatch). There were no equipment faults. Complete days of actigraphy data were calculated for each participant. This corresponded to the number of days with a full night's rest interval (whether within the original 24-hour period or spliced together), once excluded periods were omitted. Overall, participants had good compliance with using actigraphy. The PWD who completed the full trial had an average of 7.1 complete days recorded at Time 1 (range = 5-11 days), and 6.1 at Time 2 (range = 3-8 days). Those who withdrew from the study had an average of 5.5 complete days of recording at Time 1(range = 3-7). Carers who used actigraphy also had good compliance. Those who completed the full trial had an average of 7.1 (range = 7-8) and 6.7 (range =

5-7) complete days of recording at Times 1 and 2 respectively. Carers who withdrew had an average of 5.4 complete days at Time 1 (Range = 0-7).

5.2 Description of the Full Sample at Time 1

5.2.1 Demographic Details

Table 5.1 summarises the demographic details of all 15 dyads. A brief outcome summary or reason for withdrawal is also provided for reference. All but two pairs were in a spousal relationship. Pairs 6 and 14 consisted of a PWD and their adult children who were carers. The majority (n=11) of the carers were not involved with any kind of paid employment. However four were employed (ID's 5, 6, 8 and 14) and were working 20-40 hour weeks as well as providing informal dementia care (two of these workers were the younger son/daughter carers).

The most common comorbid health conditions amongst the PWD were high blood pressure (60%), heart disease (45%), and lung disease (33%). Of the full sample of PWD, 27% had depression. Amongst the carers, arthritic and back pain were most common (both 27%), followed by high blood pressure (20%). Just one carer (6%) had a diagnosis of depression. Appendix 20 contains tables summarising the medications that were being used by the PWD and carers, with indications of any side effects pertaining to sleep. The PWD were taking a mean of 4.6 medications (SD = 2, range = 1-8), and 40% were taking more than five. The carers were taking a mean of 2.3 medications (SD = 3.1, range = 0-12), and 6% were taking more than five. The majority of medications being used by both the PWD and carers were for cardiovascular-related conditions.

None of the participants had been diagnosed with any sleep disorders except for PWD 9 who had a previous diagnosis of OSA, however this was considered to be resolved at the time of the study. Despite the absence of diagnosed sleep disorders, in the PSQI, seven of the PWD were identified as snoring loudly at least once or twice per week. Of these seven, four also reported some long pauses in their breathing during sleep, and three of the PWD reported that their snoring disturbed their sleep. Four PWD were also reported to have symptoms of RLS.

Table 5.1

Demographics of the Full Sample and Summary of Each Dyad's Outcome

ID	Pseudonym (PWD & carer)	PWD age	Carer age	Dementia Type	PWD MMSE	Outcome summary
Those who completed:						
1	Liam & Sophie	73	73	VaD	15	Dementia-related deterioration, PWDs' subjective sleep disturbances declined
2	Jack & Ella	82	74	VaD	13	Dementia-related deterioration, PWD and carers' subjective sleep disturbances declined
3	George & Amy	72	71	AD	14	Good quality sleep at onset, some subjective improvement
4	Thomas & Sarah	76	74	Undefined	23	Good quality sleep at onset, no improvements observed, daytime sleepiness
5	Adam & Claire	82	68	LBD	5	Subjective and objective improvement in sleep and QOL ratings
6	Anne, Matthew, & Katie*	83	46	Undefined	27	Minor subjective and objective improvements to sleep, however carer reported deterioration
7	Violet & Charlie	73	78	AD	16	Good quality sleep at onset, no improvements observed
8	Liam & Phoebe	66	67	AD	18	Good sleep quality at onset, no improvements observed. Struggled with protocol
9	Andrew & Emily	80	54	VaD	7	Carers' sleep disturbed by PWD, little improvements observed, carer stressed
Those who withdrew:						
10	Nicole & Claude	80	81	AD	21	Withdrew due to PWD moving into care home at end of week 1
11	Henry & Beverly	72	68	AD	13	Withdrew as PWD admitted to hospital
12	Sidney & Fiona	88	75	MCI	29	Withdrew after losing motivation after period of respite care
13	Maria & Felix	83	82	VaD	20	Withdrew at the end of week 1, did not consider sleep problematic enough
14	Georgina & Lisa*	89	55	Undefined	23	Withdrew during as intervention protocol considered too stressful
15	Edward & Rose	73	55	VaD	28	Withdrew during week 1 due to PWD moving into care facility after a fall

* = carer was the child of the PWD rather than spouse

Of the carers, four reported that they were a loud snorer, one of whom had pauses in their breathing. All four reported that their snoring disturbed their sleep at least once or twice per week. Two of the carers reported symptoms of RLS at least once or twice per week.

5.2.2 Summary of Results at Time 1

Tables 5.2 - 5.5 summarise the questionnaire and actigraphy data of both the PWD and carers. Table 5.2 shows how variable the PWD in the sample were with regards to cognitive impairment and dementia-related behaviours. On average, the sample had moderate cognitive impairment. However the MMSE scores ranged from severe to normal. The most prevalent dementia-related behaviours identified by carers were related to their partner's memory (as indicated by the RMBPC scores). On average the PWD and carers both rated the PWD's quality of life within the 75th percentile, however the carers consistently rated their partner's quality of life more poorly than the individual did.

The carers were all within the normal range of cognitive functioning. Scores from their HADS and COPE scales indicated that, on average, they had reduced likelihood of anxiety, depression or carer burden. However there were individual cases where mental health and carer burden were an issue.

On average, the carers had poorer subjective ratings of their sleep compared to the PWD. This was indicated by a higher mean PSQI score (8 vs. 4) and diary-rated sleep quality scores (median = 3 vs. 2).

Table 5.3 shows the itemised statistics associated with the sleep disturbances reported within the PSQI. These indicate that the carers endorsed more symptoms related to insomnia as occurring at least once per week, whereas the PWD were more likely to endorse symptoms associated with sleep apnoea as well as disorientation and confusion in the night. The SDI scores were, in general, very low, indicating that the PWD had only mild sleep complaints recorded by carers. The itemised results in Table 5.4 indicate that the carers reported some symptoms of insomnia and daytime sleepiness concerning the PWD, as well as some disruptive behaviours at night. However, none of these symptoms were reported to be particularly frequent, severe, or distressing by the carers.

Table 5.5 shows that carers were going to bed later (median difference = 45 minutes) and getting up earlier (median difference = 32 minutes) compared to PWD. Therefore, carers had less time in bed or sleep compared to the PWD. Participants with dementia had more wake bouts and spent an average of 33 minutes more awake at night than the carers, therefore they had a lower sleep efficiency (median difference = 4.6%). The PWD also had much more variable sleep timing and quality as indicated by the greater range of bedtimes, sleep duration, and sleep efficiency compared to the carers. Twelve of the PWD slept in the daytime during the recording week, compared to eight of the carers. On average, the PWD slept approximately 40 minutes more in the daytime than the carers.

Table 5.2

Summary of Questionnaire and Diary Data for PWD and Carers at Time 1 (N=15)

	PWD					Carers				
Variable (range of scale)	N	Mean	(SD)	Median	(Range)	N	Mean	(SD)	Median	(Range)
PSQI (0-21)	15	5.1	(2.9)	4.0	(1.0-13.0)	14	7.1	(2.4)	8.0	(1.0-10.0)
SDI (0-12)	14	0.6	(1.2)	0.2	(0.0-4.2)	-	-	-	-	-
Diary rating of nights' sleep (1-7)	14	2.4	(1.3)	2.0	(1.0-6.0)	14	3.2	(1.1)	3.0	(2.0-6.0)
MMSE (30-0)	14	18.4	(5.8)	18.0	(7.0-29.0)	13	28.2	(1.6)	28.0	(25.0-30.0)
SCQ - number comorbidities	15	2.9	(2.1)	3.0	(1.0-7.0)	15	1.7	(1.5)	3.0	(0.0-5.0)
SCQ - number limiting comorbidities	15	1.3	(1.1)	1.0	(1.0-7.0)	15	0.4	(0.8)	0.0	(0.0-3.0)
RMBPC memory frequency (0-28)	15	21.0	(6.3)	24.0	(5.0-28.0)	-	-	-	-	-
RMBPC memory carer reaction (0-28)	14	7.5	(5.4)	6.0	(2.0-22.0)	-	-	-	-	-
RMBPC depression frequency (0-36)	10	7.1	(4.6)	6.5	(0.0-14.0)	-	-	-	-	-
RMBPC depression carer reaction (0-36)	9	8.5	(5.3)	4.5	(0.0-17.8)	-	-	-	-	-
RMBPC disruption frequency (0-32)	15	4.7	(4.1)	3.5	(0.0-11.0)	-	-	-	-	-
RMBPC disruption carer reaction (0-32)	15	5.2	(5.7)	3.5	(0.0-19.8)	-	-	-	-	-
RMBPC global frequency (0- 96)	10	30.7	(11.1)	28.1	(16.0-52.0)	-	-	-	-	-
RMBPC global frequency (0-96)	9	18.7	(9.3)	14.0	(8.6-35.8)	-	-	-	-	-
QOL-AD (PWD, 52-13)	14	38.5	(5.9)	39.0	(24.0-48.0)	-	-	-	-	-
QOL-AD (Carer, 52-13)	15	30.6	(5.2)	30.0	(23.0-40.0)	-	-	-	-	-
COPE positive (15-0)	-	-	-	-	-	15	10.6	(3.1)	11.0	(4.0-14.0)
COPE negative (0-18)	-	-	-	-	-	15	5.8	(2.9)	5.0	(1.0-12.0)
HADS-A (0-21)	-	-	-	-	-	15	4.8	(3.2)	4.0	(0.0-12.0)
HADS-D (0-21)	-	-	-	-	-	15	4.2	(3.2)	4.0	(0.0-9.0)

- Scale not applicable to group

Table 5.3

Pittsburgh Sleep Quality Index: Item Level Descriptive Statistics

Symptom	PWD			Carer		
	Frequency (0-3) Mean (SD)		Endorsement^	Frequency (0-3) Mean (SD)		Endorsement^
Not being able to get to sleep within 30 minutes	0.6	(1.0)	13.3%	1.2	(0.9)	40.0%
Waking up in the middle of the night or early morning	1.9	(1.2)	53.3%	2.1	(1.0)	80.0%
Having to get up to use the bathroom	2.2	(1.1)	73.3%	2.8	(0.4)	100.0%
Not being able to breath comfortably	0.3	(0.7)	13.3%	0.1	(0.2)	0.0%
Coughing or snoring loudly	0.7	(1.2)	20.0%	0.7	(1.2)	26.7%
Feeling too hot	0.3	(0.8)	6.7%	0.2	(0.5)	6.7%
Feeling too cold	0.4	(0.9)	13.3%	0.8	(1.0)	26.7%
Having bad dreams	0.6	(0.9)	13.3%	0.7	(0.9)	20.0%
Having pain	0.3	(0.8)	6.7%	0.3	(0.7)	13.3%
Using sleeping medication	0.4	(0.9)	13.3%	0.6	(1.0)	20.0%
Trouble staying awake in the daytime	0.6	(0.7)	13.3%	0.5	(0.7)	13.3%
Additional Partner-rated:						
Loud snoring	1.3	(1.3)	46.7%	0.6	(1.1)	20.0%
Long pauses between breaths when asleep	0.9	(1.3)	26.7%	0.1	(0.3)	20.0%
Legs twitching or jerking when asleep	0.6	(1.0)	13.3%	0.4	(0.9)	13.3%
Episodes of disorientation or confusion while asleep	1.3	(1.3)	40.0%	0.2	(0.6)	6.7%
Other restlessness	1.3	(1.3)	33.3%	1.0	(1.2)	13.3%

^ Endorsement = proportion of group rating symptom as occurring at least once per week

Table 5.4

Sleep Disorders Inventory: Item Level Descriptive Statistics

Symptom	Frequency (0-4) Mean (SD)		Severity (0-3) Mean (SD)		Distress (0-5) Mean (SD)		Endorsement^	Mod/marked severity^	Mod/extreme distress ^
Difficulty falling asleep	0.6	(1.0)	0.4	(0.5)	0.4	(0.8)	21.4%	0.0%	7.1%
Getting up during the night	0.8	(1.1)	0.7	(1.0)	0.9	(1.3)	21.4%	21.4%	14.3%
Wandering, pacing, or getting involved in inappropriate activities at night	0.3	(0.8)	0.3	(0.7)	0.5	(1.2)	7.1%	14.3%	14.3%
Awakening others during the night	0.9	(1.1)	0.7	(1.0)	0.8	(1.3)	28.6%	21.4%	14.3%
Awakening at night, dressing, and planning to go out, thinking that it is morning and time to start the day	0.1	(0.3)	0.2	(0.6)	0.3	(0.8)	0.0%	7.1%	7.1%
Awakening too early in the morning	0.4	(0.9)	0.2	(0.8)	0.3	(1.0)	14.3%	7.1%	7.1%
Sleeping excessively in the day	1.3	(1.3)	0.9	(1.0)	0.5	(0.7)	42.9%	28.6%	0.0%
Any other night time behaviours that are bothersome	0.5	(0.7)	0.4	(0.5)	0.5	(0.7)	14.3%	0.0%	0.0%

^ Endorsement = proportion of group rating symptom as occurring at least once per week; Mod-marked severity = proportion of group reporting moderate or marked severity; Mod-extreme distress = proportion of group expressing moderate to extreme distress.

Table 5.5

Summary of Actigraphy Data for all PWD (n=15) and Carers (n=14) at Time 1

	PWD				Carers			
Variable	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	21:40		22:00	(19:00-23:20)	20:53		22:45	(23:30-00:23)
Risetime	07:37		07:35	(06:46-08:30)	07:09		07:03	(05:58-08:20)
Time in bed, minutes	596.9	(56.7)	600.0	(495.0-715.0)	507.7	(40.9)	510.3	(422.0-587.0)
Total sleep per night, minutes	487.7	(75.9)	482.0	(332.0-637.5)	442.5	(49.2)	449.0	(354.0-532.0)
Sleep efficiency (% sleep night)	82.7	(10.6)	85.0	(61.3-98.4)	88.0	(5.4)	89.6	(72.5-93.7)
Wake time night, minutes	105.5	(68.4)	86.0	(10.0-247.0)	60.6	(27.9)	53.0	(34.0-141.0)
Number of night awakenings	29.9	(16.5)	25.4	(3.0-63.0)	23.0	(5.5)	22.0	(14.0-33.0)
Total sleep per day, minutes	42.0	(24.5)	47.0	(0.0-78.0)	18.6	(25.5)	7.0	(0.0-80.0)

5.2.3 Questionnaire Data by Completion Status

The questionnaire results from each PWD and carer at Time 1 are shown in *Figure 5.1 -Figure 5.8*. These enable visual comparisons between participants who completed the full trial (IDs 1-9) with those who withdrew (IDs 10-15), as well as situating the individual case studies presented in section 5.3 and Appendix 19. Where standardised thresholds are available, these are marked on the figures. Otherwise the median score for group is marked. Note figures concerning RMBPC scores are presented for the memory and disruptive behaviour scales only, due to the large amount of missing data within the depression and global scales.

The PWD who withdrew appeared to be of higher cognitive functioning (median = 20.5, range = 13.0-29.0) compared to those who completed (median = 15.5, range = 7.0-27.0, *Figure 5.1*). However, carers' ratings on the RMBPC showed that the PWD who withdrew had more frequent disruptive behaviours (median = 6.3, range = 0.0-10.5) compared to those who completed (median = 3.0, range = 0-11). Those who withdrew also had a greater reaction rating of these behaviours (median= 5.8, range = 0-19.8) compared to those who completed (median = 3.0, range = 0-12, *Figure 5.2*). Conversely, those who completed reported more problems with the PWD's memory (median = 24.0, range = 5.0-27.0) compared to those who withdrew (median = 21.0, range = 14.0-28.0), and had greater reaction to these memory-related symptoms (median = 6.5, range = 2.3-22.0 vs. 5.0, range = 2.0-12.0, *Figure 5.3*). Quality of life of the PWD was rated similarly between those who completed and those who withdrew, as rated by the PWD (median = 39.5, range = 24.0-45.0, vs. median = 39.0, range 34.0-48.0 respectively), and as rated by the carer (median = 30.0, range =23.0-40.0, vs. median = 29.9, range = 23.0-35.0 respectively, *Figure 5.4*).

Carers had similar self-rated symptoms of anxiety (median = 4.0 for both groups) and depression (median = 3.5 vs. 4.0, as indicated by the HADS *Figure 5.5*). Carers who withdrew and carers who completed both had low COPE negative scores (median= 5, range = 4-9, vs. median = 6, range = 1-12 respectively). However, scores concerning the positive aspects of coping appeared to be slightly greater

within the group who withdrew (median = 12.5, range = 6-14) compared to those who completed (median = 10, range = 4-14, *Figure 5.6*).

The subjective sleep results for the PWD were similarly distributed. The median PSQI scores of those who withdrew was 4.5 (range = 3.0-13.0) versus 4.0 for those who completed (range =1.0-8.0, *Figure 5.7*). The SDI scores were very low for both those who withdrew (median = 0.1, range = 0.0-2.0) and those who completed (median = 0.2, range = 0.0-4.2, *Figure 5.8*). Carers who withdrew also had similar PSQI scores to those who completed (median=8.0, range = 1-9, vs. 7.5, range = 4-10 respectively, *Figure 5.7*).

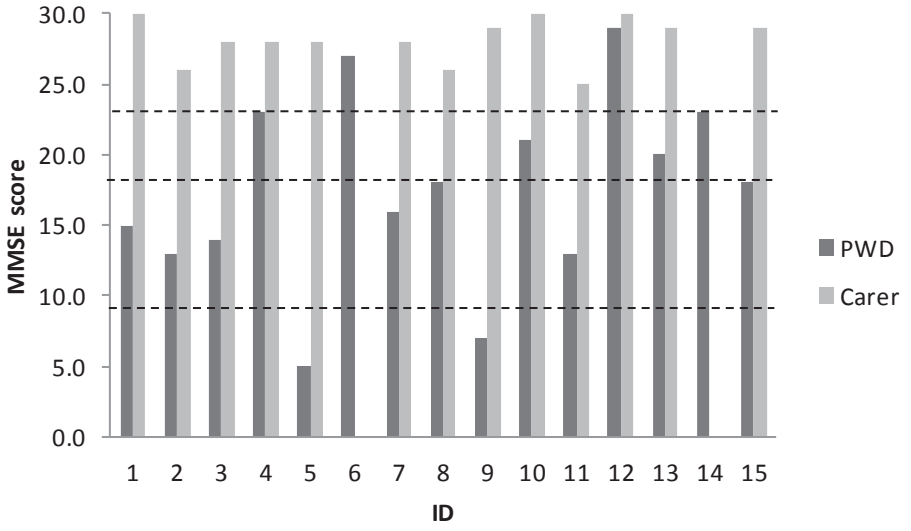


Figure 5.1. Global MMSE scores of PWD and carers who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data).

Dashed lines represent cut off scores for normal cognitive functioning (24-30), mild cognitive impairment (19-23), moderate cognitive impairment (10-18), and severe cognitive impairment (<9).Note Carer 6's score is missing.

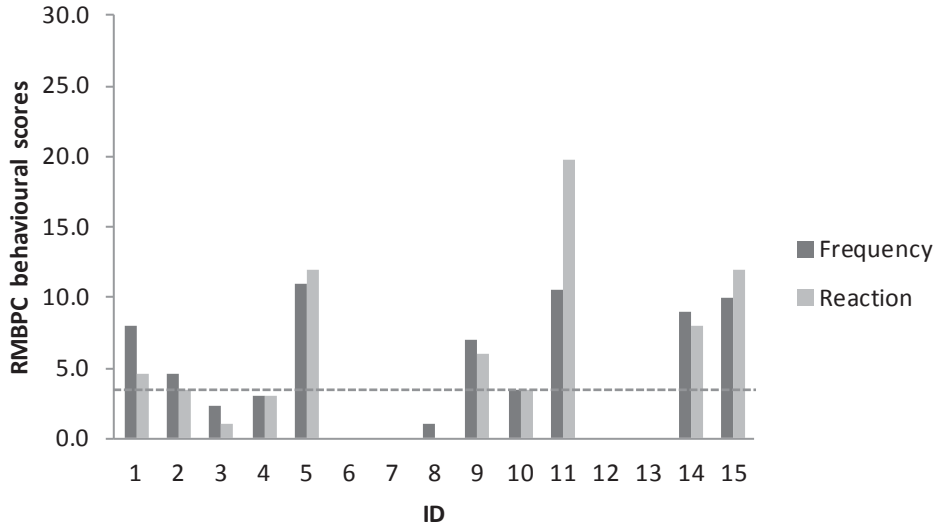


Figure 5.2. RMBPC-disruptive behaviour scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15).

Split by frequency and carers' reaction (Time 1 data). Higher scores indicate increased frequency or reaction. Group median frequency and reaction = 3.5 (dotted line). Note Carer 6's reaction score is missing.

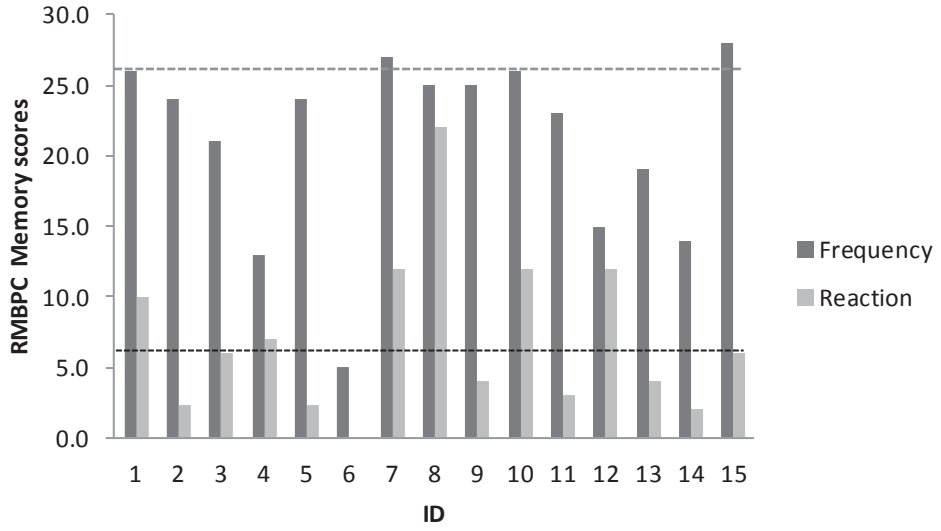


Figure 5.3. RMBPC-memory scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not IDs 10-15).

Split by frequency and carers' reaction (Time 1 data). Higher scores indicate increased frequency or reaction. Group median frequency = 24.0, reaction = 6.0 (dotted lines).Note Carer 6's reaction score is missing.

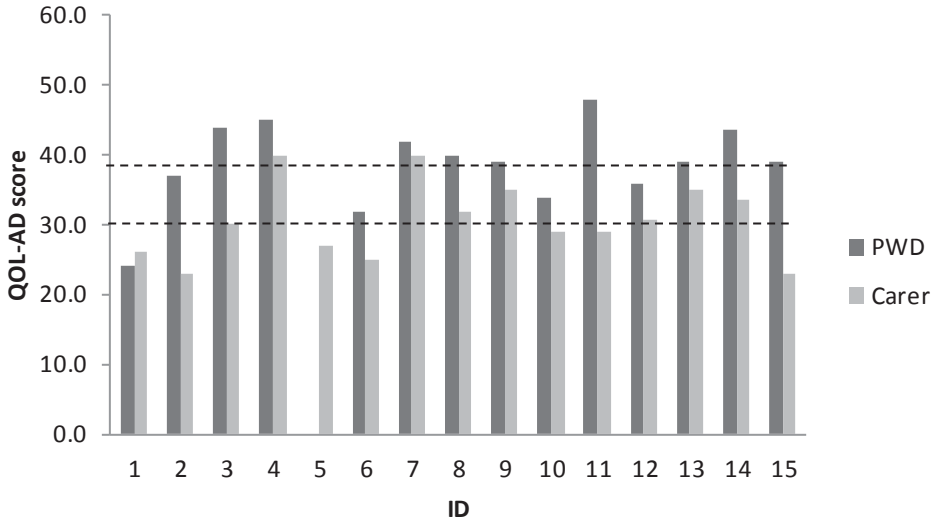


Figure 5.4. QOL-AD scores concerning the PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15).

Split by PWD completed and carer completed questionnaires (Time 1 data). Higher scores indicate greater rating of quality of life, group median for the PWD rated QOL-AD = 39. group median for the carer rated QOL-AD = 30 (dotted lines). Note PWD 5's score is missing.

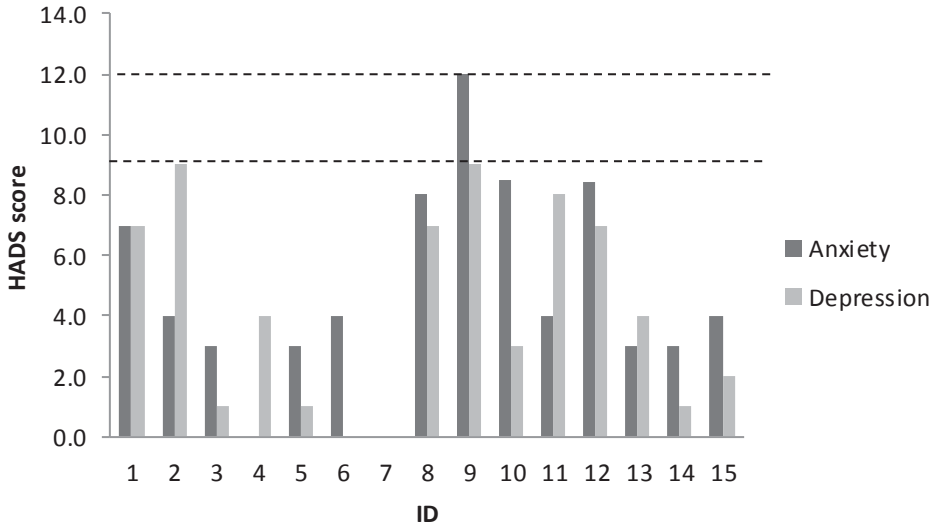


Figure 5.5. HADS anxiety and depression scores of carers who completed the full trial (IDs 1-9) and those who did not (IDs 10-14, Time 1 data).

Dashed lines indicate cut off scores for risk of anxiety or depression (0-7 normal, 8-10 borderline, and 11-21 heighted).

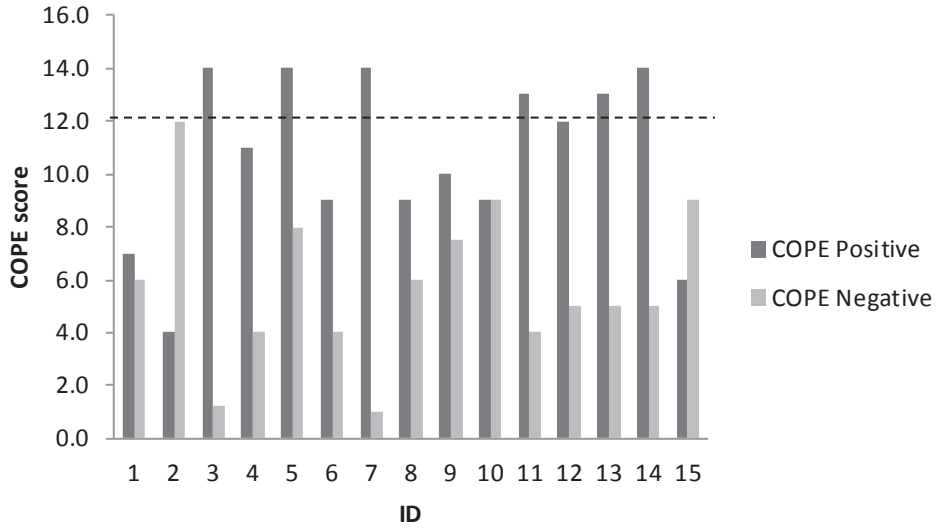


Figure 5.6. Positive and negative COPE scores of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Scores below 12 indicate poor coping on the positive scale, scores above 12 indicate poor coping on the negative scale (dashed line).

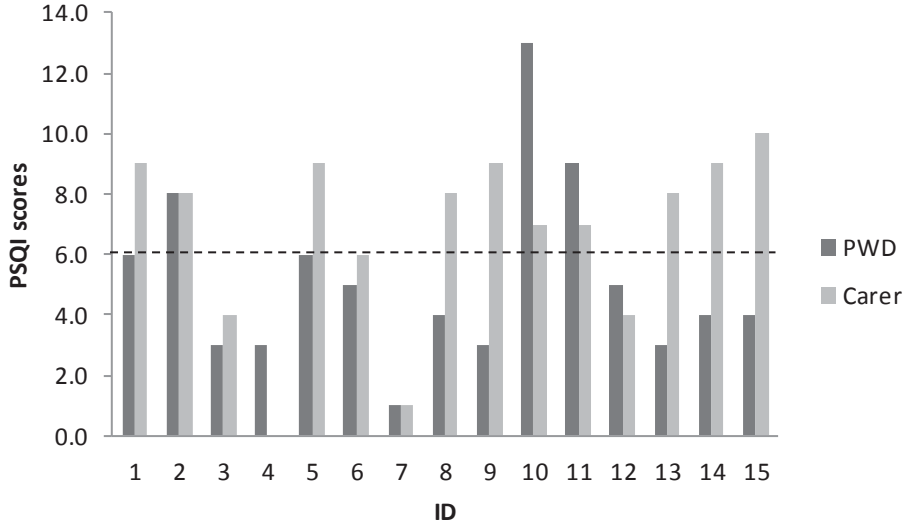


Figure 5.7. Global PSQI scores of PWD and carers who completed the full trial (IDs1-9) and those who did not (IDs 10-15, Time 1 data).

Higher scores indicate increased sleep disturbance, dashed line represents the cut off score of 5 for defining problem sleep. Note Carer 4's score is missing.

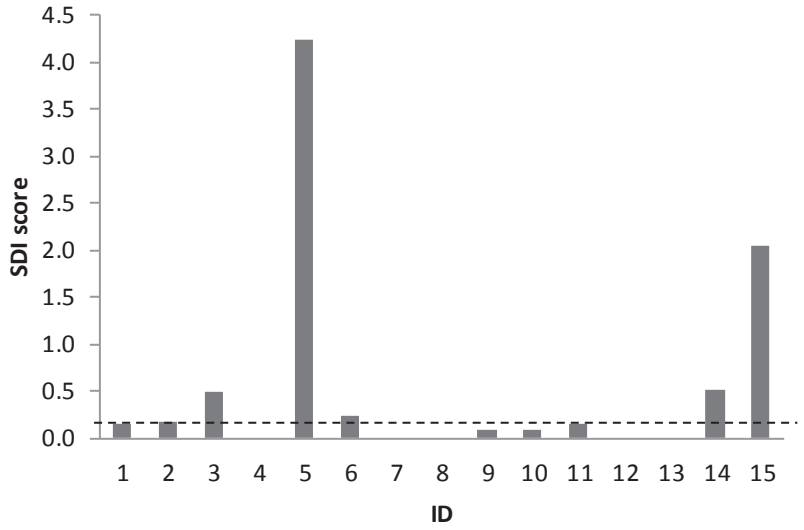


Figure 5.8. Global SDI scores of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data).

Higher scores indicate greater sleep disturbance, group median = 0.2 (dotted line). Note PWD 8's SDI score is missing.

5.2.4 Actigraphy Data by Completion Status

The median actigraphy results concerning individual PWD and carers are shown in *Figure 5.9* - *Figure 5.16*. Median scores for the group are marked on each graph (dotted line). Note carer 6 has missing actigraphy data due to refusal to use the Actiwatch.

The PWD who withdrew had slightly less sleep at night compared to those who completed (median = 7.9 hours, range = 5.5-10.6 vs. 8.1 hours, range = 6.7-9.8, Figure 5.9). On average this group had eight less wake bouts per night (median = 19.0, range = 3.0-61.0 vs. 27.5, range =17.5-63.0, *Figure 5.11*), their median sleep efficiency was just 1.5% less (median = 83.5%, range = 61.3-98.4) than the PWD who completed (median = 85.0%, range = 62.0-95.9, *Figure 5.13*) and they spent an average of 5.5 minutes less time asleep during the daytime (median = 46.0 minutes, range = 0-63.5 vs.51.5, range = 0-78, *Figure 5.15*).

The carers who withdrew were having almost 50 minutes less sleep at night compared to those who completed the study (median =7.1 hours, range = 5.9-7.5 vs. 7.9, range = 6.1-8.9 *Figure 5.10*).Both groups had high median sleep efficiencies (87.9% and 90.0% respectively). However, carers

who withdrew were more likely to sleep during the day than those who completed the study (median = 11.0 minutes, range = 0-60.0 vs. 0.0, range = 0.0-80.0 *Figure 5.16*.)

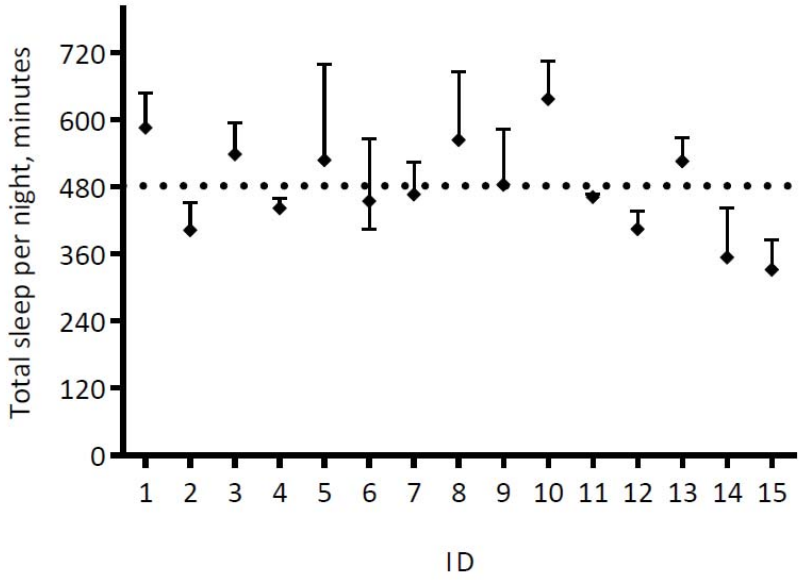


Figure 5.9. Total sleep time at night of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data).

Median and range (group median = 482 minutes, dotted line).

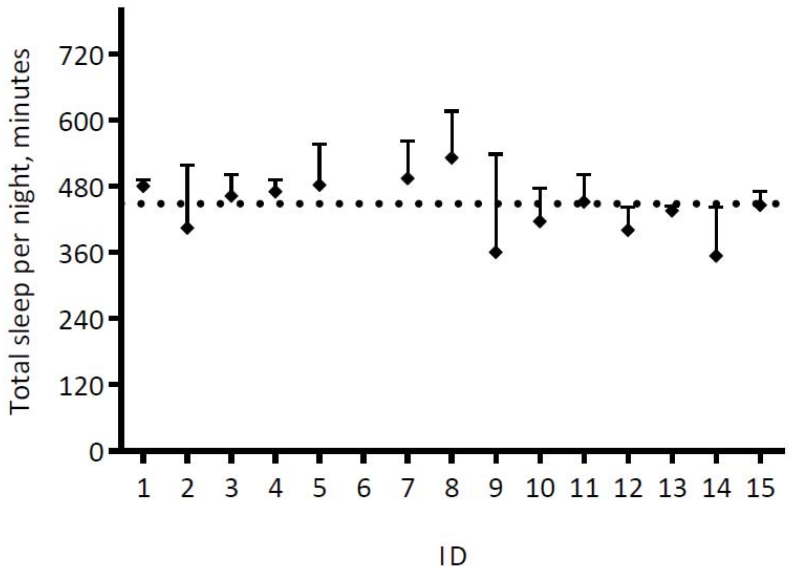


Figure 5.10. Total sleep time at night of carers who completed the full trial (IDs 1-9) and those who did not (IDs10-15, Time 1 data).

Median and range (group median = 449.0 minutes, dotted line).

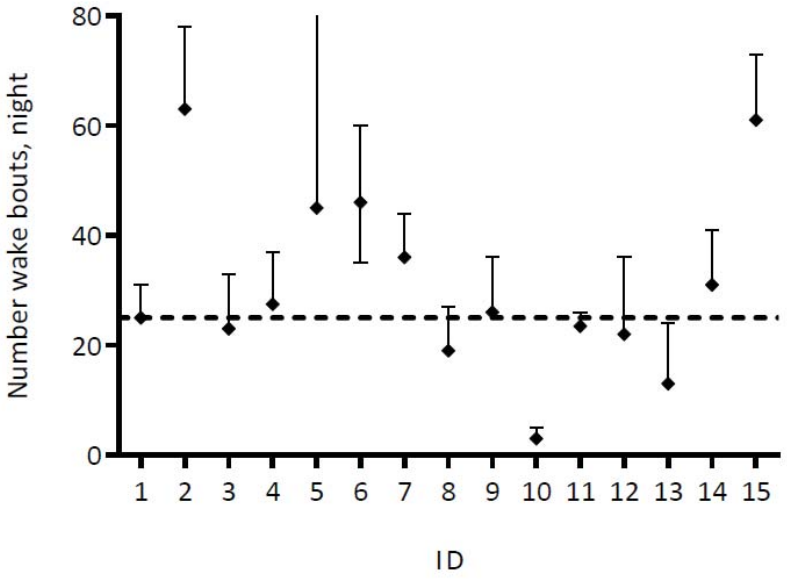


Figure 5.11. Number of wake bouts at night of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and range, group median = 25 (dotted line).

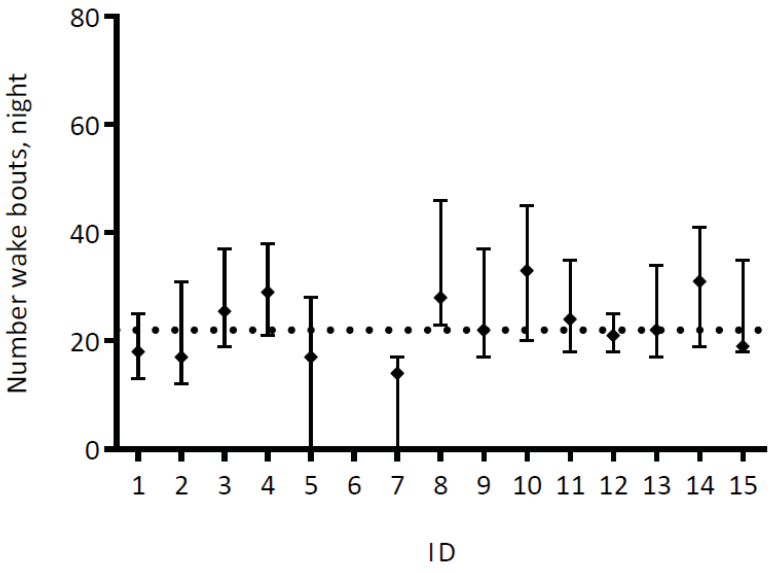


Figure 5.12. Number of wake bouts at night of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and range, group median = 22 (dotted line).

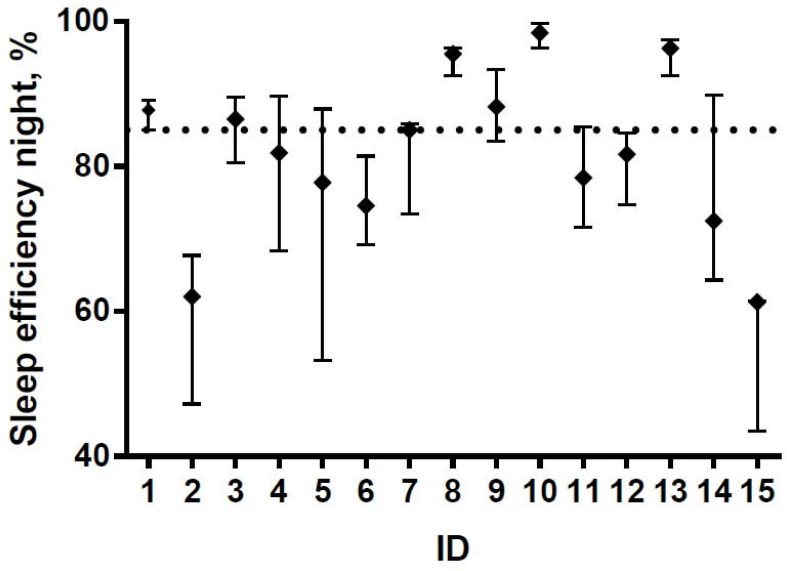


Figure 5.13. Sleep efficiency of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and range, group median = 85% (dotted line).

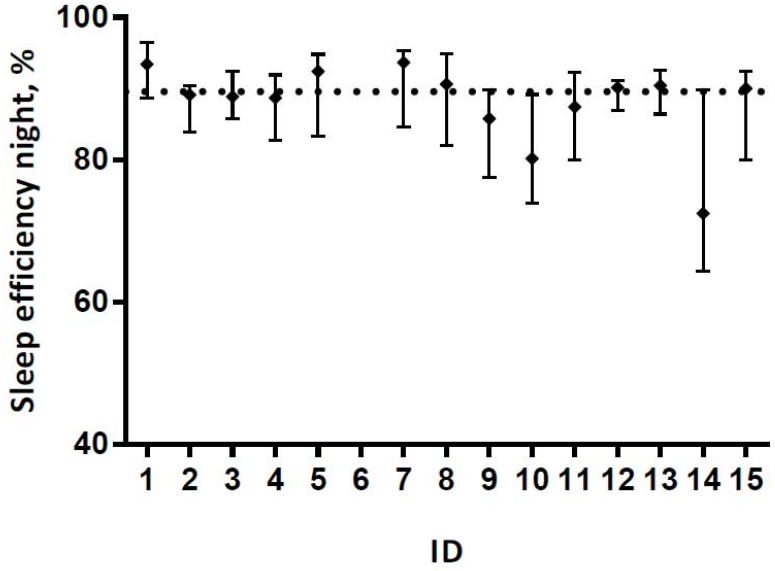


Figure 5.14. Sleep efficiency of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and range, group median = 89.6% (dotted line).

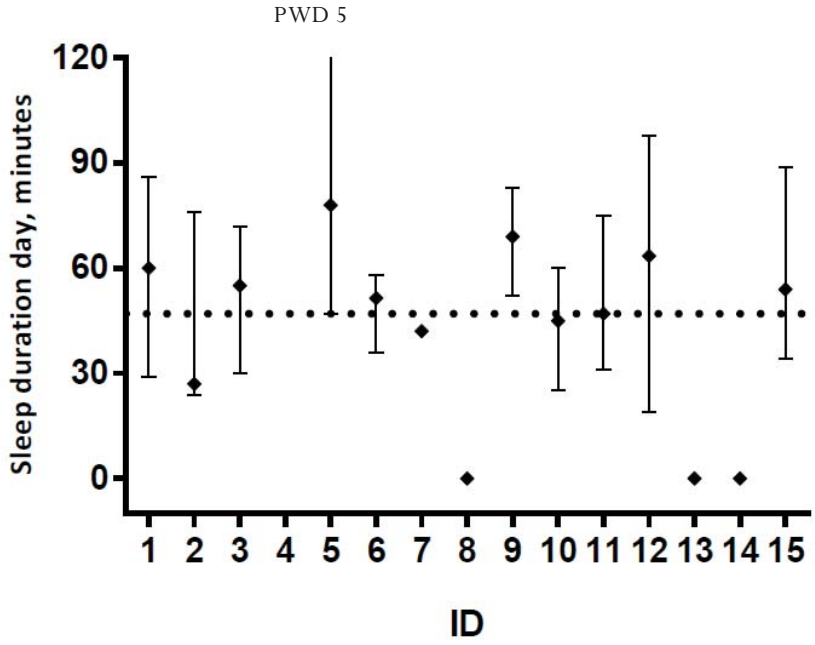


Figure 5.15. Total sleep duration in the daytime of PWD who completed the full trial and those who did not (Time 1 data).

Median and range, group median = 47 minutes (dotted line). Note PWD 4's data is missing due to unreliable sleep diaries during the day.

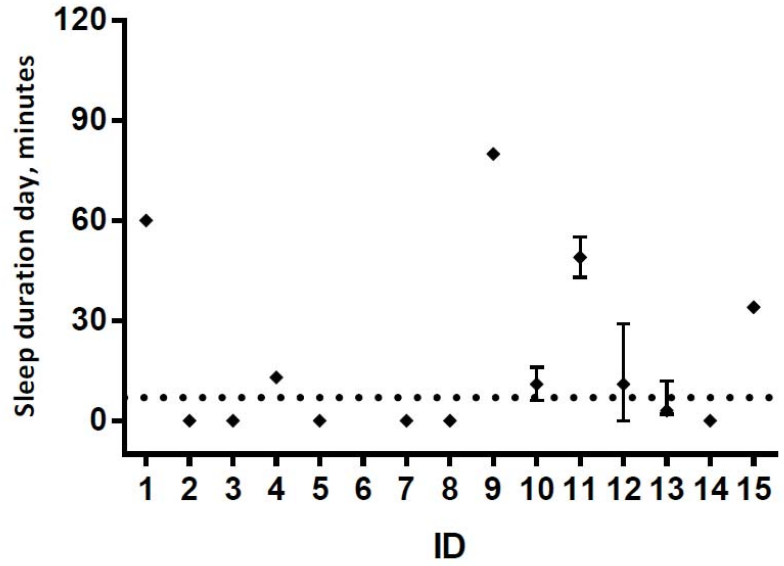


Figure 5.16. Total sleep duration in the daytime of carers who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and range, group median = 7 minutes (dotted line).

5.2.5 Light and Activity, Time 1

Table 5.6 shows the descriptive data for the amount of ambient light and activity that the PWD were receiving at Time 1, as recorded by their Actiwatches during the timeframes used for the subsequent intervention. *Figure 5.17* shows the median ambient light exposure for each PWD between 9-11am at Time 1. The PWD who withdrew were exposed to less intense light during this time (median = 2,805.3 lux, range = 1,345.3-13,492.8) compared to those who completed (median =2,960.8 lux, range = 473.6-18,717.0). *Figure 5.18* shows the median activity counts per minute for each PWD between 11am-2pm at Time 1. This shows that the PWD who withdrew had slightly lower records of activity between 11am-2pm (median = 101.3 counts, range = 14.8-365.2) compared to those who completed (135.0 counts, range = 37.2-348.3).

Table 5.6

Summary of Light Exposure (9-11am) and Physical Activity (11am-2pm) for all PWD at Time 1

	Mean	(SD)	Median	(Range)
Total light exposure (lux)	4,948.7	(5,015.2)	2,960.8	(473.0-18,717.0)
Average activity (counts/minute)	141.1	(99.7)	129.6	(14.8-365.2)

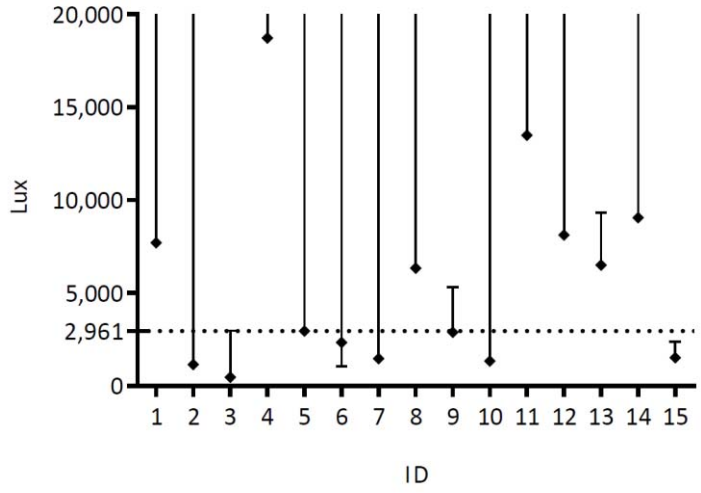


Figure 5.17. Total light exposure recorded between 9-11am of PWD who completed the full trial (IDs 1-9) and those who did not (IDs 10-15, Time 1 data).

Median and range, group median = 2,960.8 (dotted line).

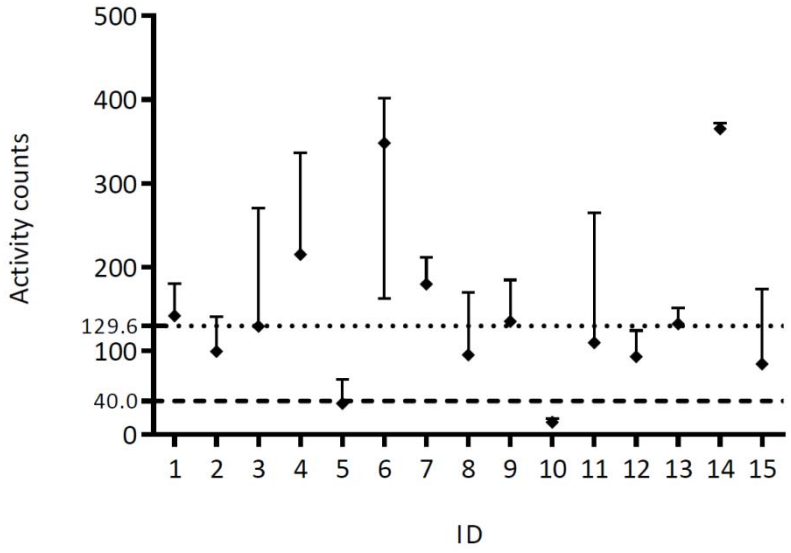


Figure 5.18. Average activity counts per minute between 11am-2pm of PWD who completed the full trial (ID's 1-9) and those who did not (ID's 10-15, Time 1 data).

Median and ranges, group median = 129.6 (dotted line), counts below 40 are defined as 'sleep' (dashed line).

5.2.6 Participant Feedback

All 15 pairs sent back a feedback form after completing or withdrawing from the study. These were typically completed by the carer, except for pair 12 where the PWD also completed a form. All 16 people who completed forms agreed that the information provided by the researcher prior to the study gave them "a full picture" of what the study entailed. All but one pair agreed that they were satisfied with the way the researcher answered any queries they had concerning the study (the other pair did not respond to the question). Tables 5.7 and 5.8 give the frequencies of how participants answered the questions concerning the data collection and intervention aspects of the protocol.

Of the nine pairs who completed the trial, seven said that they would recommend the interventions to others. The other two said they maybe would. Participants had space to provide more detailed comments regarding data collection and the interventions as well as the study in general or reasons for withdrawal. This qualitative feedback is presented within individual case studies.

Table 5.7

Frequencies and Percentages of How Participants Rated Data Collection (n=16)

	Easy	OK	Difficult
Actiwatch use	10 (62%)	6 (38%)	0
Diary use	10 (62%)	5 (31%)	1 (6%)
Questionnaire use	9 (56%)	7 (44%)	0

Table 5.8

Frequencies and Percentages of How Participants Rated Aspects of the Intervention (n=9)

	Easy	OK	Difficult	Missing
Light box	3 (33%)	3 (33%)	0	3 (33%)
Exercise DVD	2 (22%)	4 (44%)	2 (22%)	1 (11%)
Sleep support handbook	7 (77%)	2 (22%)	1 (11%)	0

5.3 Summary of Pairs who withdrew

Case studies concerning the six pairs who withdrew from the trial can be found in Appendix 19. These illustrate that there were two types who withdrew: Those who had early-stage dementia, with minor sleep problems, and who lost interest or were confused by the study protocol (Pairs 12, 13, and 14); and those who had more advanced stages of dementia, with substantial sleep problems, but were admitted to hospital or a care facility within the trial period (Pairs 10, 11, and 15). Table 5.9 summarises the factors affecting compliance and withdrawal amongst Pairs 10-15.

Pair 14 highlighted some particular challenges concerning conducting research involving a PWD who experiences paranoia. The presence of the researcher, the protocol, and associated equipment, were all treated as suspicious by Georgina. Her daughter, Lisa, attempted to support Georgina with the interventions. Although she consented to taking part, Georgina resisted using the Actiwatch or light box once the researcher had left. Lisa reasoned that the research experience may have felt clinical or threatening to her mother.

Carers of the PWD with more severe dementia and sleep problems expressed disappointment at not being able to continue. These carers, particularly carer 15, were longing for a solution for their partner's sleep problems. They were potentially ideal participants for the trial. However, the rapid progression of dementia meant that the opportunity for them to trial the interventions was hampered.

Table 5.9

Factors Affecting Compliance and Study Withdrawal: Case Studies 10-15

Factors given in feedback	Pair/s affected
Minor or no sleep problems:	
PWD slept well so saw little point in continuing the trial	12 & 13
PWD found completing the diary challenging	12
PWD did not realise how much work and time the study would require	12
Carer admitted to hospital	13
Already doing a lot of the intervention-type activities	13
PWD unaware/hostile surrounding diagnosis	14
Paranoia concerning the presence of the researcher and equipment	14
PWD refused to use light box	14
PWD felt challenged by data collection process due to clinical feeling	14
PWD due to move out of family home	14
PWD went into short-term respite and lost interest in the study	12
Substantial sleep problems:	
Carer concerned whether PWD could physically do the exercises	11
PWD taking Actiwatch off	11 & 14
Substantial changes to PWDs' medications	11 & 15
Challenging to keep PWD sat in front of light box	11
Found the exercise DVD difficult	11
Carer tired/overwhelmed by managing study and supporting PWD	11 &14
PWD admitted to hospital	11 & 15
PWD moved into care facility	10 & 15

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5.4 Case Studies of Participants who Completed the Full Study

The format for presenting the case studies is shown in *Figure 5.19*. This uniform style is maintained in order to provide a consistent and easily accessible set of cases.

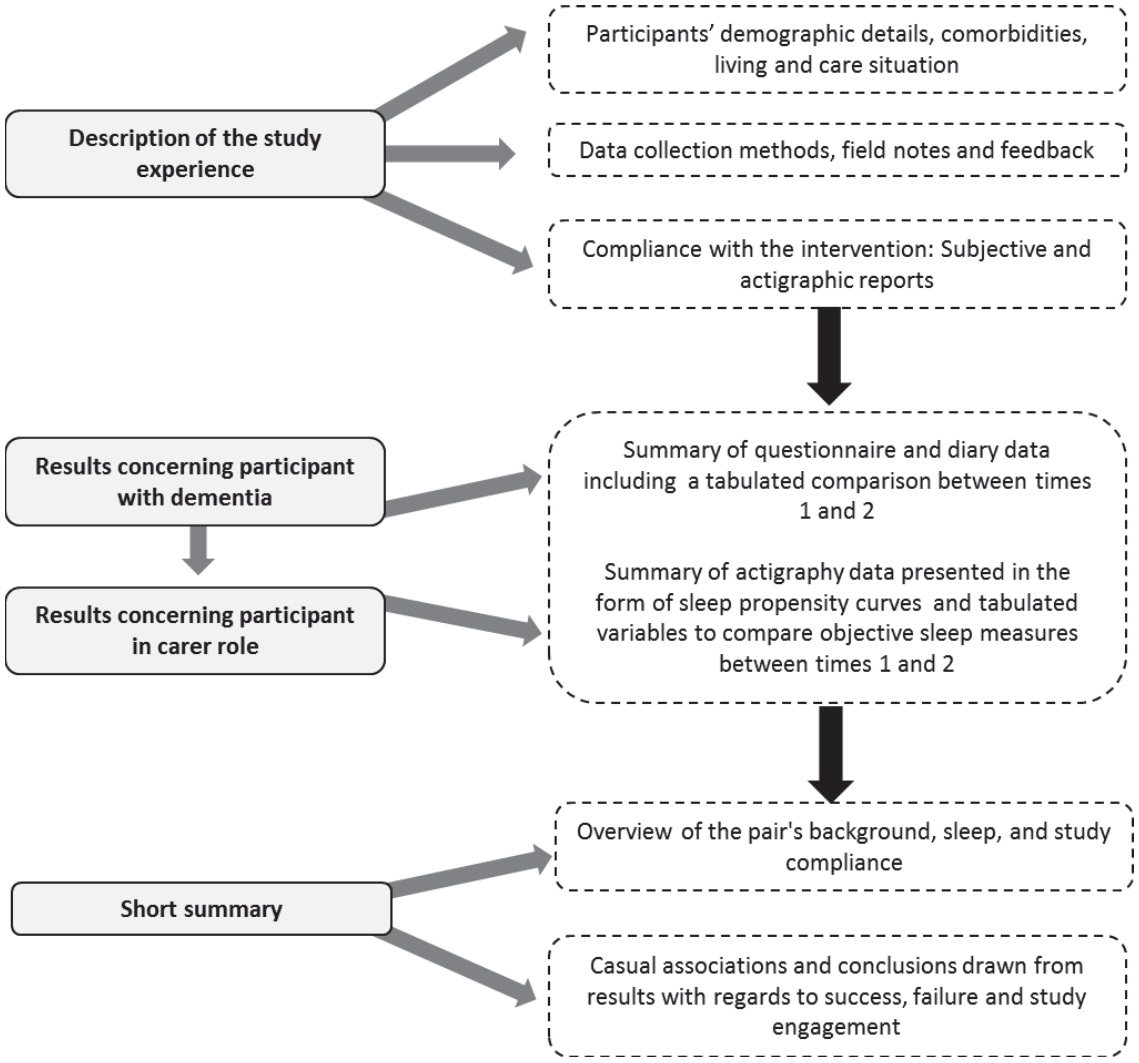

Figure 5.19. Flow chart illustrating the presentation of case studies.

5.4.1 Pair 1 (Liam and Sophie)

Description of study experience

Participants.

Pair 1 consisted of a husband and wife who had taken part in sleep-related research in the past and appeared to have a genuine interest in the topic and outcomes. Liam was a 73-year-old man with VaD, which was diagnosed two years prior to the study. He had an MMSE score of 15, indicating moderate cognitive impairment. His comorbidities included heart disease, anaemia or blood disease, depression, and arteriosclerosis. He received treatment for all of these conditions, however his depression and arteriosclerosis still limited his activities. Many of his medications had dizziness or somnolence as possible side effects, and one also had insomnia and nightmares listed (see Appendix 20).

Sophie was aged 73 years at the time of the study. She had been providing dementia-related care for her husband for three years. She had support from a day care centre which her husband attended four days a week. Otherwise she was providing care all day and night. Her comorbidities were high blood pressure and migraine. These were both treated however the migraines still limited her activities. Some of the medications Sophie was taking had dizziness or somnolence listed as possible side effects (Appendix 20), she commented that the medications she used for her migraines made her sleepy.

Data collection and intervention compliance.

Liam signed the consent form himself. Questionnaires were completed by Liam answering verbally, with the researcher clarifying the questions and Sophie completing the paperwork. They both noted that Liam was sensitive to bright lights. He had recently had cataract surgery and was taking a diuretic which had photosensitivity as a side effect. Therefore he was prescribed timed natural light rather than the light box for the intervention period. Both Liam and Sophie commented that he received a reasonable amount of light and exercise already at the day care centre.

No problems were reported during the first weeks of the intervention period. Sophie regularly sent Liam on walks and there had been plenty of sunshine. She noted that he seemed unfazed by the physical activity regime. By the fourth week of the intervention Sophie commented that Liam was

sometimes reluctant to go for his walks unless made aware that he was doing it for the researcher. Liam only attempted the exercise DVD once or twice and didn't enjoy it as much as his walks. This was because some parts of the programme involved coordinating arms and legs which he found difficult and frustrating. Cold weather including snow in the final week of their study meant that day care and outdoor activities were somewhat disrupted. This may have affected their compliance with the intervention.

In the diaries, Sophie recorded that Liam had bright light exposure on 30 out of the 35 days. However, only seven of these instances were within the 9-11am timeframe. Likewise he had 27 bouts of physical activity noted, but just 10 of these were within the prescribed 11am-2pm timeframe. This may be due to the established routines Liam was following at his day care centre.

Table 5.10 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. Liam's median light exposure at between 9-11am was within the group's 75th percentile at Time 1. This reduced to within the 50th percentile at Time 2.

Table 5.10

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 1

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux)	7,707.2	(466.0-22,314.1)	5,984.3	(426.5-414,937.7)	-1,722.9
Average activity (counts/minute)	141.9	(40.0-180.0)	129.8	(47.4-434.1)	-12.1

Completion of the trial.

At completion of the trial, Sophie thought that Liam was going to bed a little earlier and was less interested in the television in the evening compared to Time 1. However this was not considered problematic as he was not getting up any earlier in the morning. Liam had been having a lot of dreams during the study period, some frustrating and some nightmares. He and Sophie were interested in the links between sleep, memory and dreaming. They both noted how Liam's memory was getting noticeably worse, he also struggled more with language which, he commented, was frustrating. He spoke of other people he knew with dementia who lacked any communication skills, which worried him.

In the feedback form, Sophie noted that Liam found wearing the Actiwatch a little uncomfortable and also confusing as it was similar to his wristwatch. The exercise regime was not always easy for them. This was because it required Liam's cooperation, and there wasn't always time to fit it into their schedule. She also mentioned that the sleep support handbook was easy to use, but they were already aware of a lot of the information and were practicing a lot of the tips.

Results for PWD 1

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.11. Note, Pair 1 was missing part of the RMBPC and therefore depression and global scores were unavailable from this scale.

Liam slept in a separate room from Sophie. At Time 1 he rated his sleep as "very good". His PSQI score indicated a mild sleep disturbance. This was mostly related to waking in the night, bad dreams and getting up to use the bathroom (at least once or twice per week). He also reported occasional (less than once per week) problems getting to sleep within 30 minutes, and some daytime sleepiness. Sophie reported that Liam had occasional episodes of disorientation or confusion (occurring less than once a week), as well as waking to external noises or their cat. On the SDI, Sophie reiterated reports of Liam's trouble getting to sleep, waking in the night, and sleeping during the day. However these behaviours were not very frequent or severe, hence the low score.

At Time 2, Liam's sleep quality rating was poorer than Time 1 ("fairly good"). But his average diary rating of sleep quality remained unchanged. He and Sophie noted an increase in episodes of disorientation or confusion during sleep, as well as legs twitching and worry keeping him awake. Liam's PSQI score reduced to within normal range, and Sophie's SDI rating remained low. However, this did increase due to Liam getting up more in the night and causing greater disturbance to her.

Changes in the MMSE and QOL-AD scores between Times 1 and 2 indicate an increase of dementia-related symptoms during the study period. However there was some improvement in the frequency of memory-related symptoms at Time 2 (as recorded on the RMBPC). Sophie noted in her questionnaire that she felt that the reduced quality of life ratings were more likely related to Liam's deteriorating abilities in the daytime rather than his quantity or quality of sleep.

Table 5.11

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 1

Variable	PWD T1	PWD T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.5	1.0	+0.5
Night sleep (hrs)	8.0	9.0	+1.0
PSQI (0-21)	6.0	5.0	+1.0
SDI (0-12)	0.2	0.6	-0.4
Rating of nights' sleep (1-7 median, range)	3.0 (2-6)	3.0(2-4)	No change
MMSE (30-0)	15.0	11.0	-4.0
QOL-AD PWD (52-13)	24.0	18.0	-6.0
QOL-AD carer (52-13)	26.0	22.0	-4.0
RMBPC Memory frequency (0-28)	26.0	23.0	+3.0
RMBPC Memory carer reaction (0-28)	10.0	12.0	-2.0
RMBPC disruption frequency (0-32)	8.0*	6.9*	-1.1
RMBPC disruption carer reaction (0-32)	4.6*	4.6*	No change

* = Item imputation used due to missing data from <20% of the component scores.

Actigraphy data.

There were seven complete days of actigraphy data available from Liam at both Times 1 and 2 (Appendix 21, *Figure A.1*). The sleep propensity curves using these data show little change to the timing of his night time sleep (*Figure 5.20*). However, at Time 2 there appears to be less napping during the morning, with a more consolidated daytime nap after midday. Table 5.12 compares Liam's actigraphic sleep variables between Times 1 and 2. Liam had a good sleep efficiency (over 85%) and was also getting at least 9.5 hours sleep at both Times 1 and 2.

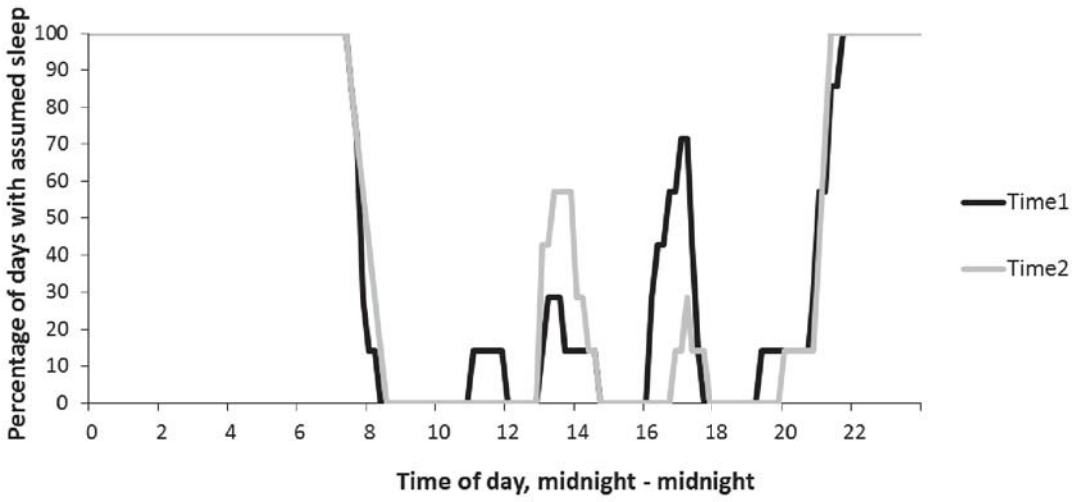

Figure 5.20. Sleep timing of PWD 1 at Time 1 and Time 2 (7 days).

Results for Carer 1

Questionnaire and diary data.

Questionnaire data concerning Sophie's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.13. At Time 1, Sophie rated her sleep as "fairly good". However her PSQI score indicated poor quality sleep. This was mostly related to not being able to get to sleep within 30 minutes, waking in the night, and getting up to use the bathroom (at least one or twice per week). Less often, her sleep was disrupted by bad dreams, feeling too hot, or breathing difficulties (less than once per week). She would take half a sleeping tablet once or twice a week and when suffering from migraines.

At Time 2, Sophie still rated her sleep as "fairly good". Her average diary rating of her night time sleep improved a little. Her PSQI score remained raised. At Time 2 she noted having trouble sleeping due to providing more night time support to Liam. Sophie's scores on the HADS increased to above the threshold, indicating a greater likelihood of anxiety and depression at this time. She also had increased likelihood of carer-related burden (as indicated by her scores on the positive COPE index). However she marked that she "often" felt well supported in her caregiving role at both times.

Table 5.12

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 1

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	20:42		21:00	(19:15-21:00)	20:52		21:00	(20:00 - 21:05)	No change
Risetime	7:54		8:00	(7:30-8:15)	8:04		8:15	(7:30-8:30)	+15.0
Time in bed night, minutes	672.0	(28.9)	666.0	(645.0-735.0)	671.6	(33.0)	676.0	(630.0-735.0)	+10.0
Total sleep per night, minutes	587.7	(29.2)	586.9	(559.0-648.0)	604.1	(26.0)	601.0	(576.0-667.0)	+14.1
Sleep efficiency (% sleep night)	87.4	(1.3)	87.6	(85.0-89.1)	90.0	(1.5)	90.0	(87.1 - 93.0)	+2.4
Wake time night, minutes	84.3	(8.2)	85.1	(72.0-99.0)	67.4	(11.7)	68.0	(44.0-89.0)	-17.1
Number of night awakenings	25.9	(4.2)	25.4	(18.0-31.0)	22.1	(4.0)	22.0	(14.0 - 27.0)	-3.4
Total sleep per day, minutes	62.8	(17.6)	60.0	(29.0-86.0)	56.8	(23.2)	59.5	(15.0-99.0)	-0.5

Table 5.13

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 1

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.3	0.6	+0.3
Night sleep (hrs)	7.0	7.0	No change
PSQI (0-21)	9.0	8.0	+1.0
Rating of nights' sleep (1-7 median, range)	3.0(1-6)	2.0 (1-4)	+1.0
MMSE (30-0)	30.0	30.0	No change
HADS-A (0-21)	7.0	9.0	-2.0
HADS-D (0-21)	7.0	8.0	-1.0
COPE positive (15-0)	7.0	5.0	-2.0
COPE negative (0-18)	6.0	8.0	-2.0

Actigraphy data.

There were seven complete days of actigraphy data available from Sophie at both Times 1 and 2 (Appendix 21, *Figure A.2*). The sleep propensity curves using these data show little change to the timing of her night time sleep (*Figure 5.21*). At Time 2 there are more days with a midday nap compared to Time1. Table 5.14 compares actigraphic sleep variables for Sophie between Times 1 and 2. Sophie had a good sleep efficiency (over 88%) and was also getting at least 7.1 hours sleep at both Times 1 and 2.

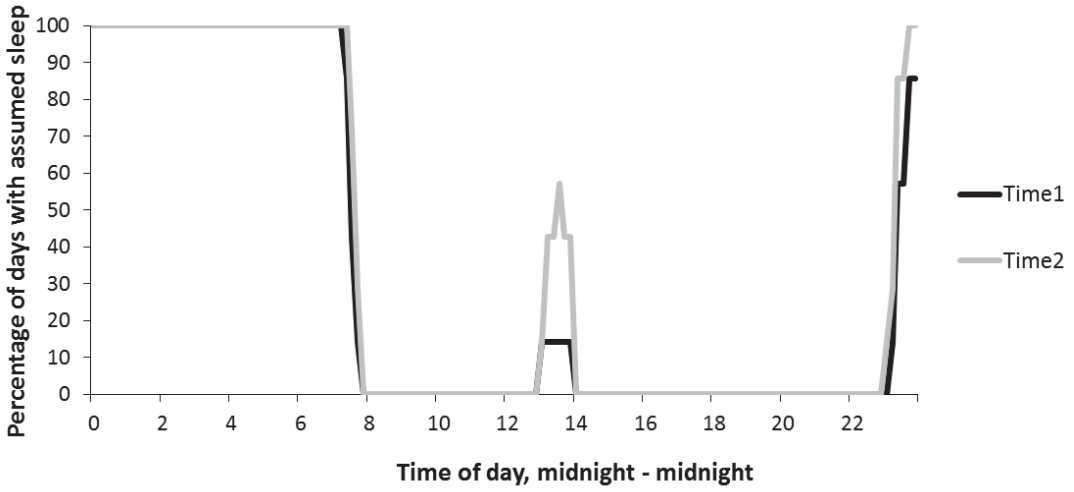

Figure 5.21. Sleep of carer 1 at Time 1 and Time 2 (7 days).

Table 5.14

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 1

Variable	Carer T1				Carer T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:12		23:10	(22:45-23:59)	23:00		23:00	(23:00-23:15)	-00:10
Risetime	7:39		7:40	(7:30-7:50)	7:43		7:45	(7:30-8:10)	+00:05
Time in bed night, minutes	507.6	(22.8)	510.0	(461.0-545.0)	519.6	(10.4)	517.0	(510.0-540.0)	+7.0
Total sleep per night, minutes	472.7	(19.4)	481.0	(425.0-492.0)	478.9	(14.8)	477.0	(449.0-499.0)	-4.0
Sleep efficiency (% sleep night)	93.2	(2.2)	93.4	(88.6-96.5)	92.2	(1.8)	92.4	(88.0-94.9)	-1.0
Wake time night, minutes	34.9	(12.1)	34.0	(18.0-62.0)	40.7	(8.9)	40	(27.0 - 61.0)	+6.0
Number of night awakenings	17.9	(3.9)	18.0	(13.0-25.0)	17.0	(2.4)	18.0	(12.0-19.0)	No change
Total sleep per day, minutes	60.0	(0.0)	60.0	(60.0-60.0)	47.5	(8.0)	47.5	(39.0-56.0)	-12.5

Short summary

Pair 1 completed the sleep study using natural light rather than the light box due to the side effects of Liam's medications and recent cataract surgery. He preferred using walking rather than the DVD as his form of exercise. Actigraphically, Liam had good sleep quantity and quality (as indicated by his high sleep efficiency and sleep duration).There were some objective improvements to Liam's sleep at Time 2 compared to Time 1, with approximately quarter of an hour more sleep at night, and less awakenings. However his and Sophie's subjective reports revealed that his sleep was not as good at Time 2 compared to Time 1. They reported an increase in dementia-related symptoms, including more confused awakenings at night, restlessness, and nightmares. Sophie's actigraphic sleep records changed very little between Times 1 and 2. Although her PSQI score and diary ratings improved by one point, she reported more disruptions related to being woken by Liam at Time 2 compared to Time 1.

The questionnaire results supported the pair's reports of dementia-related deterioration with reduced scores related to Liam's cognitive functioning and quality of life. However, Sophie reported less frequent memory-related problems at Time 2. Sophie's questionnaire results also indicated an increased risk of anxiety, depression and carer burden at Time 2 compared to Time 1 which could have been attributed to the increase in dementia-related symptoms of her husband.

5.4.2 Pair 2 (Jack and Ella)

Description of study experience

Participants.

Pair 2 consisted of a married couple who were living in a motel while their home was undergoing renovations. Jack was an 82-year-old man with VaD, which was diagnosed 11 years prior to the study. He had an MMSE score of 13, indicating moderate cognitive impairment. His comorbidities included heart and lung disease (treated) as well as a colostomy and urinary incontinence. The heart disease and incontinence were considered to limit his activities. One of the medications he was taking had somnolence listed as a possible side effect (see Appendix 20).

Ella was aged 74 years at the time of the study. She had been providing dementia-related care for her husband for over 11 years. She had support from a relief carer for about eight hours per week (split between two days). Apart from this, she was providing care all day and night. Ella had an arthritic condition that she was being treated for but that limited her activities. One of the medications she was taking had somnolence listed as a possible side effect (see Appendix 20). She noted that she felt fortunate to have had a rich life, full of career, travel and family. She felt she had the life experience to manage caring for her husband. She noted the only challenge for her had been to try and make each day special and to care for her husband in a way that did not make him feel like a burden.

Data collection and intervention compliance.

Ella gave consent on behalf of Jack, using the statement by partner form, as she considered him too impaired to fully understand the nature of the research. Questionnaires were completed by Jack answering the questions in the form of an interview, with the researcher completing the paperwork. They were issued with a light box. Due to living in a motel, they only had a small space with no means to play the exercise DVD. They already did some walking, and were content to continue this form of exercise for the physical activity aspect of the intervention. However, the weather was cold during their intervention period and they admitted missing some of their scheduled exercise.

Ella reported that they used the light box successfully and that Jack enjoyed using it while reading. However, he sometimes fell asleep in front of it which meant Ella had to monitor him carefully and try and keep him awake during the therapy time (she reported that he was typically more alert in the afternoon). Ella thought that her sleep had improved and so had the impression that the intervention was helping.

In the diaries, Jack was documented as using the light box on 22 out of 35 days during the 9-11am time period (although some instances began prior to 9am). Ella also recorded five episodes of natural light exposure for Jack during the allotted timeframe. Physical activity was recorded on 20 out of the 35 days between 11-2pm. Table 5.15 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. This shows that Jack's amount of light recorded was less than the group's 25th percentile at both Times 1 and 2 with a reduction between times. He scored within the 25th percentile for activity at both times and showed an increase in his activity count at Time 2 compared to Time 1.

Table 5.15

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 2

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	1,160.4	(125.6-27,817.1)	573.7	(267.9-1,021.5)	-586.7
Average activity (counts/minute)	99.4	(53.3-140.6)	103.2	(71.3-176.8)	+3.8

Completion of the trial.

At the end of the trial, Ella commented that Jack's behaviour and memory had become much worse. He had increased difficulty with language, would refuse to get ready for bed, and was having more episodes of confused awakenings and agitated behaviour at night. He also had periods when he would resist care assistance. Ella noted that Jack was still sleepy in the day and it was hard work encouraging him to do any activities. She felt she was so tired at night it was likely that she was sleeping through some of Jack's awakenings and potentially underreporting the degrees of his sleep disturbance. Jack had some minor changes to his medications during the trial, but these were not considered relevant enough to exclude his Time 2 data from analyses.

Ella mentioned that the questionnaires required some thought to complete. Jack said that he found questions more difficult when there were more than two options, because it was difficult for him to retain and contemplate all of the options at once. In the feedback form, Ella reiterated that the light box was sometimes hard to use as Jack would close his eyes. They felt that walking was their only option for exercise and the weather affected their compliance with this. They found the sleep support handbook informative, Ella noted that getting enough good quality sleep was a major problem for carers and so she would recommend the interventions to others.

Results for PWD 2

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia related disruption can be found in Table 5.16. Note, pair 2 was missing part of the RMBPC and therefore depression and global scores were unavailable from this scale.

Table 5.16

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 2

Variable	PWD T1	PWD T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	2.0	0.75	-1.25
Night sleep (hrs)	7.0	8.0	+1.0
PSQI (0-21)	8.0	5.0	+3.0
SDI (0-12)	0.2	0.1	+0.3
Rating of nights' sleep (1-7 median, range)	2.0 (2-4)	2.0 (1-2)	No change
MMSE (30-0)	13.0	6.0	-7.0
QOL-AD PWD (52-13)	37.0	34.0	-3.0
QOL-AD carer (52-13)	23.0	25.0	+2.0
RMBPC Memory frequency (0-28)	24.0	21.0	+3.0
RMBPC Memory carer reaction (0-28)	2.3*	6.0	-3.7
RMBPC disruption frequency (0-32)	4.6*	8.0*	-3.4
RMBPC disruption carer reaction (0-32)	3.4*	3.4*	No Change

* = Item imputation used due to missing data from <20% of the component scores.

Jack slept in the same bed as Ella. At Time 1 he rated his sleep as "fairly good". His PSQI score indicated a mild sleep disturbance. This was mostly related to getting up to use the bathroom in the night (three or more times per week) and daytime sleepiness (once or twice per week). In addition, Ella reported that Jack had occasional episodes of disorientation or confusion, loud snoring (occurring once or twice a week), as well as occasional apnoeas. Ella's response to the SDI indicated some issues with getting to sleep as well as daytime sleepiness however these were not defined as severe or occurring every day (hence the low score). However Ella reported being moderately distressed by Jack's difficulties getting to sleep.

At Time 2, Jack still rated his sleep quality as "fairly good" and his diary ratings reflected this. Jack's PSQI score improved to be within normal range. However, some of this reduction could be attributed to the pair "not knowing" his sleep onset timing at Time 2. Ella's SDI rating also improved,

despite her reports of more difficulty at bedtime. Changes in the scores from the MMSE and RMBPC between Times 1 and 2 corroborate the pair's comments concerning an increase of dementia-related symptoms during the study period. However, the frequency of memory-related symptoms was slightly improved.

Actigraphy data.

There were six complete days of actigraphy data available from Jack at both Times 1 and 2 (Appendix 21, *Figure A.3.*). The sleep propensity curves using these data show that Jack had variable sleep timing. He had a later bedtime at Time 2, and he was also more likely to wake up earlier. However, his sleep during the night appeared to be more consistent at this time. He regularly napped at both Times 1 and 2, but at Time 2 he napped less in the morning compared to Time 1.

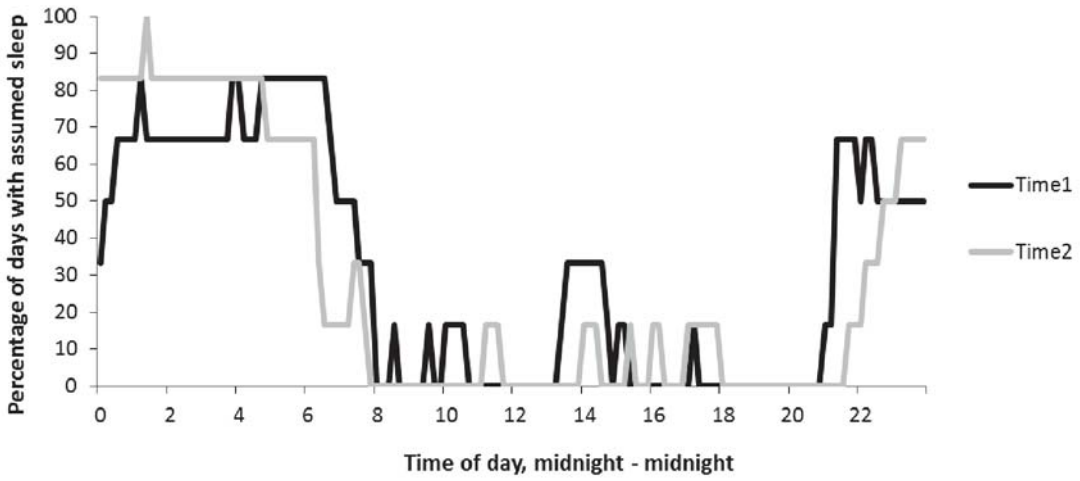

Figure 5.22. Sleep timing of PWD 2 at Time 1 and Time 2 (6 days).

Table 5.17 compares Jack's actigraphic sleep variables between Times 1 and 2. Jack had poor quality of sleep at both Times 1 and 2 as indicated by the low percentage of time spent asleep whilst in bed at night (approximately 62% at both times). He was having as little as 4.3 hours sleep at Time 1 and 3.0 hours at Time 2. Jack's sleep was exceptionally disrupted at night. Excerpts from 48-hours of his and Ella's raw actigraphy data from Times 1 and 2 are provided in *Figure 5.23* in order to further illustrate the degrees of fragmentation to his and subsequently her sleep.

Table 5.17

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 2

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	21:21		21:10	(20:48-22:27)	22:18		22:20	(21:15-23:00)	+70.0
Risetime	07:41		08:00	(07:00-08:00)	07:13		07:40	(04:50-08:10)	-20.0
Time in bed night, minutes	620.1	(56.6)	647.0	(524.0-669.0)	535.0	(79.7)	555.0	(350.0-625.0)	-92.0
Total sleep per night, minutes	380.9	(61.5)	403.0	(255.0-453.0)	322.3	(76.5)	341.0	(181.0-444.0)	-62.0
Sleep efficiency (% sleep night)	61.1	(6.1)	62.0	(47.2-67.7)	59.5	(7.0)	61.4	(48.3-71.0)	-0.6
Wake time night, minutes	239.3	(28.4)	247.0	(193.0-285.0)	212.7	(33.0)	214.0	(169.0-279.0)	-33.0
Number of night awakenings	66.0	(5.8)	63.0	(61.0-78.0)	56.4	(12.2)	61.0	(31.0-70.0)	-2.0
Total sleep per day, minutes	40.3	(18.4)	27.0	(24.0-76.0)	48.8	(18.0)	47.0	(23.0-78.0)	+20.0

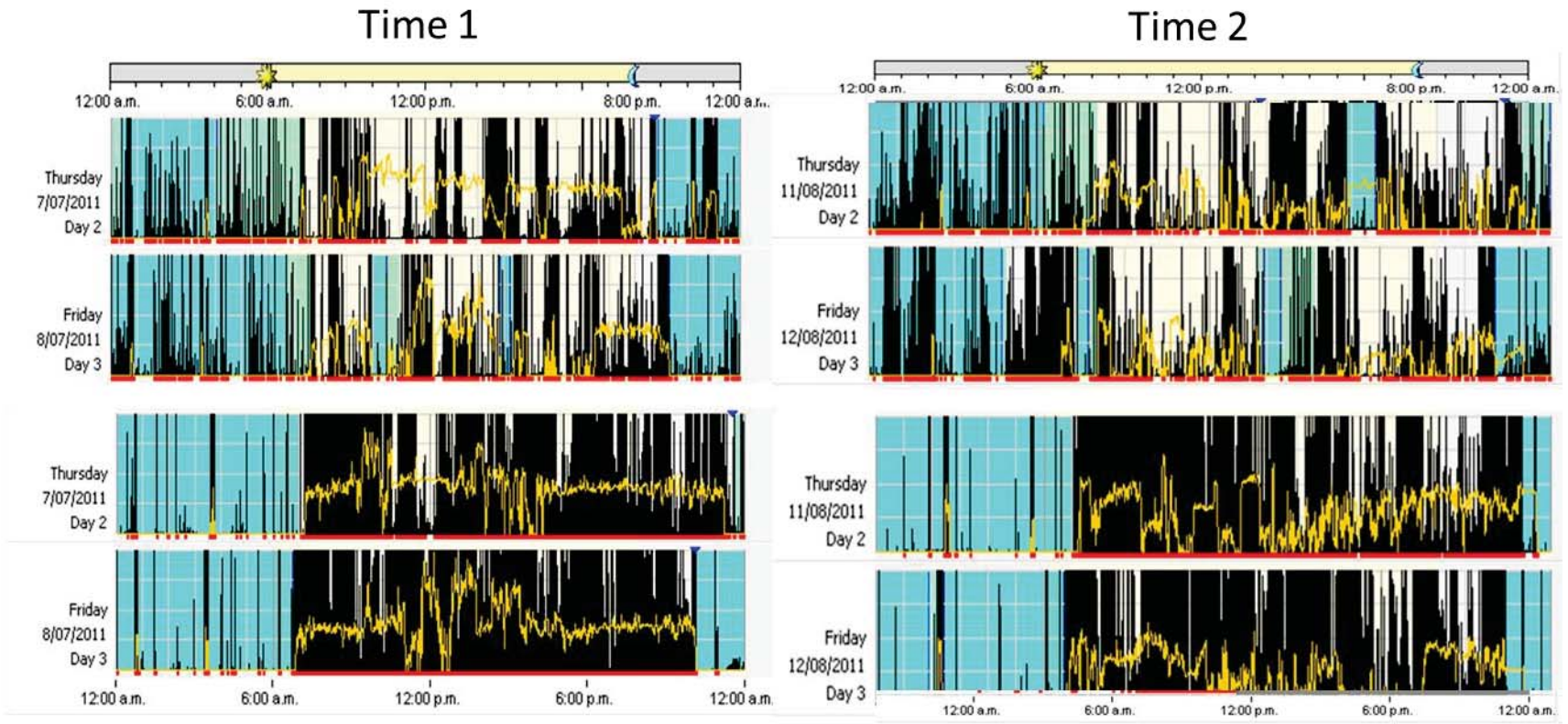

Figure 5.23. Two days of actigraphy data extracted from Jack's (top) and Ella's (bottom) sleep studies at Times 1 and 2.

Results for Carer 2

Questionnaire and diary data.

Questionnaire and diary data concerning Ella's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.18. Ella had not been diagnosed with any sleep disorders. At Time 1 she rated her sleep as "fairly bad". Her daily diary ratings were average. Her PSQI score indicated poor quality sleep. This was mostly related to waking up early, getting up to use the bathroom, and coughing or snoring (at least once or twice per week).

Table 5.18

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 2

Variable	Carer T1	Carer T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	0.0	0.0	No change
Night sleep (hrs)	6.0	7.5	1.5
PSQI (0-21)	8.0	3.0	+5.0
Rating of nights' sleep (1-7 median, range)	3.0 (2-5)	2.0 (1-4)	+1.0
MMSE (30-0)	26.0	25.0	-1.0
HADS-A (0-21)	4.0	5.0	-1.0
HADS-D (0-21)	9.0	8.0	+1.0
Cope positive (15-0)	12.0	12.0	No change
Cope negative (0-18)	4.0	4.0	No change

At Time 2 Ella rated her sleep as "very good". Her diary ratings improved by one point and her PSQI score reduced to within normal range, despite her commenting that Jack had been more disruptive at night. At Time 2 she noted that she had trouble with sleeping because of her anxiety associated with coping, as well as getting up to assist Jack with toileting. Ella's HADS scores indicated increased likelihood for depression at both Times 1 and 2. Her COPE index indicated low risk of carer burden. These scores remained stable between Times 1 and 2. Ella reported that she "always" felt well supported in her carer role at both times.

Actigraphy data.

There were seven complete days of data available from Ella at both Times 1 and 2 (Appendix 21, *Figure A.4*). The sleep propensity curves using these data show little change to the timing of her night time sleep. However, she had one night with long periods of wake at Time 2 (Figure 5.2). The fragmentation within the sleep intervals and in relation to Jack's sleep is illustrated in the excerpts of the actograms above in *Figure 5.23.*

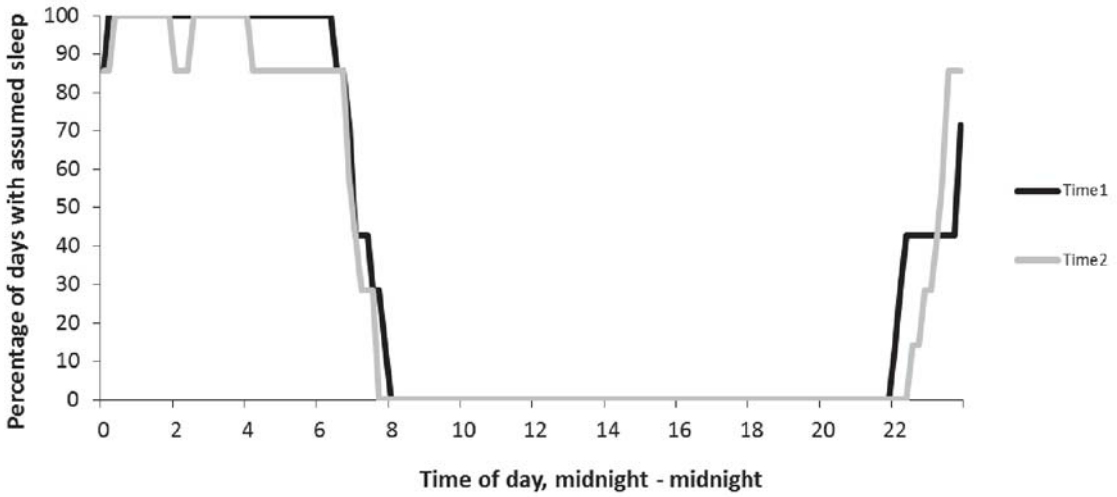

Figure 5.24. Sleep of carer 2 at Times 1 and 2 (7 days).

Table 5.19 compares actigraphic sleep variables for Ella between Times 1 and 2. At Time 2 Ella was spending more time in bed but also more time awake compared to Time 1. Although her average sleep efficiency was above 80%, on average she was having less than 7 hours sleep at night at both times.

Table 5.19

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 2

Variable	Carer T1				Carer T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	22:54		23:10	(21:59-23:42)	22:56		23:00	(22:00-23:30)	-00:10
Risetime	07:15		07:00	(06:38-8:00)	06:51		07:00	(04:15-08:15)	No change
Time in bed night, minutes	501.0	(69.8)	470.0	(416.0-590.0)	472.1	(86.6)	480.0	(290.0-595.0)	+10.0
Total sleep per night, minutes	439.9	(62.2)	405.0	(368.0-519.0)	397.1	(58.3)	414.0	(266.0-460.0)	+9.0
Sleep efficiency (% sleep night)	87.7	(2.5)	89.1	(83.9-90.4)	84.9	(5.6)	83.3	(77.3-91.9)	-5.8
Wake time night, minutes	62.0	(15.8)	61.0	(42.0-95.0)	75.0	(37.3)	80.0	(24.0-135.0)	+19.0
Number of night awakenings	17.9	(5.9)	17.0	(12.0-31.0)	15.7	(7.0)	14.0	(6.0-29.0)	-3.0
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)	No change

Short summary

Pair 2 completed the research whilst living in a motel unit. They used walking instead of the exercise DVD due to limited space and resources. Their compliance was sometimes affected by the bad weather. They reported using the light box, however had trouble with compliance due to Jack closing his eyes or sleeping during the therapy time.

Although their questionnaire data indicated improvements to their sleep, Ella reported that Jack's sleep had become more disturbed during the study period and that he had increased daytime sleepiness. She also reported deterioration to her own sleep due to providing more care at night and increased anxiety. The actigraphy recordings showed no improvement in objective sleep variables. They both had reduced sleep efficiencies at Time 2, indicating more time in bed spent awake compared to Time 1.

The questionnaire results supported Ella's reports of dementia-related deterioration, with reduced scores associated with Jack's cognitive functioning and dementia-related disruptions. However, the frequency of memory-related symptoms appeared to improve between Times 1 and 2. Ella's questionnaire showed an increased risk of depression and carer burden at both Times 1 and 2. This may have contributed to her trouble sleeping.

5.4.3 Pair 3 (George and Amy)

Description of Study Experience

Participants.

Pair 3 consisted of a married couple. George was a 72-year-old man with AD, which he was diagnosed with one year prior to the study. He had an MMSE score of 14, indicating moderate cognitive impairment. His comorbidities included heart disease and high blood pressure. He received treatment for these, however he still reported that the conditions limited his activities. Many of his medications had fatigue or tiredness listed as possible side effects, and one also listed insomnia and nightmares (see Appendix 20).

Amy was aged 71 years at the time of the study. She had been providing dementia-related care for her husband for just over one year. She was providing care day and night, as required by her husband. Amy had heart disease, high blood pressure, type-two diabetes, depression, as well as arthritis. She received treatment for many of these conditions and she didn't feel as though any of them limited her activities. Many of her medications had dizziness or fatigue listed as possible side effects, and two also listed insomnia and nightmares (see Appendix 20).

Data collection and intervention compliance.

George signed the consent form himself. Questionnaires were completed together, with George reading the questionnaires whilst the researcher read the questions out loud and completed the paperwork based on his responses. They were issued with a light box as well as exercise DVD, they already took regular walks together. During the initial week of the intervention they reported that George had had occasional headaches while using the light box. These were resolved after he was reminded not to look directly at the light and sit a little further away. By the third week they were using the light box routinely and felt they were getting on well. They enjoyed using the exercise DVD. Amy also participated, as well as their grandchildren which they had found fun. Towards the end of the intervention, poorer weather meant they hadn't had as much physical activity as usual, but they had continued to use the light box and DVD. Both Amy and George read the sleep support handbook.

In the diaries, it was documented that George used the light box on 31 out of the 35 days during the 9-11am time period. Physical activity was recorded on 21 of the 35 days, 18 of these instances were between 11-2pm.

Table 5.20 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. This shows that George's amount of light recorded was less than the 25th percentile at Time 1 but within the 50th percentile at Time 2. He scored within the 50th percentile for activity at both times and showed an increase in his activity count at Time 2 compared to Time 1.

Table 5.20

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 3

	Time 1		Time 2		
	Median	(Range)	Median	(Range)	Median change
Total light exposure (lux-minutes)	473.6	(177.5-2,977.2)	4,130.9	(629.3-134,316.0)	+3,657.3
Average activity (counts/minute)	129.6	(61.5-270.1)	197.6	(105.1-311.4)	+68.0

Completion of the trial.

At the end of the trial George and Amy reiterated that they had enjoyed taking part. They had used the light box at breakfast time and enjoyed the light from it. The exercise DVD had been fun for both of them. They found the sleep support handbook interesting however felt they were already doing a lot of what was advised. Amy also commented that they felt well supported by their local dementia community workers.

Results for PWD 3

Questionnaire and diary data.

Questionnaire data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.21. Note, pair 3 was missing part of the RMBPC and therefore depression and global scores were unavailable from this scale.

George slept in the same bed as Amy. At Time 1 he rated his sleep as "very good". His PSQI score was within the normal range. However he did have some instances of waking up early or waking to use the bathroom (three or more times per week). Amy also noted periods of confusion or disorientation in the night, symptoms of sleep apnoea, general restlessness, having incidents with toileting, as well as laughing in his sleep. Amy's response to the SDI indicated that George was getting up in the night, waking her, and that he was sleeping in the day. However, none of these behaviours were classed as very frequent or severe, hence the low SDI score.

Table 5.21

Comparison of Questionnaire Data at Times 1 and 2 Concerning PWD 3

Variable	PWD T1	PWD T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	2.0	0.5	-1.5
Night sleep (hrs)	10.5	9.5	-1.0
PSQI (0-21)	3.0	2.0	+1.0
SDI (0-12)	0.5	0.2*	+0.3
Rating of nights' sleep (1-7 median, range)	2.0 (1-3)	3.0(2-5)	-1.0
MMSE (30-0)	14.0	18.0	-4.0
QOL-AD PWD (52-13)	44.0	49.0	+5.0
QOL-AD carer (52-13)	30.0	35.0	+5.0
RMBPC Memory frequency (0-28)	21.0	22.0	-1.0
RMBPC Memory carer reaction (0-28)	6.0	7.0	-1.0
RMBPC disruption frequency (0-32)	2.3*	4.6*	-2.3
RMBPC disruption carer reaction (0-32)	1.1*	0.0*	+1.1

* = Item imputation used due to missing data from <20% of the component scores.

At Time 2 George still rated his sleep quality as "very good". His PSQI and SDI scores improved slightly. This was related to George being considered less likely to wake in the night or disturb Amy. The frequency of his confused episodes had also reduced. However, in the sleep diaries his sleep quality score deteriorated by one point. Amy noted that he was still tired in the day and sometimes had micro naps (too short to be noted in the diary or score actigraphically). They had some later nights at Time 2 due to watching the rugby on television.

Changes in the scores from the MMSE and RMBPC between Times 1 and 2 show some deterioration with regards to George's cognition, memory and dementia-related behaviours. However he and Amy both rated his quality of life more highly at Time 2.

Actigraphy data.

There were seven complete days of actigraphy data available from George at Time 1, and six at Time 2 (Appendix 21, Figure A.5). The sleep propensity curves using these data show that George had reasonably stable sleep timing. He was having later and more consolidated afternoon naps at Time 2 compared to Time 1 (*Figure 5.25*).

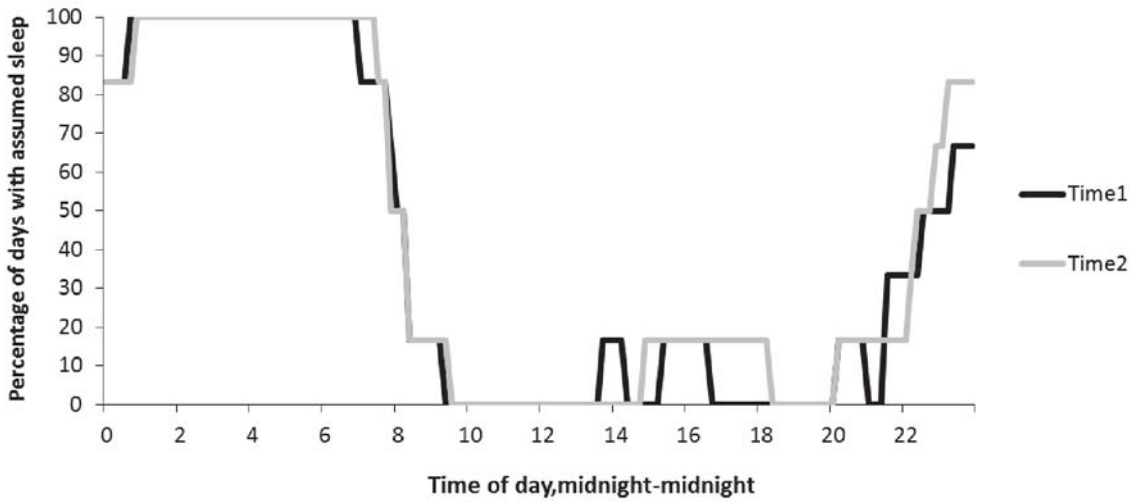

Figure 5.25. Sleep timing of PWD 3 at Time 1 and Time 2 (6 days).

Table 5.22 compares George's actigraphic sleep variables between Times 1 and 2. George had good quality of sleep at both times as indicated by the percentage of time spent asleep whilst in bed at night being ≥ 80%. He was getting a minimum of 6.0 hours sleep at night at Time 1 and 7.7 hours at Time 2.

Results for Carer 3

Questionnaire and diary data.

Questionnaire data concerning Amy's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.23. At Time 1 Amy rated her sleep as "very good". Her daily dairy rating also indicated good sleep. Her PSQI score indicated a minor sleep disturbance. This was related to having to get up to use the bathroom (three or more times per week) as well as sometimes (less than once per week) being unable to get to sleep within 30 minutes.

Table 5.22

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 3

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	22:26		22:05	(09:20-00:28)	22:33		22:24	(20:32-00:50)	+19
Risetime	08:18		08:15	(07:04-09:46)	08:12		08:00	(07:19-09:38)	-15.0
Time in bed night, minutes	583.6	(72.0)	610.0	(442.0-663.0)	578.8	(52.7)	573.5	(516.0-672)	-36.5
Total sleep per night, minutes	501.6	(76.7)	539.0	(362.0-594.0)	505.8	(45.1)	493.0	(461.0-588)	-46.0
Sleep efficiency (% sleep night)	85.6	(3.2)	86.5	(80.5-89.6)	87.4	(1.5)	87.4	(85.2-90.1)	+0.9
Wake time night, minutes	82.0	(11.8)	80.0	(69.0-105.0)	73.0	(11.3)	76.5	(51.0-84.0)	-3.5
Number of night awakenings	24.9	(5.0)	23.0	(18.0-33.0)	24.7	(6.8)	25.0	(14.0-33.0)	+2.0
Total sleep per day, minutes	53.0	(13.1)	55.0	(30.0-72.0)	72.3	(31.0)	67.0	(36.0-119.0)	+12.0

Table 5.23

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 3

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.0	0.0	No change
Night sleep (hrs)	Missing	8.0	N/A
PSQI (0-21)	4.0*	4.0	No change
Rating of nights' sleep (1-7 median, range)	2.0 (1-2)	2.0 (2-3)	No change
MMSE (30-0)	28.0	30.0	+2.0
HADS-A (0-21)	3.0	1.0	+2.0
HADS-D (0-21)	1.0	1.0	No change
Cope positive (15-0)	14.0	15.0	+1.0
Cope negative (0-18)	1.2*	2.5	-1.3

* = Item imputation used due to missing data from <20% of the component scores.

At Time 2 Amy also rated her sleep as "very good", and her PSQI score remained within the normal range. Amy's HADS scores indicated a reduced likelihood for anxiety or depression at Times 1 and 2. She also had low risk of carer burden (as indicated by her COPE scores). At both times she reported that she "always" felt well supported in her carer role.

Actigraphy data.

There were eight complete days of data available from Amy at Time 1, and six at Time 2 (Appendix 21, *Figure A.6*). The sleep propensity curves using these data show little change to the timing of her sleep between Times 1 and 2 (*Figure 5.26*). Table 5.24 compares actigraphic sleep variables for Amy between Times 1 and 2. Amy had good quality of sleep at both Times 1 and 2 as indicated by the percentage of time spent asleep whilst in bed at night being ≥ 85%. She was getting at minimum of 6.3 hours sleep at Time 1 and 6.8 hours at Time 2.

Table 5.24

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 3

Variable	Carer T1				Carer T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:54		23:44	(23:23-01:06)	23:30		23:19	(23:05-00:09)	-19.0
Risetime	08:20		08:14	(07:22-09:35)	08:16		08:03	(7:44-9:36)	-11.0
Time in bed night, minutes	506.9	(41.2)	510.5	(431.0-555.0)	526.1	(27.9)	525.0	(483.0-67.0)	+14.5
Total sleep per night, minutes	451.6	(38.0)	463.0	(378.0-502.0)	459.3	(30.2)	455.0	(412.0-508.0)	-8.0
Sleep efficiency (% sleep night)	89.1	(2.0)	88.9	(85.8-92.5)	87.2	(1.9)	86.4	(85.0-90.4)	-2.4
Wake time night, minutes	55.3	(11.9)	51.0	(41.0-79.0)	66.9	(9.0)	69.0	(54.0-79.0)	+18.0
Number of night awakenings	26.5	(5.8)	25.5	(19.0-37.0)	27.3	(3.1)	27.0	(24.0-33.0)	+1.5
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)	No change

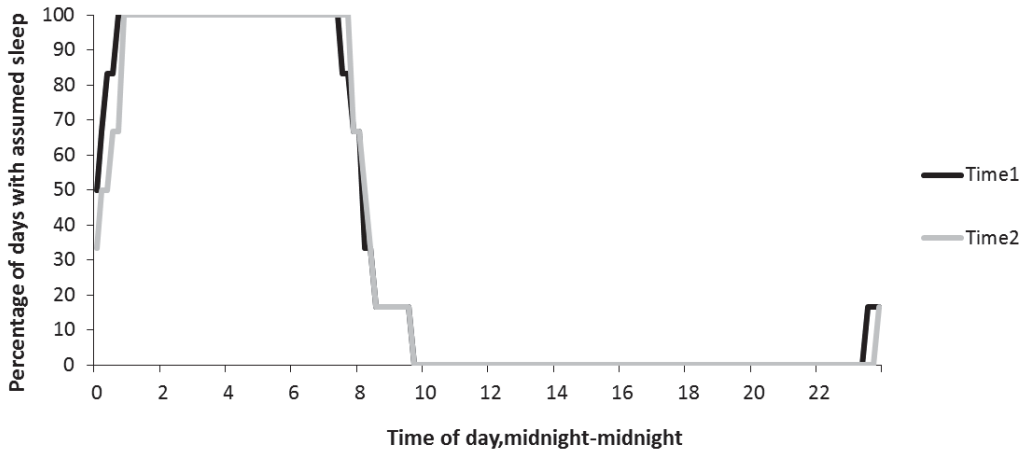

Figure 5.26. Sleep of carer 3 at Times 1 and 2 (6 days).

Short summary

Pair 3 completed the sleep study using both the light box and exercise DVD regularly. George had very good compliance with using the therapies in the allotted times and they reported enjoying taking part. Both George and Amy had good quality sleep at the study onset. Although there appeared to be some subjective improvements to George's sleep at Time 2 compared to Time 1, his actigraphy recordings showed no improvement in his objective sleep variables. George reported spending less time asleep in the day at Time 2. However, Amy noted that daytime sleepiness was still an issue for George, and his actigraphy records showed a small increase in daytime sleep between Times 1 and 2. Amy reported that he was restless at night at both Times 1 and 2 however this may have reduced somewhat at Time 2. Amy's actigraphy reports showed that she was spending more time in bed awake at Time 2 compared to Time 1. However her sleep duration and efficiency remained good.

George's questionnaire results indicated some deterioration with regards to his cognitive functioning and memory. Amy appeared to be of low risk for anxiety or depression, and was coping well with her caregiving role. They appeared to be a very happy couple and enjoyed the research process. It seems that George and Amy had reasonably good sleep in the outset and therefore, despite good compliance with the intervention there were no positive effects. Negative changes to their sleep were minor and could have been related to their late nights watching television, or George's dementia-related deterioration at Time 2.

5.4.4 Pair 4 (Thomas and Sarah)

Description of study experience

Participants.

Pair 4 consisted of a married couple. Thomas was a 76-year-old man with an undefined type of dementia, which he was diagnosed with approximately six years prior to the study. He had an MMSE score of 23, which is within the normal range for cognitive functioning. He also had heart disease which he received treatment for and didn't affect his activities. Many of his medications had fatigue or tiredness listed as possible side effects (see Appendix 20).

Sarah was aged 74 years at the time of the study. She had been providing dementia-related care for her husband for six years, for approximately an hour each day. Sarah also had high blood pressure (treated) and anaemia or blood disease. Neither of these affected her daytime activities. One of her medications had fatigue listed as a possible side effect as well as (more rarely) dizziness and nightmares (see Appendix 20).

Data collection and intervention compliance.

Thomas signed the consent form himself. Questionnaires were completed together, with Thomas reading the questionnaires whilst the researcher read them out loud and completed the paperwork based on his and Sarah's responses. They were issued with a light box as well as exercise DVD. They reported that, although the exercise DVD seemed good, they preferred walking as Thomas was still fit enough and able to take walks alone. They felt that the DVD would be a useful option for those less able. During the intervention they reported getting on well with the light box and exercises.

In the diaries, they documented using the light box on 34 out of the 35 days during the 9-11am time period. On one day they used the light box after 11am. Physical activity was recorded on 30 out of the 35 days between 11-2pm. Table 5.25 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. This shows that Thomas had the highest total light exposure for the group at Time 1, this increased even more at Time 2. He also scored above group's 75th percentile in activity counts at both times, however there was a slight decrease at Time 2 compared to Time 1.

Table 5.25

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 4

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux)	18,717.0	(606.1-118,603.9)	48,136.1	(8,362.5-83,781.7)	+29,419.1
Average activity (counts/minute)	215.1	(123.5-336.6)	169.4	(98.6-257.1)	-45.7

Completion of the trial.

At the end of the trial Thomas and Sarah reiterated that they had used the interventions and got on well. They had had some trouble with the Actiwatch event marker. They found it difficult to know if they had pressed it properly, because it was very small and did not make a noise. They reported that it was useful to know all of the study details, particularly the length of the study in order to be prepared for the protocol. Sarah mentioned that Thomas' daytime sleeping was an issue and as he dozed on and off a lot of the day it was impossible to keep track of these times in the diary. Therefore these periods were not represented and could not be reliably scored actigraphically.

Results for PWD 4

Questionnaire and diary data.

Questionnaire data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.26. Thomas slept in the same room but a different bed from Sarah. At Time 1 he rated his sleep as "very good" in both his questionnaire and daily diary ratings. His PSQI score was within the normal range with just some instances of waking up to use the bathroom (three or more times per week), and sometimes waking up too early (less than once per week). He also reported that sometimes his sleep was disturbed by Sarah's snoring.

Table 5.26

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 4

Variable	PWD T1	PWD T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	1.0	1.5	+0.5
Night sleep (hrs)	7.5	7.0	-0.5
PSQI (0-21)	3.0	3.0	No change
SDI (0-12)	0.0	0.0	No change
Rating of nights' sleep (1-7 median, range)	1.0 (1-1)	1.0 (1-1)	No change
MMSE (30-0)	23.0	23.0	No change
QOL-AD PWD (52-13)	45.0	48.0	+3.0
QOL-AD carer (52-13)	40.0	40.0	No change
RMBPC memory frequency (0-28)	13.0	17.5*	-4.5
RMBPC memory carer reaction (0-28)	7.0	5.8*	+1.2
RMBPC depression frequency (0-36)	2.3*	0.0	+2.3
RMBPC depression carer reaction (0-36)	1.1*	0.0	+1.1
RMBPC disruption frequency (0-32)	3.0	2.0	+1.0
RMBPC disruption carer reaction (0-32)	3.0	2.0	+1.0
RMBPC global frequency (0-96)	18.3	19.9	-1.6
RMBPC global reaction (0-96)	11.1	8.0	+3.1

* = Item imputation used due to missing data from <20% of the component scores.

Sarah's response to the SDI also indicated that Thomas was a good sleeper. Although she did report that he slept excessively in the daytime, this was in the form of multiple micro naps which were less than 10 minutes so were not marked in the diary. Sarah commented that, depending on the day, he could doze on and off all afternoon. She said Thomas was unaware of this and/or didn't consider it to be a problem. At Time 2 Thomas still rated his sleep quality as "very good". His PSQI and SDI scores remained normal. Sarah reported that he was still excessively sleepy in the daytime.

Thomas's MMSE scores indicated that his cognitive functioning was within the normal range. The RMBPC score showed some improvement in Thomas' mood and behaviour. However, his memory appeared to have deteriorated between Times 1 and 2. Thomas and Sarah both rated his quality of life as high at Times 1 and 2. Sarah's reaction to his dementia-related behaviours was low, indicating reduced carer burden.

Actigraphy data.

There were seven complete days of actigraphy data available from Thomas at both Times 1 and 2 (Appendix 21, *Figure A.7*). The sleep propensity curves using these data show that Thomas had stable sleep timing between Times 1 and 2 (*Figure 5.27*).

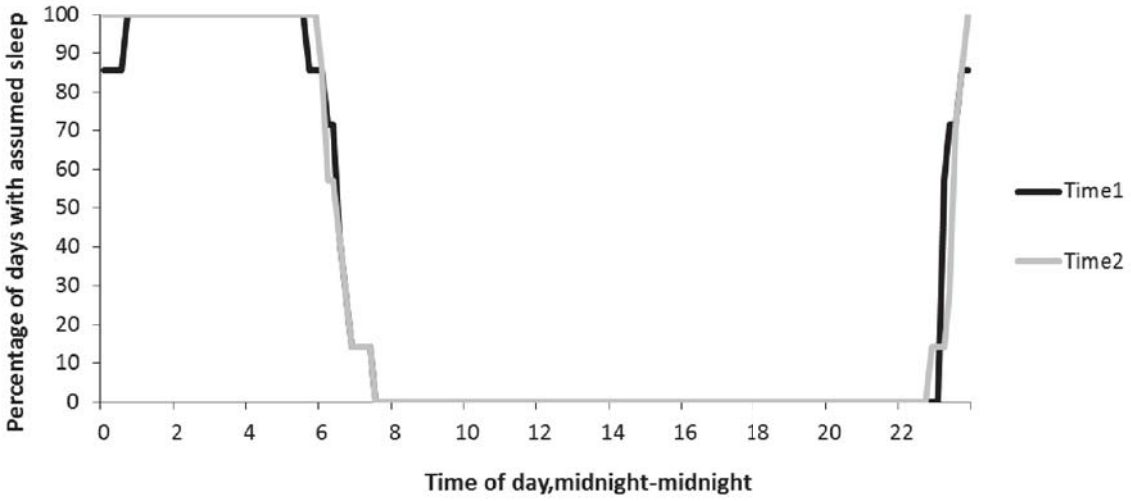

Figure 5.27. Sleep timing of PWD 4 at Time 1 and Time 2 (7 days).

Table 5.27 compares Thomas' actigraphic sleep variables between Times 1 and 2. Thomas had good quality of sleep at both Times 1 and 2, as indicated by his average time percentage of time spent asleep whilst in bed at night being over 80%. He was having a minimum of 5.3 hours sleep at Time 1 and 6.5 hours at Time 2. However, on average, he spent less time in bed or asleep at night at Time 2 compared to Time 1.

Table 5.27

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 4

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:03		23:00	(22:35-23:46)	23:01		23:04	(22:40-23:12)	+5.0
Risetime	07:40		07:38	(07:30-07:59)	07:28		07:38	(06:56-07:42)	No change
Time in bed night, minutes	517.2	(25.8)	522.5	(464.0-542.0)	507.0	(21.4)	508.0	(478.0-542.0)	-14.5
Total sleep per night, minutes	418.0	(49.3)	442.5	(317.0-460.0)	430.6	(29.4)	417.0	(389.0-487.0)	-25.5
Sleep efficiency (% sleep night)	80.6	(6.8)	81.9	(68.3-89.7)	84.9	(3.4)	85.4	(80.7-89.9)	+3.5
Wake time night, minutes	99.2	(30.4)	98.0	(53.0-147.0)	76.4	(16.3)	76.0	(55.0-100.0)	-22.0
Number of night awakenings	29.5	(4.8)	27.5	(24.0-37.0)	25.7	(4.2)	27.0	(19.0-31.0)	-0.5
Total sleep per day, minutes	Missing	Missing	Missing	Missing	Missing	Missing	Missing	Missing	Missing

Results for Carer 4

Questionnaire and diary data.

Questionnaire data concerning Sarah's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.28. At Time 1 Sarah rated her sleep as "very good". Her global PSQI score was missing due to her not knowing her sleep latency or sleep time. Within the PSQI, she reported occasional times (once or twice per week) that she couldn't get to sleep within 30 minutes, or was waking too early. She also got up at night to go to the bathroom three or more times per week. In her diary, Sarah's average sleep quality rating was mid-range at Time 1, she also noted some breathing difficulties that interfered with her sleep.

Table 5.28

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 4

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.17	0.0	-0.17
Night sleep (hrs)	Missing	8.0	N/A
PSQI (0-21)	Missing	2.0	N/A
Rating of nights' sleep (1-7 median, range)	3.0 (1-6)	1.0 (1-2)	+2.0
MMSE (30-0)	28.0	29	+1.0
HADS-A (0-21)	0.0	2.0	-2.0
HADS-D (0-21)	4.0	5.0	-1.0
Cope positive (15-0)	11.0	12.0	-1.0
Cope negative (0-18)	4.0	2.0	+2.0

At Time 2, Sarah still rated her sleep as "very good". Her PSQI score was within the normal range. Sarah's HADS scores indicated a reduced likelihood for anxiety or depression, however these scores rose slightly at Time 2. Although she had a reduced risk of carer burden (as indicated by her COPE scores), she reported that overall she felt less supported in her carer role at Time 2 compared to Time 1 ("often" vs. "always").

Actigraphy data.

There were seven complete days of actigraphy data available from Sarah at both Times 1 and 2 (Appendix 21, *Figure A.8*). The sleep propensity curves using these data show little change to the timing of her sleep between Times 1 and 2 (*Figure 5.28*).

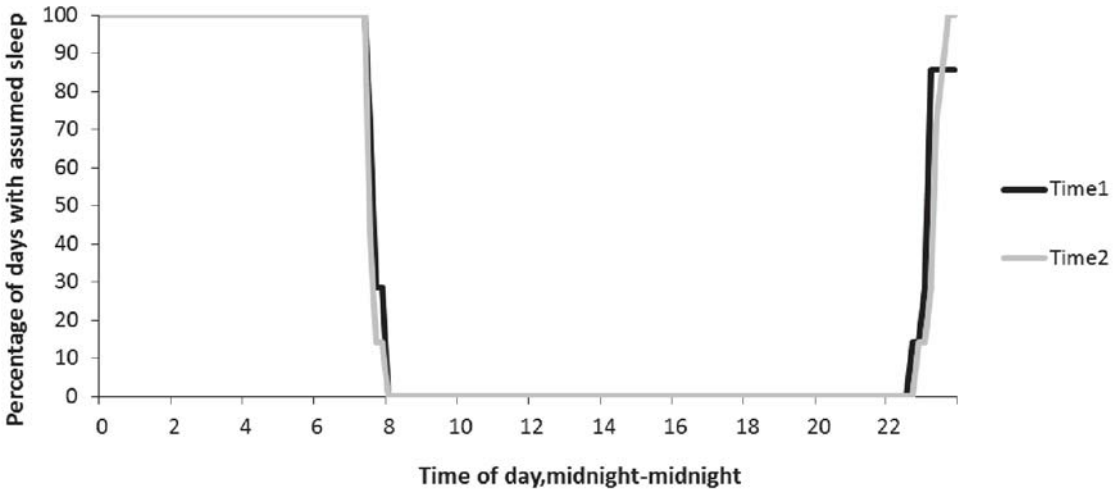

Figure 5.28. Sleep of carer 4 at Times 1 and 2 (7 days)

Table 5.29 compares actigraphic sleep variables for Sarah between Times 1 and 2. Sarah had good quality of sleep at both Times 1 and 2, as indicated by the percentage of time spent asleep whilst in bed at night being at least 83%. She was having at minimum of 6.7 hours sleep at night at Time 1 and 7.3 hours at Time 2.

Short summary

Pair 4 completed the sleep study using both the light box and exercise DVD regularly. Thomas' ambient light exposure and physical activity was high at Time 1. He had good compliance with using the light, further increasing his exposure, however his physical activity during the allotted time frame reduced between Times 1 and 2.

Table 5.29

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 4

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:01		22:57	(22:36-23:42)	23:00		22:58	(22:42-23:13)	+00:01
Risetime	07:45		07:45	(07:30-08:01)	07:58		07:52	(07:45-08:18)	+00:07
Time in bed night, minutes	523.7	(23.8)	528.0	(473.9-551.0)	537.9	(12.7)	534.0	(516.0-552.0)	+6.0
Total sleep per night, minutes	459.9	(28.4)	471.0	(402.0-492.0)	468.7	(16.4)	468.0	(437.0-492.0)	-3.0
Sleep efficiency (% sleep night)	87.8	(2.9)	88.7	(82.8-91.9)	87.1	(2.3)	87.1	(83.8-90.6)	-1.6
Wake time night, minutes	63.9	(14.4)	61.0	(42.0-91.0)	69.1	(12.7)	69.0	(50.0-89.0)	+8.0
Number of night awakenings	29.7	(5.5)	29.0	(21.0-38.0)	32.3	(2.4)	32.0	(29.0-35.0)	+3.0
Total sleep per day, minutes	13.0	(0.00)	13.0	(13.0-13.0)	0.0	(0.0)	0.0	(0.0-0.0)	-13.0

Thomas had good quality sleep at the study onset. His actigraphy recordings showed just a minor improvement in his sleep efficiency. There were no other positive changes between his objective sleep variables between Times 1 and 2. Unfortunately there were no reliable actigraphy data available for Thomas' daytime sleeps. This was due to the fragmented and sporadic nature of his dozing, which meant Sarah was unable to track it in the diaries. In the questionnaires, Thomas reported a 30 minute increase in his daytime sleeping, and Sarah reported that he was still excessively tired in the day at Time 2.

Sarah appeared to be of low risk for anxiety or depression and was coping well with her caregiving role. Her subjective and objective data indicated that she was having good quality sleep throughout. Sarah self-rated her night sleeps more highly at Time 2. She had a little more time in bed compared to at Time 1, but more time spent awake. However these differences are negligible considering her high baseline.

Thomas' questionnaire results indicated that he was in the mild stages of dementia, with an MMSE score within the normal range. Between Times 1 and 2 there was some deterioration with regards to memory, however his mood and dementia-related behaviours appeared to improve. It seems that Thomas and Sarah had reasonably good sleep at the outset and therefore, despite reasonable compliance with the intervention, there were no positive effects. Unfortunately, due to the protocol and methodology of the study, no changes could be observed in daytime sleeping which was the crux of Thomas' sleep-related problem.

5.4.5 Pair 5 (Adam and Claire)

Description of Study Experience

Participants.

Pair 5 consisted of a married couple. Adam was an 82-year-old man with LBD which he was diagnosed with approximately five years prior to the study. He had an MMSE score of five, indicating severe cognitive impairment. He also had Parkinson's disease. Initially Adam was excluded from taking part due to the possibility of his Parkinsonian tremor affecting the reliability of the actigraphy recording.

However, due to his wife's enthusiasm to try the interventions and changes to the study protocol, the pair were included. Adam also had lung disease which was treated but still limited his activities. Some of his medications had fatigue listed as a potential side effect, two listed insomnia, and one also listed nightmares (see Appendix 20).

Claire was aged 68 years at the time of the study. She had been providing dementia-related support for her husband for six years. She also worked a 40 hour week, during which time she had a relief carer assisting Adam. Claire had haemochromatosis and also suspected she had arthritis. She took two medications which had drowsiness and fatigue listed as possible side effects, as well as (more rarely) dizziness, insomnia or nightmares (see Appendix 20).

Data collection and intervention compliance.

Claire gave consent on behalf of Adam using the statement by partner form, as she considered him too impaired to fully understand the nature of the research. Adam was in the advanced stages of dementia and was too tired to complete all of the questionnaires with the researcher. Adam did not have the cognitive capacity to complete his QOL-AD questionnaire at either Time 1 or 2. The MMSE was conducted at Time 1 only as he was asleep when the researcher visited at Time 2. Questionnaires regarding Adam were completed by Claire with the researcher present to clarify any questions.

Pair 5 were issued with a light box as well as exercise DVD. During the initial weeks of the trial, Claire reported that Adam got on very well with the light therapy. They were using it most days (except when he seemed agitated). Claire had noticed that he was much more active during the days that he used it. She commented that he would wake up more "sprightly" and at a more convenient time in the morning. She also noticed that Adam was more lucid during the times he was using the light box. For example, he would be more likely to participate in completing the crossword or feeding himself breakfast. She commented that he seemed less disturbed at night and not as sleepy in the day. This had been good for them as they were able to go out on trips and in the car, with him staying awake and enjoying their time together.

Later in the trial Claire reported that Adam was still sleepy in the day but not as much as he had been. She associated his sleepiness with his comorbidities. Some of these other conditions were considered to be more of a burden than his sleep disturbances (for example his Parkinson's and digestive problems). They did not use the exercise DVD because Adam was not physically able. He only walked inside and up their stairs. Claire commented that he became less physically active during the trial, but she felt he was more alert when awake. Claire reported that the sleep support handbook was clear and informative. One aspect they reported trying was changing the time of day that Adam took his AChEI medication to try and relieve some of his daytime sleepiness.

In the diaries, Claire documented using the light box on 23 out of 35 days during the 9-11am time period. On four days they used the light box after 11am. Physical activity was documented on just one day during the five week intervention. Table 5.30 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. This shows that Adam's light exposure was within the 50th percentile at Time 1, but it reduced to the 25th percentile at Time 2, despite their reports of compliance. Adam had very low activity recorded at both Times 1 and 2, with an average score of less than 40 counts.

Table 5.30

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 5

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	2,960.8	(1,739.0-42,337.7)	1,883.2	(124.9- 5,645.0)	-1,077.6
Average activity (counts/minute)	37.2	(4.2-65.6)	33.4	(5.1-59.0)	-3.8

Completion of the trial.

At the end of the trial Claire reiterated that they had got on very well using the light box and had been very happy with the results. She felt that Adam's sleep and waking function had improved. Claire commented on the irony that once the Actiwatch was put on at Time 1, Adam's sleep seemed to improve.

Before the trial his sleep had been worse still. He had been getting up more in the night, banging on the walls and getting up very early, but once the study began some of these problems appeared to resolve.

In her final questionnaire, Claire commented that taking part had helped her to identify a pattern of sleep and wake for Adam. Before he had dementia, he would get up between 1-3am to make a cup of tea as his legs were restless (on retrospect she associates this with the early stages of his Parkinson's disease). With the progression of dementia, she felt that he still had this pattern of getting up but he would wander and be more confused instead. As this awakening was predictable, Claire would leave a light on for Adam and would instinctively wake and get up around that time to guide him back to bed.

In their feedback form Claire noted that using the Actiwatch was no problem at all. She had expected that Adam would try to remove it, but that hadn't been the case. Keeping the diaries had been a "commitment, but the positive outcome with the analysis has been amazing". Overall Claire reported finding the experience very informative and enjoyable, with positive results.

Results for PWD 5

Questionnaire and diary data.

Questionnaire data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.31. Adam slept in a different room from Claire. At Time 1 his sleep was rated as "fairly good". His PSQI indicated some sleep disturbance. This was related to waking up in the middle of the night or early morning, coughing or snoring, feeling too hot or cold, as well as bad dreams (three or more times per week). He also had some daytime sleepiness and, less often (once or twice per week), uncomfortable breathing which disrupted his sleep. Claire added that he had frequent symptoms of RLS, would walk around at night, have episodes of confusion or disorientation, and would come and wake her up (three or more times per week). Claire's response to the SDI also indicated that Adam's sleep was disturbed, including wandering-type behaviours at night, wakefulness at night and sleepiness in the daytime. Many of these behaviours were considered severely distressing for Claire.

At Time 2 Claire rated Adam's sleep more poorly than at Time 1 (as indicated by an increased PSQI score and the daily diary ratings). Overall she rated his sleep as "fairly bad". Claire reported that, in addition to the symptoms listed at Time 1, he had been having instances of talking and hallucinations during his sleep at Time 2. However, the SDI score improved. This was related to Adam not being as sleepy in the daytime or waking so early. Furthermore the severity ratings and carer-related distress associated with Adam's sleep disturbances reduced at Time 2.

Table 5.31

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 5

Variable	PWD T1	PWD T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	5.0	2.0	-3.0
Night sleep (hrs)	12.0	12.0	No change
PSQI (0-21)	6.0	7.0	-1.0
SDI (0-12)	4.2	2.9	+1.3
Rating of nights' sleep (1-7 median, range)	2.0 (1-2)	6.0 (5-7)	-4.0
MMSE (30-0)	Missing	5.0	N/A
QOL-AD PWD (52-13)	Missing	Missing	N/A
QOL-AD carer (52-13)	27.0	34.0	+7.0
RMBPC memory frequency (0-28)	24.0	15.0	+9.0
RMBPC memory carer reaction (0-28)	2.3*	5.0	-3.7
RMBPC depression frequency (0-36)	8.0	11.0	-3.0
RMBPC depression carer reaction (0-36)	6.0	6.0	No change
RMBPC disruption frequency (0-32)	11.0	10.0	+1.0
RMBPC disruption carer reaction (0-32)	12.0	12.0	No change
RMBPC global frequency (0-96)	43.0	36.0	+7.0
RMBPC global reaction (0-96)	20.4	23.0	-2.6

* = Item imputation used due to missing data from <20% of the component scores.

Adam's MMSE and RMBPC scores indicated that he had severe cognitive impairment, with poor memory and disruptive behaviours. However his mood and quality of life ratings were good, and Claire rated his quality of life more highly at Time 2. The frequency of his memory-related behaviours improved between Times 1 and 2. Claire's reactions to Adams dementia-related behaviours were relatively low, however seemed to increase between Times 1 and 2 (as indicted by her RMBPC reaction scores).

Actigraphy data.

There were seven complete days of actigraphy data available from Adam at Time 1, and six at Time 2 (Appendix 21, *Figure A.9*). The sleep propensity curves using his data show that Adam had variable sleep timing at both times, however more so at Time 1. His night time sleep shifted earlier at Time 2, and became more consolidated compared to Time 1. His daytime sleep also became more consistent at Time 2 (*Figure 5.29*).

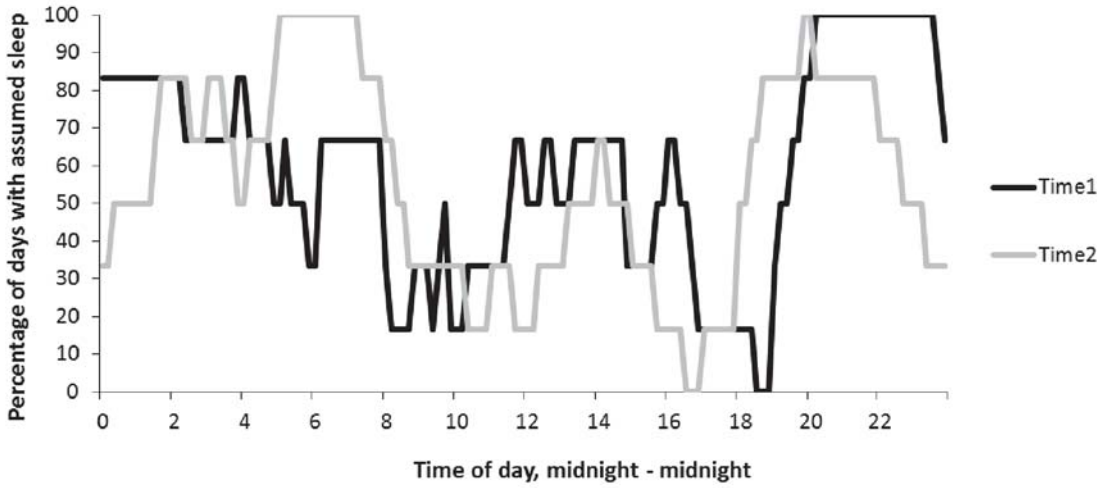

Figure 5.29. Sleep timing of PWD 5 at Time 1 and Time 2 (6 days).

Table 5.32 compares Adam's actigraphic sleep variables between Times 1 and 2. This shows great improvements to Adam's sleep at Time 2. He was getting almost 3.5 hours more sleep at night at Time 2 and, despite being in bed for longer, he had less awakenings during the night. This is also reflected in the increase in his percentage of sleep whilst in bed increasing from 78% to 81%. Adam's daytime sleep increased. However, as shown in *Figure 5.29*, his naps became later in the day and more consolidated (starting between 10:20-14:00) compared to during Time 1 (06:10-17:10). Adam's sleep was exceptionally disrupted at night. Excerpts from 48 hours of his and Claire's raw actigraphy data from Times 1 and 2 are also provided in *Figure 5.30* in order to illustrate the degrees of fragmentation and changes between times (see Appendix 21, *Figures A.9 &A.10* for full studies).

Table 5.32

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 5

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(range)	Mean	(SD)	Median	(range)	
Bedtime	19:11		19:00	(18:40-20:05)	18:07		18:08	(17:00-19:00)	-52.0
Risetime	07:04		07:00	(05:40-9:20)	09:09		09:20	(07:40-10:20)	+140.0
Time in bed night, minutes	713.6	(71.5)	715.0	(620.0-795.0)	902.5	(49.1)	907.5	(805.0-960.0)	+192.5
Total sleep per night, minutes	523.3	(92.0)	528.0	(398.0-699.0)	745.8	(73.0)	736.5	(662.0-864.0)	+208.5
Sleep efficiency (% sleep night)	73.7	(12.1)	77.8	(53.1-87.9)	82.9	(8.8)	81.3	(69.3-96.9)	+3.6
Wake time night, minutes	190.3	(96.8)	159.0	(88.0-372.0)	156.2	(84.8)	161.0	(28.0-295.0)	+2.0
Number of night awakenings	50.3	(22.6)	45.0	(27.0-88.0)	25.3	(10.8)	24.5	(11.0-47.0)	-20.5
Total sleep per day, minutes	111.6	(82.5)	78.0	(47.0-369.0)	144.8	(62.3)	192.0	(58.0-198.0)	+114.0

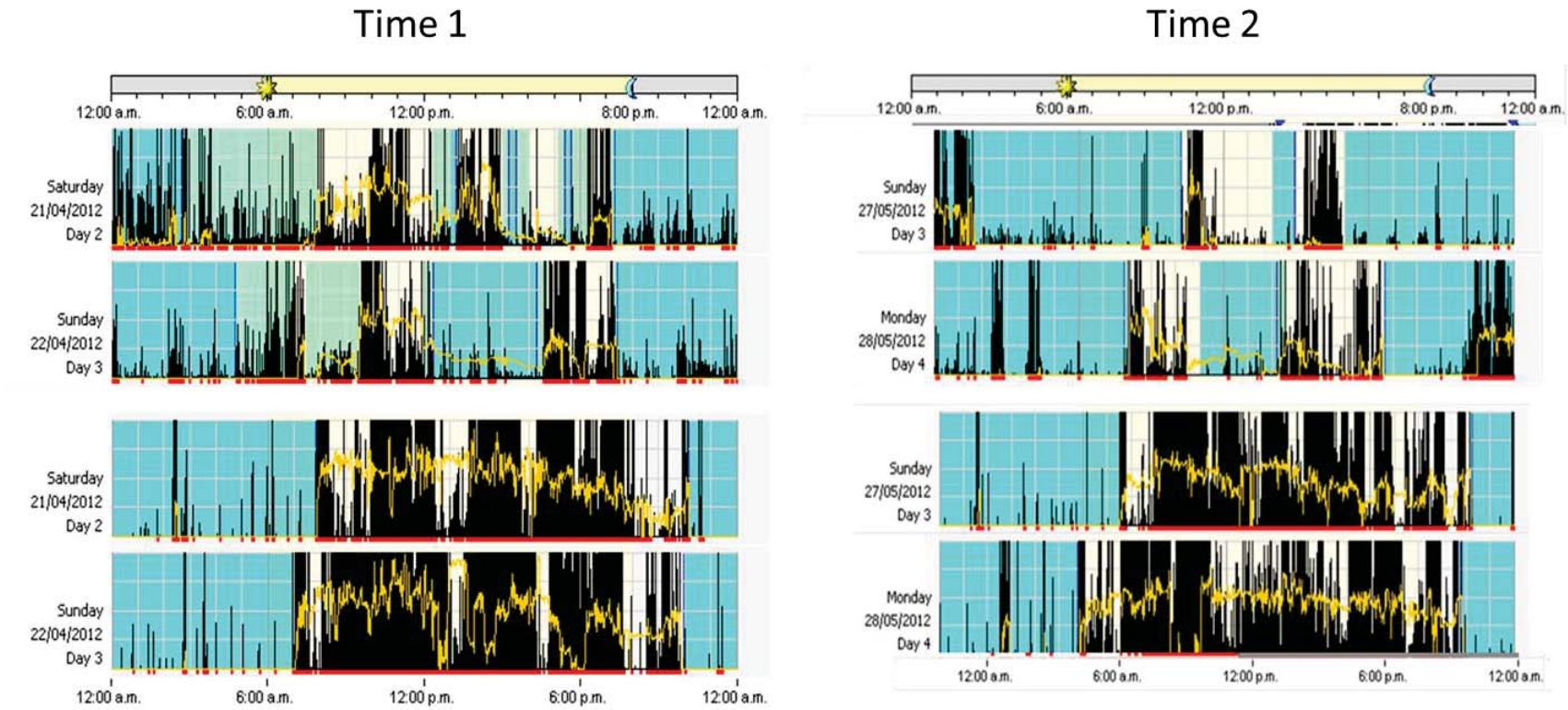

Figure 5.30. Two days of actigraphy data extracted from Adam (top) and Claire's (bottom) sleep studies at Times 1 and 2.

Results for Carer 5

Questionnaire and diary data.

Questionnaire data concerning Claire's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.33. At Time 1 she rated her sleep as "fairly good", however her PSQI score indicated that she had disturbed sleep. This was related to being unable to get to sleep within 30 minutes, being woken by Adam in the night, and waking in the middle of the night or early morning (at least once or twice per week). She also got up at night to go to the bathroom three or more times per week. She was taking a sleeping medication once or twice a week.

Table 5.33

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 5

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.17	0.17	No change
Night sleep (hrs)	7.0	6.5	-0.5
PSQI (0-21)	9.0	6.0	+3.0
Rating of nights' sleep (1-7 median, range)	2.0 (1-3)	Missing	N/A
MMSE (30-0)	28.0	29.0	+1.0
HADS-A (0-21)	3.0	6.0	-3.0
HADS-D (0-21)	1.0	0.0	+1.0
Cope positive (15-0)	14.0	13.0	-1.0
Cope negative (0-18)	8.0	7.0	+1.0

At Time 2 Claire also rated her sleep as "fairly good", but her PSQI score improved by three points. She was having less trouble getting to sleep and was not taking her sleeping medications as often (less than once a week). She no longer reported being disturbed by Adam. However, she was waking with "a sudden need to check on him". Claire's HADS scores indicated a reduced likelihood for anxiety or depression across the study. However, her anxiety score rose slightly at Time 2. She had low risk of carer burden (as indicated by her COPE scores) and reported that she "often" felt supported in her role as a caregiver.

Actigraphy data.

There were seven complete days of actigraphy data available from Claire at Time 1, and six at Time 2 (Appendix *21, Figure A.11*). The sleep propensity curves using these data show a slightly later bedtime at Time 2 (*Figure 5.31*).

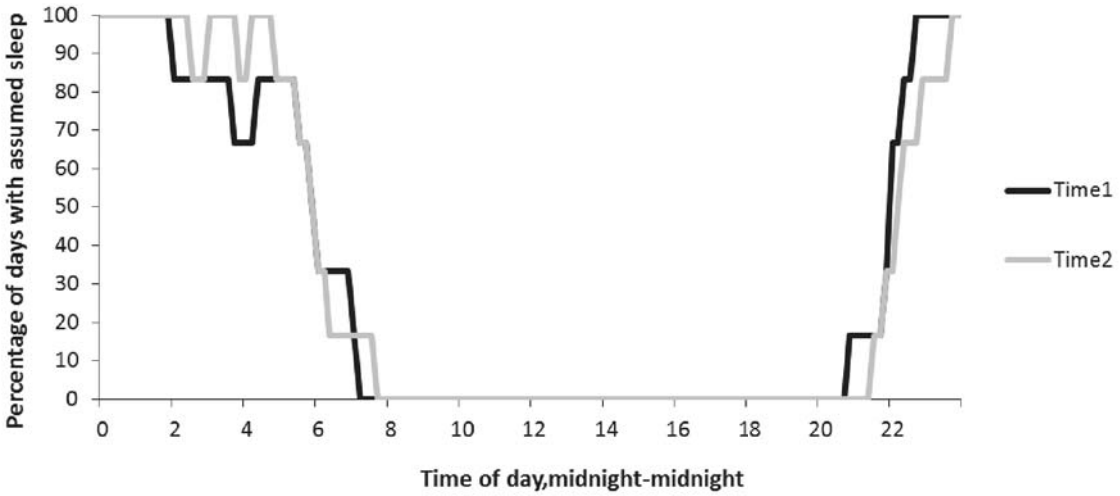

Figure 5.31. Sleep of carer 5 at Times 1 and 2 (6 days).

Table 5.34 compares actigraphic sleep variables for Claire between Times 1 and 2. Claire had good quality of sleep at both Times 1 and 2 as indicated by ≥82% of her time spent asleep whilst in bed at night. However, on average, she had less sleep at Time 2 compared to Time 1 (6.7 hours vs. 8.0 hours).

Table 5.34

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 5

Variable	Carer T1 (8 days)				Carer T2 (7days)				Median change
	Mean	(SD)	Median	(range)	Mean	(SD)	Median	(range)	
Bedtime	21:45		22:00	(20:40-22:20)	22:15		22:08	(21:30-23:35)	+8.00
Risetime	06:30		06:00	(05:00-8:00)	06:00		05:55	(04:50-07:35)	-5.00
Time in bed night, minutes	525.0	(68.2)	540.0	(420.0-630.0)	464.2	(50.6)	470.0	(400.0-545.0)	-70.0
Total sleep per night, minutes	470.1	(54.0)	483.0	(390.0-556.0)	419.5	(47.7)	400.5	(365.0-507.0)	-82.5
Sleep efficiency (% sleep night)	89.8	(4.2)	92.4	(83.3-94.8)	90.5	(4.6)	91.0	(82.1-97.3)	-1.4
Wake time night, minutes	54.9	(26.3)	41.0	(23.0-97.0)	44.7	(22.4)	40.0	(11.0-86.0)	-1.0
Number of night awakenings	18.3	(5.0)	18.0	(11.0-28.0)	14.5	(4.8)	15.5	(5.0-19.0)	-2.5
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)	0.0

Short summary

Pair 5 completed the sleep study using the light box regularly. They did not use the exercise DVD. They found the sleep support handbook useful and changed the time that they administered one of Adam's medications after reading it. Claire recorded good compliance with Adam's use of the light, however the recordings from the Actiwatch did not validate an increase in light exposure between Times 1 and 2.

Adam had very fragmented and poor quality sleep at Time 1 as well as wandering and disruptive behaviours at night. His actigraphy recordings showed substantial improvements in his sleep at Time 2. He had more time in bed and more time asleep compared to Time 1, thereby improving his percentage of sleep whilst in bed. His naps shifted to later in the day and were more consolidated. It appears that Adam's sleep became more organised and predictable. This is reflected in his objective data as well as Claire's reports of Adam sleeping to a more convenient time and being more lucid and cooperative during the day.

Adam was in the severe stages of dementia. However between Times 1 and 2 there was some improvement recorded with regards to the frequency of memory problems. Claire also recorded an improvement in his quality of life and commented on changes to his behaviour (e.g. able to feed himself) and cognition (e.g. take part in the crossword) during the days they used the light box.

Claire's PSQI scores and actigraphy reports showed that she was also having disturbed sleep. Much of this disturbance was related to being woken by Adam. Claire's actigraphy recordings showed no positive changes in her sleep. However she was having reasonable sleep at baseline. Despite no objective changes in her sleep, Claire's subjective ratings improved. She was no longer having trouble getting to sleep or being woken as often or as early by Adam.

Adam and Claire enjoyed taking part in the study and were as compliant as possible considering Adam's severe dementia-related impairments. Sleep was described as a huge problem for them at the study onset. They found the intervention beneficial for Adam's sleep. This had a subsequent influence on how Adam behaved in the daytime and also how Claire felt she slept.

5.4.6 Pair 6 (Anne, Matthew and Katie)

Description of study experience

Participants.

Pair 6 consisted of a mother and son who were living together. However participation was initiated and supervised by Anne's daughter, Katie. Anne was an 83-year-old woman with short term memory loss that began after she had a stroke two years prior to the study. Dementia had yet to be officially diagnosed; Anne wasn't interested in being investigated due to her anxiety around the prognosis. However she had symptoms of dementia as well as sleep disruptions so was keen to take part in the study. She had an MMSE score of 27, which was in the normal range for cognitive functioning. Her comorbidities included heart, lung and liver disease, high blood pressure, depression, arthritis, and back pain. She received treatment for three of these conditions. She considered the heart disease, arthritis and back pain limited her activities. Five of her medications had somnolence or fatigue listed as possible side effects, four listed dizziness, and three also listed insomnia as a rarer side effect (see Appendix 20).

Matthew was aged 46 years at the time of the study. He had been providing dementia-related support at night for his mother for two years. He worked a 44-hour week, during which time relief carers came to their home. His sister, Katie was also working but available to support her mother. Matthew had back pain which interfered with his daily activities, he did not disclose taking any medications.

Data collection and intervention compliance.

Katie gave consent on behalf of Anne using the statement by partner form. This was used due to protect Anne's anxiety surrounding a diagnosis of dementia. Matthew was happy to take part in the study, however he refused to have his own sleep monitored using actigraphy. Due to the nature of his work, he was unable to wear the actigraph watch during the day, and he was not interested in wearing it at night. Matthew was prepared to support his mum's participation and complete his questionnaires. However the home visits were attended by Katie due to restraints on his time. Due to Matthew and Katie's work commitments, Anne had multiple people supporting her. This made conducting the study more complicated regarding communication. This led to concerns as to whether information was being relayed

between Katie and Matthew, as well as the day-carers who supported Anne. Due to her children's work commitments, Anne began her intervention period 11 days into the study rather than 7.

Anne was hard of sight, therefore her questionnaires were completed interview-style with the researcher completing the paperwork. She had recently had cataract surgery and was due to have the other eye operated on soon. Because of this, she was uncomfortable with very bright lights. Therefore she was deemed unsuitable to use the light box. She used natural light therapy instead, and had a chair set up by a window that received the morning sun. They were issued with the exercise DVD which they seemed enthusiastic about. Anne was anxious about keeping the sleep diaries up to date and needed reassurance that Matthew would be maintaining them with/for her.

During the initial intervention weeks, Katie reported that Anne was only managing about five minutes of the DVD each time, as she found it tiring. They were encouraged to continue using it, just doing as much as she felt able to do. Over the following weeks Anne was able to manage a little more of the exercise DVD. Katie commented that she wondered if she was doing it as much as implied, however observed an improvement in technique so was reassured. They also reported that she had been sitting in her chair in the sun in the mornings.

Unfortunately Anne's compliance with the exercise DVD or natural light exposure was not documented in her daily diaries, possibly because Matthew was not supervising these activities, and their day-carers may not have been fully briefed. Table 5.35 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods. This shows that Anne's light exposure between 9-11am was within the group's 25th percentile at both Times 1 and 2, with a small increase of at Time 2. Her activity counts between 11am-2pm were within the group's 75th percentile at both times. Despite reports of her undertaking more physical activity, there was a reduction at Time 2 compared to Time 1.

Completion of the trial.

At the end of the trial Katie reported that, although Anne was happy to take part in the light and exercise related activities, she needed reminding a couple of times a week of why she was supposed to do them. Anne still had some anxiety around the study paperwork being completed. Matthew reported that

the sleep support handbook had too much information in it for Anne to consider or remember. They reiterated that Anne had enjoyed using the DVD; they were hoping to buy a copy to continue using after the trial. However, they were not sure if there had been any positive effects regarding her sleep.

Table 5.35

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 6

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	2,338.6	(1,065.3-38,3378.2)	2,873.0	(0.9-40,359.3)	+534.4
Average activity (counts/minute)	348.3	(162.4-401.5)	325.4	(139.6-440.7)	-22.9

Results for PWD 6

Questionnaire and diary data.

Questionnaire data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia related disruption can be found in Table 5.36. Matthew did not complete the carer reaction scales within the RMBPC at Time 1, therefore only the frequency scores are available.

Anne slept in a separate bedroom from Matthew. At Time 1 she rated her sleep as "fairly good". However her PSQI score indicated a borderline sleep disturbance. This was mostly related to waking up in the middle of the night or early morning, and getting up to use the bathroom (three or more times per week). She sometimes had problems going to sleep within 30 minutes, and less than once per week, was disturbed by being too hot or too cold, or bad dreams. Anne also noted that her neighbours sometimes woke her. Matthew's response to the SDI indicated that he considered Anne's sleep problems to mostly be related to excessive daytime sleepiness and waking others in the night. However these symptoms were not considered severe by Matthew, hence the low SDI score.

Table 5.36

Comparison of Questionnaire Data at Times 1 and 2 Concerning PWD 6

Variable	PWD T1	PWD T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	2.0	1.0	-1.0
Night sleep (hrs)	10.0	9.5	-0.5
PSQI (0-21)	5.0	3.0	+2.0
SDI (0-12)	0.2	0.7	-0.5
Rating of nights' sleep (1-7 median, range)	2.0 (1.0-6.0)	2.0 (2.0-4.0)	No change
MMSE (30-0)	27.0	26.0	-1.0
QOL-AD PWD (52-13)	32.0	29.0	-3.0
QOL-AD carer (52-13)	25.0	28.3	+3.3
RMBPC Memory frequency (0-28)	5.0	17.0	-12.0
RMBPC Memory reaction (0-28)	Missing	8.2*	Missing
RMBPC depression frequency (0-36)	11.0	15.0	-4.0
RMBPC depression reaction (0-36)	Missing	21.0	
RMBPC disruption frequency (0-32)	0.0*	2.0	-2.0
RMBPC disruption reaction (0-32)	Missing	0.0*	Missing
RMBPC global frequency (0-96)	16.0	34.0	-18.0
RMBPC global reaction (0-96)	Missing	29.4	Missing

* = Item imputation used due to missing data from <20% of the component scores.

At Time 2 Anne rated her sleep quality as "very good". Her PSQI score improved by two points. She no longer reported waking up in the middle of the night (less than once per week vs. three or more times per week at Time 1), she also felt more enthused in the daytime. However, she did report frequent coughing or snoring at Time 2. Despite Anne's subjective reports of improved sleep, Matthew's SDI rating indicated that her sleep was more disturbed at Time 2 compared to Time 1. At this point he reported that she had had increased difficulty falling asleep at night as well as still being sleepy in the day and waking others at night. However, her global score still remained low.

Changes in the RMBPC scores indicated deterioration in Anne's memory, as well as an increase in dementia-related behaviours at Time 2 compared to Time 1. Her MMSE score remained within the normal range. She and her son both rated her quality of life as mediocre.

Actigraphy data.

There were 11 complete days of actigraphy data available from Anne at Time 1, and eight at Time 2 (Appendix 21, *Figure A.11*). The sleep propensity curves using her data show that at Time 2 Anne was going to bed a little later and getting up earlier compared to Time 1. Her daytime sleep was relatively unchanged (*Figure 5.32*).

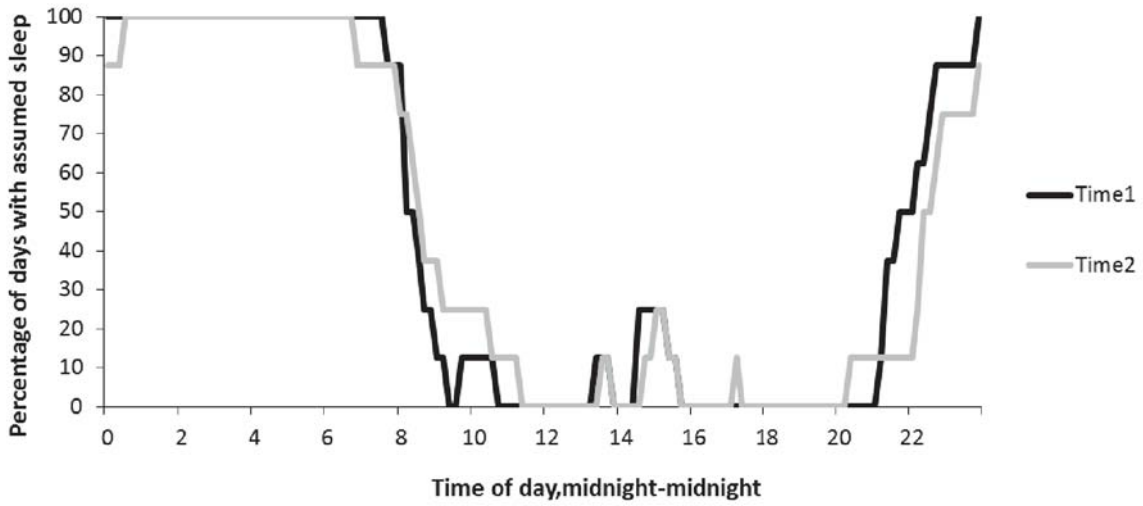

Figure 5.32. Sleep timing of PWD 6 at Time 1 and Time 2 (8 days).

Table 5.37 compares Anne's actigraphic sleep variables between Times 1 and 2. Her sleep efficiency was less than 80% at both Times 1 and 2 however her sleep did appear to improve a little at Time 2. This was indicated by her average sleep duration increasing from 7.6 hours at Time 1 to 8.2 hours at Time 2.

Table 5.37

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 6

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	22:05		22:00	(21:00-00:00)	22:27		22:25	(20:05-00:20)	+25.0
Risetime	08:39		08:30	(07:30-10:00)	09:08		08:55	(07:50-11:15)	+25.0
Time in bed night, minutes	626.4	(65.7)	620.0	(505.0-735.0)	641.9	(74.1)	645.0	(490.0-750.0)	+25.0
Total sleep per night, minutes	467.3	(49.6)	455.0	(404.0-556.0)	479.9	(72.5)	490.5	(317.0-584.0)	+35.5
Sleep efficiency (% sleep night)	74.7	(4.0)	74.6	(69.2-81.4)	74.5	(5.1)	77.2	(64.7-80.6)	+2.7
Wake time night, minutes	159.1	(34.2)	154.0	(101.0-225.0)	162.0	(28.7)	154.5	(127.0-209.0)	+0.5
Number of night awakenings	47.9	(8.5)	46.0	(35.0-60.0)	51.8	(6.1)	54.0	(41.0-60.0)	+8.0
Total sleep per day, minutes	49.3	(8.7)	51.5	(36.0-58.0)	37.2	(21.7)	29.5	(22.0-84.0)	-22.0

Results for Carer 6

Questionnaire and diary data.

Questionnaire data concerning Matthew's subjective sleep ratings as well as indicators of depression, anxiety and coping can be found in Table 5.38. Note there are no MMSE scores available from Matthew as he was not present when the researcher visited. At Time 1 Matthew rated his sleep as "fairly good". His PSQI score was raised, indicating some trouble sleeping. This was mostly related to waking up in the middle of the night or early morning, getting up to use the bathroom, feeling too cold, or being in pain (at least once or twice per week).

At Time 2 Matthew still rated his sleep as "fairly good". His PSQI score reduced to within normal range. This was due to a reduction in his night time awakenings and an increase in self-reported sleep duration. Matthew's anxiety and depression scores were within the normal range, indicating low risk. However, these scores both increased between Times 1 and 2. His scores on the positive COPE index indicated some increased risk of carer burden, this deteriorated a further two points at Time 2. At Time 1 Matthew reported that he felt "sometimes" well supported in his role as a carer, whereas at Time 2 he reported "never" feeling supported.

Table 5.38

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 6

Variable	Carer T1	Carer T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	0.0	0.0	No change
Night sleep (hrs)	6.5	7.0	+0.5
PSQI (0-21)	6.0	5.0	+1.0
Rating of nights' sleep (1-7 median, range)	3.5 (2.0-5.0)	3.0 (2.0-5.0)	+0.5
HADS-A (0-21)	4.0	6.0	-2.0
HADS-D (0-21)	0.0	3.0	-3.0
Cope positive (15-0)	9.0	7.0	-2.0
Cope negative (0-18)	4.0	4.0	No change

Short summary

Anne and Matthew were mother and son, however the study was mediated by Anne's daughter, Katie. Both Matthew and Katie worked, so during the day Anne typically had outside carers supporting her. This made conducting their study more complicated.

Anne was unable to use the light box due to recent cataract surgery however she organised her seat so that she could receive bright natural light in the mornings. They were issued with an exercise DVD which Anne reported enjoying once she'd become accustomed to it. The compliance aspect of the diaries were not maintained by Matthew or the day carers. Anne's actigraphy records only showed an increase in light exposure between Times 1 and 2.

Anne's subjective ratings of sleep improved between Times 1 and 2, whereas Matthew reported more problems. At both times, Anne's PSQI score was within the normal range. The actigraphy recordings showed some improvement in objective sleep variables. Although Anne had an average sleep efficiency of less that 80% at both Times 1 and 2, she was spending more time in bed and asleep at Time 2 compared to Time 1. She was also sleeping less during the day.

Anne had not officially been diagnosed with dementia, however suffered the symptoms, particularly short-term memory loss. Matthew reported that she had more instances of memory problems and dementia-related behaviours at Time 2 compared to Time 1. Anne was more cognitively able than the previous participants with dementia, and felt she needed to take on the study requirements herself. However, she then felt overwhelmed and anxious about maintaining the diaries and remembering to do her exercises. At each point of contact she was reminded that she could rely on her children, carers and the research team to support her with the study requirements; and that she could withdraw from the study at any time.

Matthew only completed the questionnaire and diary aspect of the study. He did not want to have his own sleep monitored. He had an increased risk carer burden which increased further at Time 2. His questionnaire showed that he had borderline sleep disruptions which improved at Time 2.

5.4.7 Pair 7 (Violet and Charlie)

Description of Study Experience

Participants.

Pair 7 consisted of a married couple. Violet was a 73-year-old woman with AD, which she was diagnosed with approximately eight years prior to the study. She had an MMSE score of 16, indicating moderate cognitive impairment. No other health problems were disclosed regarding Violet. One of Violet's medications had fatigue and difficulty sleeping as potential side effects (see Appendix 20).

Charlie was aged 78 years at the time of the study. He had been providing dementia-related support for his wife for almost three years. He provided support for her for several hours a day. He did not report having any care relief however they commented that she sometimes went to a day centre. No health issues or medications were reported for Charlie.

Data collection and intervention compliance.

At the study onset Charlie reported that they had no major sleep disruptions. However, Violet had some daytime sleepiness and was interested in taking part. Charlie had some anxiety over how much was involved with the study because Violet needed support with a lot of her activities. They were reassured that they could do as much or as little as they were able. Violet signed the consent form herself. Questionnaires regarding Violet were completed in the style of an interview, with Charlie helping Violet to answer when she struggled, and the researcher completing the paperwork. During the first week of sleep monitoring Charlie reported that he was having to do everything for Violet. She had taken the Actiwatch off one night in her sleep. He commented that sometimes it was difficult to persuade her to do things, and he was feeling anxious about the intervention phase of the study. The researcher reiterated that there was no obligation to continue with the study. The materials for the intervention were brought at the follow up home visit for the couple to consider and decide whether they wanted to continue.

They were issued with a light box as well as exercise DVD. During the trial they reported getting on well, however Violet was not able to use the light box on some days due to going to her day centre. Charlie noted that he had to fully supervise Violet when she was using the light box, otherwise she would

quickly forget what she was doing and walk away. They tried the DVD, but Violet could not concentrate for long enough. They also found some of the exercises too hard to follow or perform. They preferred the accompanying leaflet of stretches, and they already went on long walks together. Charlie did not notice any changes to sleep with using the interventions, however he reiterated that sleep was never a big problem for them in the first place.

In the diaries, it was documented that Violet used the light box on 17 out of 35 days during the 9-11am time period. On one day she used the light box after 11am. They also documented bouts of natural light between 9-11am on 10 of the 35 days. Physical activity was documented on 28 of the days. However, there was just one day when the exercise took place before 2pm. This was because of their established walking routine. Table 5.39 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods at Times 1 and 2. Violet's light exposure was within the 25th percentile at both Times 1 and 2. Despite their reports of compliance, Violet had a reduction in the amount of light exposure recorded at Time 2 compared to Time 1. Violet's median activity count between 11am-2pm was within the group's 75th percentile at Time 1 however this dropped to the middle range at Time 2. As there were limited days of data available (due to poor compliance wearing the Actiwatch) these data may not be reliable.

Table 5.39

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 7

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	1,480.3	(79.6-22,916.7)	339.4	(250.6-2,115.2)	-1,140.9
Average activity (counts/minute)	180.0	(136.6-211.4)	141.7	(105.1-178.4)	-38.3

Completion of the trial.

At the end of the trial Charlie reported feeling there was more required from him than from Violet, as he had to complete both of the diaries and make sure that she sat near the light each morning. Overall, he did not see much of a change in Violet in terms of sleep quality. However, he was very interested in seeing their actigraphy results. Charlie confirmed they had maintained their usual daily exercise routine throughout the study and preferred that to using the DVD. He reported that the sleep support handbook was informative and that he would take the advice on board, but they had not tried anything from it during the trial.

Fewer days of actigraphy were recorded at Time 2 compared to Time 1 (three versus six). This was due to a delay in a courier delivering the Actiwatches, and Violet and Charlie being about to go on holiday. Violet's actigraphy data was further limited due to the occasional night when she removed the Actiwatch. Charlie said that she found it difficult to comprehend what it was, so was inclined to take it off.

Results for PWD 7

Questionnaire and diary data.

Questionnaire data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.40. Violet slept in the same bed as Charlie. At Time 1 her sleep was rated as "very good". In the diaries, Charlie noted that Violet was sleeping pretty well and her daily sleep quality rating was good. She had a few nights interrupted to go to the bathroom, but she would go straight back to sleep afterwards. Her PSQI and SDI scores were within the normal range, she just had some problems keeping up enthusiasm to get things done once or twice per week.

At Time 2 Violet still rated her sleep as "very good". Her PSQI and SDI scores remained low, however increased in severity since Time 1. This was due to her being more sleepy in the daytime, and waking up earlier than usual. Changes to her MMSE score indicated that Violet's cognitive functioning had deteriorated between Times 1 and 2. She also had more dementia-related behaviours at Time 2, and Charlie was more affected by her symptoms (as indicated by an increase in the RMBPC reaction scores).

Table 5.40

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 7

Variable	PWD T1	PWD T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.5	0.8	+0.3
Night sleep (hrs)	9.5	9.0	-0.5
PSQI (0-21)	1.0	2.0	-1.0
SDI (0-12)	0.0	0.0	No change
Rating of nights' sleep (1-7 median, range)	1.0 (1-1)	1.0 (1-1)	No change
MMSE (30-0)	16.0	12.0	-4.0
QOL-AD PWD (52-13)	42.0	44.0	+2.0
QOL-AD carer (52-13)	40.0	37.0	-3.0
RMBPC memory frequency (0-28)	27.0	27.0	No change
RMBPC memory carer reaction (0-28)	12.0	21.0	-9.0
RMBPC depression frequency (0-36)	0.0	0.0	No change
RMBPC depression carer reaction (0-36)	0.0	0.0	No change
RMBPC disruption frequency (0-32)	0.0	2.0	-2.0
RMBPC disruption carer reaction (0-32)	0.0	3.0	-3.0
RMBPC global frequency (0-96)	27.0	29.0	-2.0
RMBPC global reaction (0-96)	12.0	24.0	-12.0

Actigraphy data.

There were five complete days of actigraphy data available from Violet at Time 1, and three at Time 2 (Appendix 21, *Figure A.12*). The sleep propensity curves using her data show that Violet had consistent sleep at Times 1 and 2, apart from a slight advance in sleep timing at Time 2 compared to Time 1.

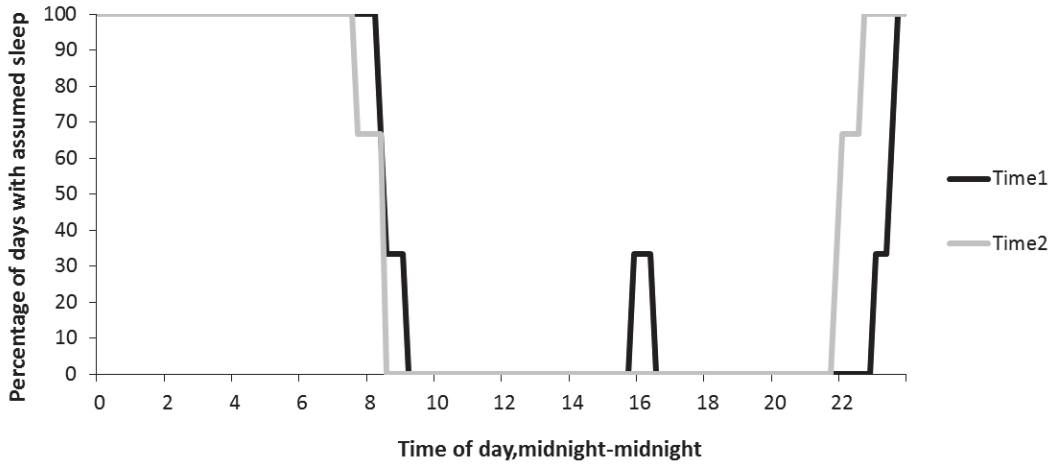

Figure 5.33. Sleep timing of PWD 7 at Time 1 and Time 2 (3 days).

Table 5.41 compares Violet's actigraphic sleep variables between Times 1 and 2. This shows that she was going to bed almost an hour and a half earlier so spending more time in bed at Time 2 compared to Time 1. However, she was only achieving a little more sleep at this time (8.1 hours vs. 7.8). Therefore, her median sleep efficiency deteriorated at Time 2.

Results for Carer 7

Questionnaire and diary data.

Questionnaire data concerning Charlie's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in **Table 5.42**. At Time 1 Charlie rated his sleep as "very good", and his daily diary ratings reflected this. His PSQI score was within the normal range. His sleep was only disturbed by some instances of coughing or snoring, as well as getting up to use the bathroom (three or more times per week). At Time 2 Charlie's PSQI score increased, but remained within the normal range. He had had occasional (less than once per week) times of waking up in the night or being too hot.

Charlie's HADS and COPE scores were all within the normal range, indicating that he had reduced likelihood for anxiety, depression, or carer burden. He appeared to be coping well with his carer role, reporting that he "always" felt well supported at both times. However, Charlie added that he sometimes felt frustrated having to repeatedly answer the same questions or keep asking Violet to do little things (to try and keep her involved and active).

Table 5.41

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 7

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:16		23:20	(22:45-23:30)	22:03		21:55	(21:45-22:30)	-85.0
Risetime	08:42		08:30	(07:45-10:00)	08:42		08:40	(08:40-08:45)	+10.0
Time in bed night, minutes	566.0	(52.1)	545.0	(495.0-640)	638.3	(20.9)	645.0	(610.0-660)	+100.0
Total sleep per night, minutes	465.6	(36.9)	467.0	(408.0-524.0)	476.3	(23.3)	487.0	(444.0-498.0)	+20.0
Sleep efficiency (% sleep night)	82.5	(4.7)	85.0	(73.4-85.9)	74.6	(1.3)	75.5	(72.8-75.5)	-9.6
Wake time night, minutes	100.4	(35.0)	86.0	(78.0-170.0)	162.0	(3.3)	162.0	(158.0-166.0)	+76.0
Number of night awakenings	36.4	(4.2)	36.0	(32.0-44.0)	39.7	(2.4)	38.0	(38.0-43.0)	+2.0
Total sleep per day, minutes	42	(0.0)	42.0	(42.0-42.0)	8.5	(7.5)	8.5	(1.0-16.0)	-33.5

Table 5.42

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 7

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.0	0.0	No change
Night sleep (hrs)	8.0	8.0	No change
PSQI (0-21)	1.0	2.0	-1
Rating of nights' sleep (1-7 median, range)	2.0 (2-5)	1.0 (1-1)	+1
MMSE (30-0)	28.0	30.0	+2
HADS-A (0-21)	0.0	1.0	-1
HADS-D (0-21)	0.0	0.0	No change
Cope positive (15-0)	14.0	14.0	No change
Cope negative (0-18)	1.0	0.0	+1

Actigraphy data.

There were seven complete days of actigraphy data available from Charlie at Time 1, and three at Time 2 (Appendix 21, *Figure A.13*). The sleep propensity curves using these data show consistent sleep timing between Time 1 and 2 (*Figure 5.34*).

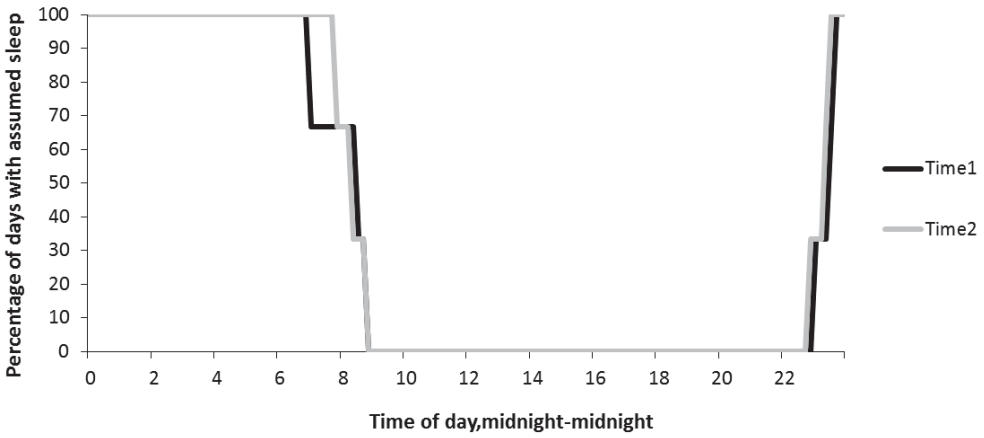

Figure 5.34. Sleep of carer 7 at Times 1 and 2 (3 days).

Table 5.43 compares the actigraphic sleep variables for Charlie between Times 1 and 2. Charlie had good quality of sleep at both times, as indicated by his spending at least 85% of his time in bed asleep and achieving at least 7.5 hours sleep. At Time 2 Charlie had a slight advance to his sleep timing and he was having more sleep at night compared to Time 1.

Table 5.43

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 7

Variable	Carer T1				Carer T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	23:13		23:20:00	(22:45-23:30)	22:45		22:44	(22:30-23:00)	-36.0
Risetime	08:15		08:20:00	(07:30-08:45)	08:33		08:45	(07:55-09:00)	+25.0
Time in bed night, minutes	542.0	(35.9)	545.0	(480.0-600.0)	588.7	(39.8)	601.0	(535.0-630.0)	+56.0
Total sleep per night, minutes	499.9	(34.7)	495.0	(455.0-562.0)	533.0	(27.9)	547.0	(494.0-558.0)	+52.0
Sleep efficiency (% sleep night)	92.3	(3.4)	93.7	(84.6-95.3)	90.7	(2.7)	92.3	(86.8-92.9)	-1.3
Wake time night, minutes	42.1	(18.9)	38.0	(24.0-84.0)	55.7	(19.3)	43.0	(41.0-83.0)	+5.0
Number of night awakenings	13.4	(2.3)	14.0	(9.0-17.0)	16.0	(5.0)	13.0	(12.0-23.0)	-1.0
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)	No change

Short summary

Pair 7 appeared to be a healthy couple. Except for Violet's AD and moderate cognitive impairment, neither had any comorbidities or many medications. They already took long walks together and were managing well with Violet's dementia-related symptoms. Sleep was not considered to be a major problem for either Violet or Charlie. This was reflected in their questionnaires as well as actigraphy data. Their actigraphy data was limited, due to a reduced number of days recorded at Time 2 and Violet's removal of the Actiwatch in her sleep. However, their sleep timing appeared consistent within the days that were recorded.

Despite Charlie's apprehensions about the interventions and their lack of sleep problems, they were keen to take part in the full study to see if there could be any improvement to Violet daytime sleepiness and dementia-related symptoms. Although they reported being compliant with the interventions, Violet's actigraphy data showed a decrease in light exposure and activity (however the actigraphy data was limited in length). Charlie commented that it was a struggle to keep Violet in front of the light box as she would lack enthusiasm or need reminding why she was using it. The majority of her physical activity was recorded as taking place in the afternoons, outside of the allotted time-frame. This was because walking was a part of their existing routine, which they found difficult to change.

Violet's actigraphy data showed minor improvements to her sleep, in that she was having more time in bed and asleep at Time 2 compared to Time 1, however she was also spending more time awake, so her sleep efficiency deteriorated. Their subjective reports were of good quality sleep at both Times 1 and 2. Violet's cognition and behavioural symptoms appeared to deteriorate between Time 1 and 2. Both of them had more a little more sleep disturbance at Time 2, due to Violet waking more at night and being sleepier in the day.

5.4.8 Pair 8 (Leonard and Phoebe)

Description of Study Experience

Participants.

Pair 8 consisted of a married couple. Leonard was a 66-year-old man with Early Onset AD, which was diagnosed approximately four years prior to the study. He had an MMSE score of 18, indicating moderate cognitive impairment. No other health problems were disclosed. He occasionally took a sleeping medication which invariably has sedating effects, as well as disruptions to sleep architecture listed as potential side effects (see Appendix 20).

Phoebe was aged 67 years at the time of the study. She had been providing dementia-related support for her husband for about an hour a day for three years. She also worked a 20-hour week and did not have any care relief. No health issues or medications were reported concerning Phoebe.

Data collection and intervention compliance.

Leonard was responsible for initiating contact with the researcher. At first it was not apparent if he had dementia or just self-diagnosed "problems with memory". He was very keen to take part, but was concerned that his wife would not be as enthusiastic because she was very busy. When the researcher visited, it was clarified that Leonard had been diagnosed with dementia and they both agreed to take part. Leonard signed the consent form himself. The intervention period was shortened by half a week due to the couple's holiday commitments. Phoebe was also due to be away for one week of the trial, but they agreed Leonard was capable of completing his own diaries and maintaining the intervention protocol.

Questionnaires were completed by Leonard, with the researcher present to clarify the questions. At the study onset Leonard reported that his sleep problem was sleeping too much, rather than too little. He was issued with a light box as well as exercise DVD. Initially Leonard reported using the light at random times of day rather than the allotted timeframe. He complained of not being able to sleep after using it on the first day. He was reminded of the importance of the routine time. He was continuing with his normal exercises and had also started playing golf some mornings or just after lunch. He read the sleep support handbook but found that there was too much information in there for him to retain or use.

Leonard had had lots of visitors and admitted he had found it difficult to maintain his diaries, especially while his wife was away. He gave the impression that he was sometimes completing it retrospectively. Later in the intervention Leonard reported using the light more successfully. He had not done as much exercise due to poorer weather.

In the diaries, Leonard documented using the light box on 13 out of 35 days. However, on four of these occasions he used the light box during the evening, and on one occasion, during the afternoon. He recorded five bouts of physical activity with no specific time frames. Table 5.44 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods at Times 1 and 2. This shows that Leonard's light exposure between 9-11am was within the group's 50th percentile at Time 1, and the 75th percentile at Time 2. His median activity count between 11am-2pm was within the 25th percentile range at both times, however an increase was recorded at Time 2.

Table 5.44

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 8

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	6,342.6	(385.1-32,872.8)	17,505.7	(351.4-68,558.7)	+11,163.1
Average activity (counts/minute)	95.4	(64.1-169.7)	124.1	(80.9-203.8)	+54.2

Completion of the trial.

At the end of the study Leonard admitted that he did not do so well with using the Actiwatch or diaries. He put this down to having lots of visitors and he and Phoebe getting ready for their holiday. He was also preoccupied with some writing work which he was doing from home. Leonard reported that Phoebe was no longer interested in having her sleep monitored. She refused to wear her Actiwatch or complete a diary at Time 2. It appeared that Phoebe was also somewhat reluctant to support his participation in the research. Leonard implied that this was because she was finding his diagnosis "difficult".

Leonard added a lot of surplus information to his diaries giving details surrounding his sleep and daytime activities that were not required of him for the study. This may have contributed to him finding

their maintenance difficult. Despite the level of detail, Leonard failed to always give times of using the light box or doing his exercises. In the feedback form, he commented that the interventions were not entirely appropriate for him at his stage, and that it was "just another thing to think about".

Results for PWD 8

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.45. Note the SDI scores were unavailable. This was because Phoebe did not complete this section of her questionnaire clearly, and was unavailable to give revisions. Leonard slept in the same bed as Phoebe. At Time 1 he rated his sleep as "very good". His daily diary rating reflected this, and his PSQI score was within the normal range. He noted that he woke up in the middle of the night or early morning or to get up to the bathroom once or twice a week, he sometimes had trouble getting to sleep, and used a sleeping medication (less than once a week). In addition, he and Phoebe noted that he had some symptoms of RLS and episodes of confusion or disorientation (less than once a week).

At Time 2 Leonard rated his sleep as "fairly good". His PSQI was still within the normal range, however his daily diary rating deteriorated. He reported sleeping less during both day and night. However, he didn't report as many instances of getting up or troubles getting to sleep at Time 2. His MMSE score improved, now indicating a mild cognitive impairment. However, changes in the RMBPC scores suggest that Phoebe noticed more dementia-related behaviours at Time 2, and these seemed to affect here more than at Time 1. She also reported that Leonard had more symptoms of depression which more strongly affected her at Time 2 compared to Time 1. Phoebe rated Leonard's quality of life more poorly than he did.

Table 5.45

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 8

Variable	PWD T1	PWD T2	Points of improvement(+) or deterioration (-)
Day sleep (hrs)	0.5	0.0	-0.5
Night sleep (hrs)	9.5	8.5	-1.0
PSQI (0-21)	4.0	3.0	+1.0
Rating of nights' sleep (1-7 median, range)	1.0 (1-3)	3.0 (1-3)	-2.0
MMSE (30-0)	18.0	21.0	+3.0
QOL-AD PWD (52-13)	40.0	38.0	-2.0
QOL-AD carer (52-13)	32.0	31.0	-1.0
RMBPC memory frequency (0-28)	25.0	26.0	-1.0
RMBPC memory carer reaction (0-28)	22.0	21.0	+1.0
RMBPC depression frequency (0-36)	14.0	18.0	-4.0
RMBPC depression carer reaction (0-36)	11.0	23.6*	-12.6
RMBPC disruption frequency (0-32)	1.0	5.0	-4.0
RMBPC disruption carer reaction (0-32)	0.0	12.0	-12.0
RMBPC global frequency (0-96)	40.0	49.0	-9.0
RMBPC global reaction (0-96)	33.0	57.0	-24.0

* = Item imputation used due to missing data from <20% of the component scores

Actigraphy data.

There were six complete days of actigraphy data available from Leonard at Time 1, and four at Time 2 (Appendix 21, *Figure A.14*). The sleep propensity curves using these data show that Leonard had consistent sleep timing at Times 1 and 2. At Time 2 Leonard was going to bed a little later at night (*Figure 5.35*).

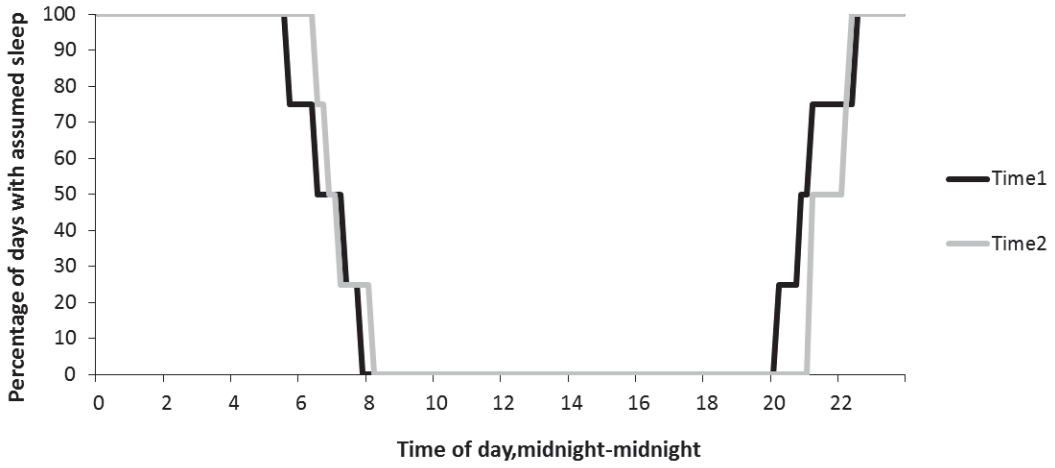

Figure 5.35. Sleep timing of PWD 8 at Time 1 and Time 2 (4 days).

Table 5.46 compares Leonard's actigraphic sleep variables between Times 1 and 2. This confirms that at Time 2 he was going to bed later. He was spending less time in bed and less time asleep at Time 2 compared to Time 1. However, his sleep efficiency and sleep duration data indicated good quality sleep at both times

Results for Carer 8

Questionnaire and diary data .

Questionnaire and diary data concerning Phoebe's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.47. At Time 1 Phoebe rated her sleep as "fairly bad". Her PSQI score and average diary sleep ratings also indicated some sleep disturbance. This was mostly related to waking up in the middle of the night or early morning, getting up to use the bathroom, and bad dreams (three or more times per week). She also mentioned that she had symptoms of RLS and general restlessness which disturbed her sleep (once or twice a week). At Time 2 Phoebe still rated her sleep as "fairly bad". However, her PSQI improved by two points. This was related to her reporting more sleep at night, and being more alert in the daytime.

Phoebe's HADS-A score was raised, indicating increased risk for anxiety at both Times 1 and 2. Her positive COPE score was also raised, indicating some carer burden. She noted that she felt supported in her role as a carer "sometimes" at Time 1 but more "often" at Time 2.

.

Table 5.46

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 8

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	20:57		20:51	(20:01-22:24)	21:36		21:35	(21:00-22:21)	-16.0
Risetime	07:03		06:56	(06:28-07:56)	07:10		07:00	(06:30-08:10)	+4.0
Time in bed night, minutes	603.3	(59.4)	599.5	(515.0-715.0)	574.5	(61.1)	568.0	(498.0-664.0)	-32.0
Total sleep per night, minutes	576.0	(59.1)	564.0	(497.0-687.0)	552.3	(64.3)	548.0	(469.0-644.0)	-16.5
Sleep efficiency (% sleep night)	95.1	(1.3)	95.5	(92.5-96.4)	96.2	(0.9)	96.5	(94.8-97.0)	+1.0
Wake time night, minutes	29.8	(7.8)	27.5	(20.0-45.0)	21.5	(3.0)	21.0	(18.0-26.0)	-6.5
Number of night awakenings	19.0	(4.2)	19.0	(14.0-27.0)	15.3	(1.5)	15.5	(13.0-17.0)	-3.5
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)	No change

Table 5.47

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 8

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.5	0.0	-0.5
Night sleep (hrs)	6.0	8.0	+2.0
PSQI (0-21)	8.0	6.0	+2.0
Rating of nights' sleep (1-7 median, range)	6.0 (3.0-7.0)	Missing	N/A
MMSE (30-0)	26.0	Missing	N/A
HADS-A (0-21)	8.0	10.0	-2.0
HADS-D (0-21)	7.0	5.0	+2.0
Cope positive (15-0)	9.0	10.0	-1.0
Cope negative (0-18)	6.0	6.0	No change

Actigraphy data.

There were seven complete days of actigraphy data available from Phoebe at Time 1, but no data available for Time 2 due to her withdrawing from this aspect of the research (Appendix 21, *Figure A.15*). Table 5.48 shows the actigraphic sleep variables for Phoebe at Time 1. This shows that she had good quality sleep, with a sleep efficiency of more than 80% and 8-10-hours sleep at night.

Table 5.48

Actigraphic Sleep Outcomes at Time 1 Concerning Carer 8

Variable	Carer T1			
	Mean	(SD)	Median	(Range)
Bedtime	21:11		21:06	(20:32-22:05)
Risetime	07:14		07:04	(06:51-08:15)
Time in bed night, minutes	602.9	(40.1)	587.0	(553.0-685.0)
Total sleep per night, minutes	544.0	(43.6)	532.0	(492.0-617.0)
Sleep efficiency (% sleep night)	90.2	(3.8)	90.6	(82.0-94.9)
Wake time night, minutes	58.9	(23.4)	55.0	(32.0-108.0)
Number of night awakenings	32.1	(7.7)	28.0	(23.0-46.0)
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)

Short summary

Pair 8 appeared to be a healthy couple. Except for Leonard's Early Onset AD, neither had any comorbidities or many medications. Phoebe only needed to provide about an hour of dementia-related support to her husband each day, therefore still worked and lived a busy life. This made her taking part and supporting Leonard's participation in the research difficult for her. Sleep was not considered to be a major problem for Leonard, however he felt he slept too much when he began the study. His questionnaires and actigraphy data showed that his sleep was within normal ranges. He had consistent and consolidated sleep timing.

Leonard was issued with a light box and exercise DVD. He used the light, however often used it at the wrong time of day and needed reminding from the researcher concerning the protocol. He preferred his established forms of exercise rather than using the DVD. He found the amount of information and content of the sleep support handbook a little overwhelming and difficult to use. Leonard's actigraphy data showed an increase in both light exposure and activity during the allotted times.

Leonard's actigraphy data showed no improvements to his sleep between Times 1 and 2. However he had high sleep duration and sleep efficiency at the outset. Leonard's MMSE score improved from moderate to mild cognitive impairment between Times 1 and 2. However, Phoebe's ratings of his mood and dementia-related behaviours indicated some deterioration. Her own questionnaires indicated that she was at increased risk of anxiety and carer burden. Phoebe rated her sleep more poorly than Leonard rated his. Her actigraphy data indicated good sleep efficiency and more than eight hours sleep per night.

This pair's study was limited by their lack of co-operation. Although Leonard only had mild cognitive impairment and could complete his questionnaires for himself, he admitted struggling to maintain his diary and use the light box at the correct times and as frequently as he should. He and Phoebe lead a busy lifestyle with lots of visitors and a holiday coming up. This distracted them from the study protocol. Although Phoebe consented to taking part, she became less committed during the study which contributed to Leonard struggling to keep up.

5.4.9 Pair 9 (Andrew and Emily)

Description of Study Experience

Participants.

Pair 9 consisted of a married couple. Andrew was an 80-year-old man with VaD, which he was diagnosed with approximately four years prior to the study. He had an MMSE score of seven, indicating severe cognitive impairment. Andrew also had heart disease, high blood pressure, as well as a stomach disease or ulcer. His heart disease was considered to limit his activities. Several of Andrew's medications had fatigue and dizziness listed as possible side effects. Some also listed insomnia or nightmares (see Appendix 20).

Emily was aged 54 years at the time of the study. She had been providing dementia-related support for her husband for almost four and a half years. She provided support all day and night, although she did have some care relief from a day care centre. Emily also had a stomach disease or ulcer, arthritis, as well as an underactive thyroid. Her arthritic condition was considered to limit her activities. One of her medications listed fatigue and insomnia as potential side effects (see Appendix 20).

Data collection and intervention compliance.

At the study onset Emily reported that they wanted to take part as they both had sleeping problems. They lived together in a small bedsit which contributed to them disturbing one another at night. They had recently moved to the Wellington region from Christchurch after the 2011 earthquakes. Emily reported that her sleep had improved somewhat since moving due to the anxiety around aftershocks which had affected her sleep.

Andrew signed the consent form himself. His questionnaires were completed in the style of an interview with the researcher clarifying questions and completing the paperwork. Their first week of actigraphy was split into two periods due to Andrew going into respite care for a few days. Beginning the intervention was further delayed due to Andrew having a fall and going to hospital for several days. The couple both had sleep disturbances surrounding this time and reported being exhausted. Therefore the actigraphy and diary data around this time were excluded from analyses. Emily initially had problems

maintaining the diaries, however she was adding a lot of surplus details about their sleep and daily activities. Once she was advised not to give so much extra information she found the subsequent diaries more straightforward.

One of Andrew's medications had photosensitivity listed as a possible side effect however his GP approved his trialling the light box. They were issued with this and an exercise DVD. They had limited space to do exercises indoors, however, they were happy going on walks which they already did together. During the initial weeks of the trial they reported using the light box. However, Andrew often slept later than 11am so they had been using it in the afternoons. They hadn't done as much walking due to poor weather and were encouraged to try the exercise DVD. Emily read the sleep support handbook and found it useful. She switched Andrew onto a decaffeinated tea and considered changing their bathroom light bulbs to a lower wattage in order to reduce the intensity of light exposure at night.

Towards the end of the trial Andrew and Emily were using the DVD on a more regular basis. They both participated and found it fun although they mentioned that the pace was a bit fast for Andrew. They reported that they were using the light more often in the mornings. Andrew and Emily had a shorter intervention period than the protocol (three weeks) due to them going on holiday and Emily being about to begin a course at university. Towards the end of their trial they also had visitors sleeping in their apartment, but Emily felt that this was no further disruption to Andrew's sleep.

In the diaries, it was documented that Andrew used the light box on 14 out of 21 days during the 9-11am time period. They also recorded bouts of bright natural light on 12 occasions but these were all outside of 9-11am. Physical activity was documented on 11 of the 21 days, however there were just five days when the exercise took place between 11am-2pm. Table 5.49 shows the light and activity data obtained from the Actiwatch during the prescribed intervention periods at Times 1 and 2. Andrew's median light exposure was within the group's 25th percentile and reduced considerably at Time 2. However he did have an increase in his median activity count at Time 2 compared to Time 1 (both of which were within the 50th percentile for the group).

Completion of the trial.

At the end of the study Emily reported that she had found it interesting. In their feedback form it was noted that they both enjoyed having the light box although they were limited for space to use it and they hadn't noticed any effects. Although they found the DVD entertaining and good exercise, they preferred their routine walks. Emily noticed that the study had made her more aware of her and Andrew's sleep habits. Taking part had allowed her to appreciate Andrew's current state of health and highlighted some changes that they might need to consider regarding their living situation with the progression of his dementia. She felt that she was now more prepared.

Table 5.49

Comparison of Light Exposure (9-11am) and Physical Activity (11am-2pm) at Times 1 and 2 for PWD 9

	Time 1		Time 2		Median change
	Median	(Range)	Median	(Range)	
Total light exposure (lux-minutes)	1,420.7	(22.6-23,442.5)	262.0	(74.1-1,077.1)	-2,698.8
Average activity (counts/minute)	135.0	(71.5-184.6)	159.2	(92.2-251.2)	+24.2

Results for PWD 9

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, memory, and dementia-related disruption can be found in Table 5.50. Andrew slept in the same bed as Emily. He had previously been diagnosed with OSA (three years prior to study). They reported that he did not have it anymore. However, no treatment was mentioned, and in the questionnaires it appears he was still snoring and having pauses in his breathing. At Time 1 he rated his sleep as "fairly good". His PSQI and SDI scores were within the normal range. However he did have some regular instances of getting up in the middle of the night or early morning, getting up to go to the toilet, as well as coughing or snoring loudly (three or more times a week). Emily also noted some periods of confusion or disorientation once or twice a week and waking up in the night. However these behaviours

were not considered too severe or distressing by Emily (hence the low SDI score). In Andrew's diaries, Emily recorded that he had lots of trips up and down to the bathroom, and she often had to actively resettle him at night.

Table 5.50

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning PWD 9

Variable	PWD T1	PWD T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	1.0	0.0	-1.0
Night sleep (hrs)	6.5	8.8	+2.3
PSQI (0-21)	3.0	5.0	-2.0
SDI (0-12)	0.1	0.0	+0.1
Rating of nights' sleep (1-7 median, range)	2.0 (2-4)	2.0 (2-5)	No change
MMSE (30-0)	7.0	6.0	-1.0
QOL-AD PWD (52-13)	39.0	32.0	-7.0
QOL-AD carer (52-13)	35.0	34.2	-0.8
RMBPC memory frequency (0-28)	25.0	21.0	+4.0
RMBPC memory carer reaction (0-28)	4.0	6.0	-2.0
RMBPC depression frequency (0-36)	3.0	1.0	No change
RMBPC depression carer reaction (0-36)	2.0	1.0	No change
RMBPC disruption frequency (0-32)	7.0	9.0	-2.0
RMBPC disruption carer reaction (0-32)	6.0	8.0	-2.0
RMBPC global frequency (0-96)	35.0	31.0	+4.0
RMBPC global carer reaction (0-96)	12.0	15.0	-3.0

At Time 2 Andrew still rated his sleep as "fairly good". His PSQI and SDI scores remained within the normal range, however increased in severity since Time 1. This was due to him having some trouble getting to sleep as well as being disturbed by Emily (less than once a week). Andrew reported that he had not been sleeping in the day at Time 2 and was having more sleep at night compared to Time 1. His MMSE and RMBPC scores indicated severe cognitive impairment and memory problems. There was some improvement in the frequency of his memory-related symptoms at Time 2. The remaining scales showed minor deterioration in Andrew's dementia-related behaviours and cognitive functioning. Emily's reactions to his symptoms were also heightened at Time 2.

Actigraphy data.

There were seven complete days of actigraphy data available from Andrew at both Times 1 and 2 (Appendix 21, *Figure A.16*). The sleep propensity curves using these data show a reduction in daytime sleep

at Time 2, he was also going to bed slightly later and getting up slightly earlier compared to Time 1 (*Figure 5.36*).

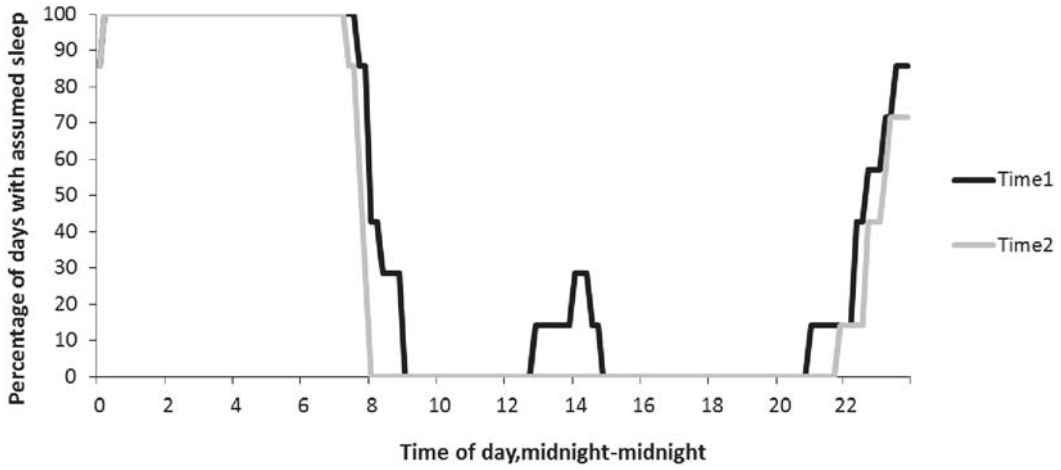

Figure 5.36 Sleep of PWD 9 at Times 1 and 2 (7 days).

Table 5.51 compares Andrew's actigraphic sleep variables between Times 1 and 2. This shows negligible changes in his sleep duration and quality, however he had reasonable sleep efficiencies at both Times 1 and 2 (≥78%) and he was getting at least eight hours sleep at night.

Table 5.51

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning PWD 9

Variable	PWD T1				PWD T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	22:34		22:40	(20:40-00:06)	00:08		23:45	(23:14-02:00)	+65.0
Risetime	08:15		08:00	(07:35-09:00)	07:45		07:45	(07:20-08:00)	-15.0
Time in bed night, minutes	581.6	(56.0)	580.0	(494.0-662.0)	552.1	(33.9)	555.0	(490.0-595.0)	-25.0
Total sleep per night, minutes	507.9	(43.4)	484.0	(461.0-584.0)	477.0	(41.9)	481.0	(406.0-542.0)	-3.0
Sleep efficiency (% sleep night)	87.5	(3.5)	88.2	(83.5-93.3)	86.3	(4.2)	87.3	(78.1-91.1)	-0.9
Wake time night, minutes	73.7	(23.6)	78.0	(33.0-105.0)	75.1	(23.3)	69.0	(53.0-127.0)	-9.0
Number of night awakenings	27.3	(7.3)	26.0	(14.0-36.0)	28.4	(4.1)	27.0	(25.0-38.0)	1.0
Total sleep per day, minutes	68.0	(12.7)	69.0	(52.0-83.0)	0.0	(0.0)	0.0	(0.0-0.0)	-69.0

Results for Carer 9

Questionnaire and diary data.

Questionnaire data concerning Emily's subjective sleep ratings as well as indicators of cognitive functioning, depression, anxiety and coping can be found in Table 5.52. A Time 1 Emily rated her sleep as "fairly good". Her average daily diary rating was mediocre. She reported that she stayed up later than Andrew in order to have some time to herself. However, Andrew was waking up several times in the night and Emily was required to provide support, so she was feeling sleep deprived.

Table 5.52

Comparison of Questionnaire and Diary Data at Times 1 and 2 Concerning Carer 9

Variable	Carer T1	Carer T2	Points of improvement (+) or deterioration (-)
Day sleep (hrs)	0.2	0.2	No change
Night sleep (hrs)	6.5	5.5	-1.0
PSQI (0-21)	9.0	9.0	No change
Rating of nights' sleep (1-7 median, range)	2.5 (2-7)	3.0 (3-4)	-0.5
MMSE (30-0)	29.0	30.0	+1.0
HADS-A (0-21)	12.0	12.0	No change
HADS-D (0-21)	9.0	11.0	-2.0
Cope positive (15-0)	10.0	8.0	-2.0
Cope negative (0-18)	8.0	7.5	+0.5

Emily's PSQI score was raised indicating some sleep disturbance. She reported instances of not being able to get to sleep within 30 minutes, waking up in the middle of the night or early morning, and getting up to the toilet (at least once or twice per week). In addition, her sleep was disturbed by her worrying, and painful joints. More rarely (less than once per week), she had trouble sleeping due to being too hot or cold, or having bad dreams. She also recorded being disturbed by Andrew's snoring, and his getting up to the toilet in the night three or more times a week. She noted that she felt their apartment was too small and that they disturbed each other throughout the night. At Time 2 Emily's PSQI remained raised due to the same reasons. She reported getting less sleep at night and rated her sleep as borderline between "fairly good" and "fairly bad".

Emily's HADS scores indicated that she was at increased risk for both anxiety and depression at Times 1 and 2. Her positive COPE score was less than 12, indicating that she was also likely to experience some carer burden. She reported "often" feeling supported as a carer at Time 1, but just "sometimes" at Time 2. In her questionnaire comments, she noted how Andrew was a good person and easy to care for, that he was "still his lovely self, without the conversation". However, by Time 2, Emily was seeking additional care assistance to help her care for Andrew as she felt she couldn't rely on family which had made her sad.

Actigraphy data.

There were seven complete days of actigraphy data available from Emily at Time 1 and 2 (Appendix 21, *Figure A.17*). The sleep propensity curves were processed using six days of data from both times to best match the days of the week (*Figure 5.37*). At Time 2 Emily was more likely to be asleep at night, particularly between 2-6am, compared to during Time 1.

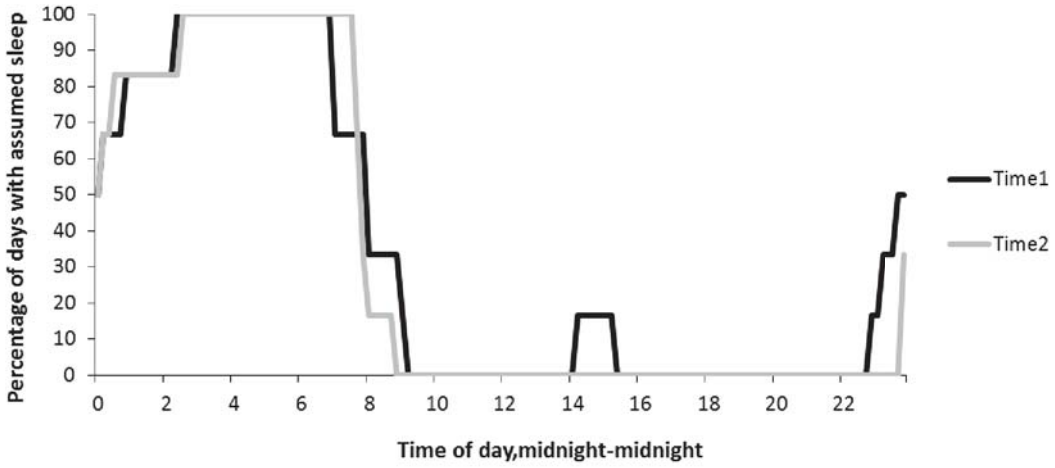

Figure 5.37. Sleep of carer 9 at Times 1 and 2 (6 days).

Table 5.53 compares actigraphic sleep variables for Emily between Times 1 and 2. Emily had reasonable quality of sleep at both times, as indicated by having at least 78% of her time in bed spent asleep. She was often going to bed after midnight, and her sleep duration was very variable at both Time 1 (range = 5.1 -9.0 hours) and Time 2 (range = 4.4-7.5 hours). However, she was getting over an hour's more sleep at Time 2 compared to Time 1.

Table 5.53

Comparison of Actigraphic Sleep Outcomes at Times 1 and 2 Concerning Carer 9

Variable	Carer T1				Carer T2				Median change
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)	
Bedtime	00:17		00:23	(22:26-02:14)	00:08		23:45	(23:14-02:00)	-38.0
Risetime	08:01		08:00	(07:00-09:10)	07:56		08:00	(07:00-08:45)	No change
Time in bed night, minutes	476.0	(105.1)	422.0	(346.0-644)	467.1	(68.0)	495.0	(330.0-540.0)	+73.0
Total sleep per night, minutes	401.7	(78.8)	363.0	(306.0-539.0)	402.6	(64.5)	436.0	(261.0-447.0)	+73.0
Sleep efficiency (% sleep night)	85.0	(3.7)	85.1	(77.6-89.9)	86.0	(3.6)	86.9	(79.1-89.9)	+1.8
Wake time night, minutes	74.3	(30.5)	63.0	(38.0-121.0)	64.6	(14.9)	65.0	(47.0-95.0)	+2.0
Number of night awakenings	26.1	(7.8)	22.0	(17.0-37.0)	24.9	(5.2)	22.0	(18.0-34.0)	No change
Total sleep per day, minutes	80.0	(0.0)	80.0	(80.0-80.0)	0.0	(0.0)	0.0	(0.0-0.0)	-80.0

Short Summary

Pair 9 were a busy couple living in a small apartment. They both had several comorbidities and Emily appeared to be suffering from stress and anxiety surrounding her coping with Andrew's care requirements, as well as taking on a paper at university. During the study they had disruptions due to friends visiting, respite care, as well as Andrew falling over and being admitted to hospital for a short time. They had a shorter intervention period due to going on holiday. Therefore they might not have received the maximum benefit of the trial. Despite these disruptions, Emily and Andrew were compliant with the study protocol. He used the light box and did exercises, however not always within the prescribed times (possibly due to routines at his day care centre). His compliance data showed an increase in activity within the allotted time frames. They also made some minor changes to their waking activities after reading the sleep support handbook.

Andrew had previously been diagnosed with OSA and still had some snoring and instances of apnoea. He was also waking up frequently. However, their subjective ratings of his sleep were fairly good. Emily reported that they disturbed one another due to their different sleep routines. At Time 2, despite their questionnaire data indicating that Andrew had more sleep at night, his actigraphy data indicated that he was having less time in bed and slightly less sleep. These changes were minor and he had reasonable sleep timing and efficiency at the outset.

Andrew had severe cognitive impairment and poor memory. His cognitive functioning deteriorated slightly between Times 1 and 2, however Emily reported less frequent problems with his memory. Emily's reactions to his dementia-related symptoms worsened, and her HADS and COPE scores indicated that she was at increased risk for anxiety, depression and carer burden. Her comments corroborated that she was experiencing some stress surrounding managing her husband's care at this time. Emily's sleep was disrupted. Her PSQI score was raised and remained so throughout the study. Her actigraphy data showed that she had very variable sleep timing, and some nights just 4-5 hours of sleep. She reported getting less sleep at Time 2 compared to Time 1, however her actigraphy data showed an improvement to her sleep duration at night.

5.4.10 Summary of Data Between Times 1 and 2

Scatter plots comparing the group's key sleep variables between Times 1 and 2 can be found in *Figures 5.38-5.45*. Equivalent plots for all other variables (including the SDI) can be found in Appendix 22. Table 5.53 shows the ranked position of the changes in the actigraphy, questionnaire, and diary variables between Times 1 and 2 for the PWD ordered by improvement in the PSQI. Improvement was determined by an increase in the following variables: Total sleep time at night, percentage sleep efficiency at night, MMSE, and QOL-AD; and by a decrease in the remaining variables: Wake time at night, number of awakenings at night, total sleep time in the day, PSQI, SDI, diary rating of sleep quality, and all RMBPC scores. This shows that the PWD with improvements to their PSQI scores (ranks 1-4) were also amongst those who had reduced wake time at night as well as improvements in their MMSE scores, and carer-rated QOL-AD.

An alternative presentation of these results ordered by position of improvement in actigraphic sleep efficiency can be found in Table 5.54. This shows those who had improvements with regards to the percentage sleep efficiency (ranks 1-3) also appear to be amongst those with the greatest improvement to total sleep time at night, carer-rated QOL-AD, and some of the dementia related behaviours. However, neither of these presentations show clear patterns of improvement, and many showed deterioration with regards to sleep and dementia-related symptoms between Times 1 and 2.

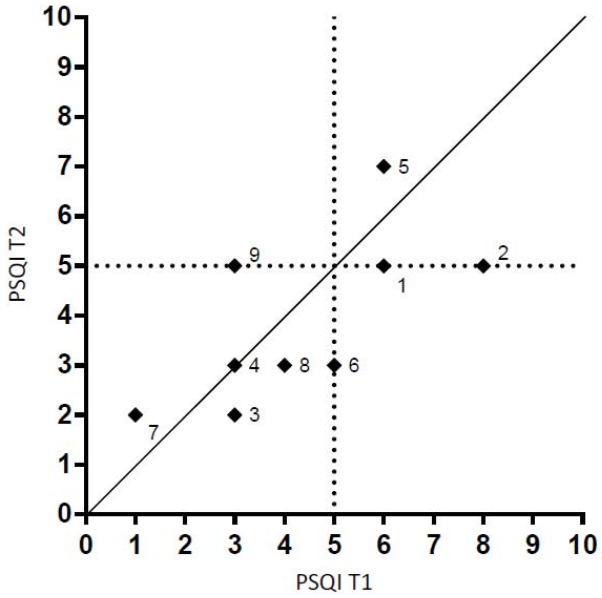

Figure 5.38. Comparison of global PSQI scores of PWD between Times 1 and 2.

Including line of no change, scores over 5 (dotted lines) indicate a sleep disturbance.

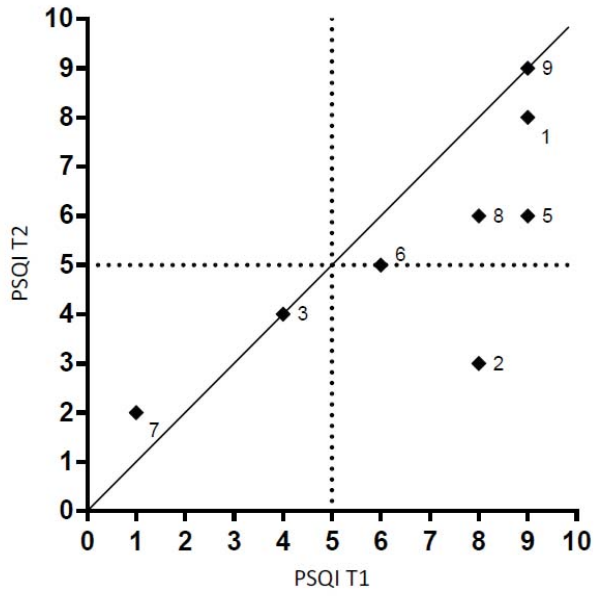

Figure 5.39. Comparison of global PSQI scores of carers between Times 1 and 2.

Including line of no change, scores over 5 (dotted lines) indicate a sleep disturbance. Note carer 4's PSQI score at Time 1 was missing.

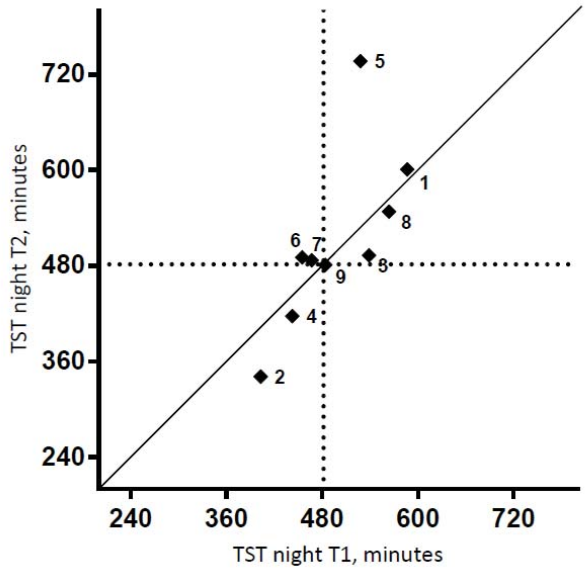

Figure 5.40. Comparison of total sleep time at night of PWD between Times 1 and 2.

Including line of no change, group median at Time 1 = 482 minutes (dotted lines).

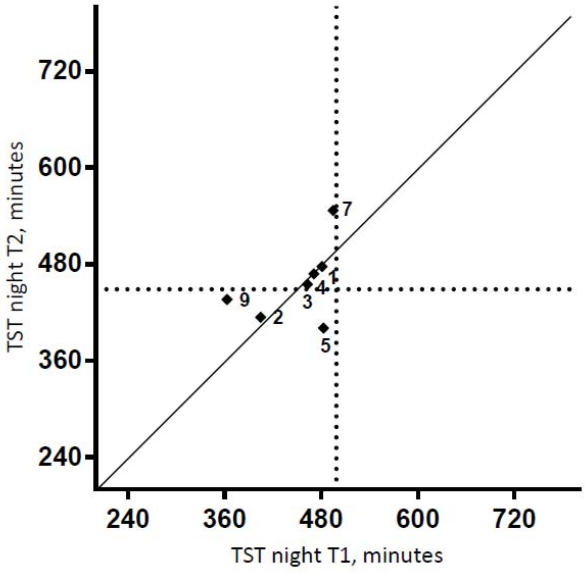

Figure 5.41. Comparison of total sleep time at night of carers between Times 1 and 2.

Including line of no change, group median at Time 1 = 449 minutes (dotted lines). Note carers 6 and 8 are not represented due to missing actigraphy data.

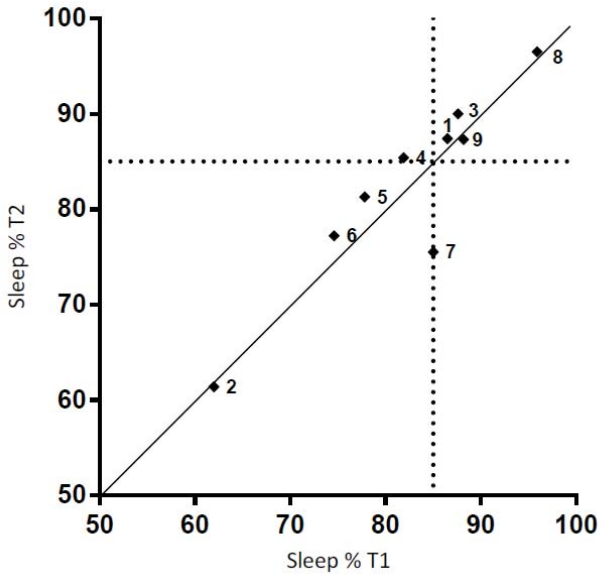

Figure 5.42. Comparison of sleep efficiency at night (sleep %) of PWD between Times 1 and 2.

Iincluding line of no change, group median at Time 1 = 85% (dotted lines).

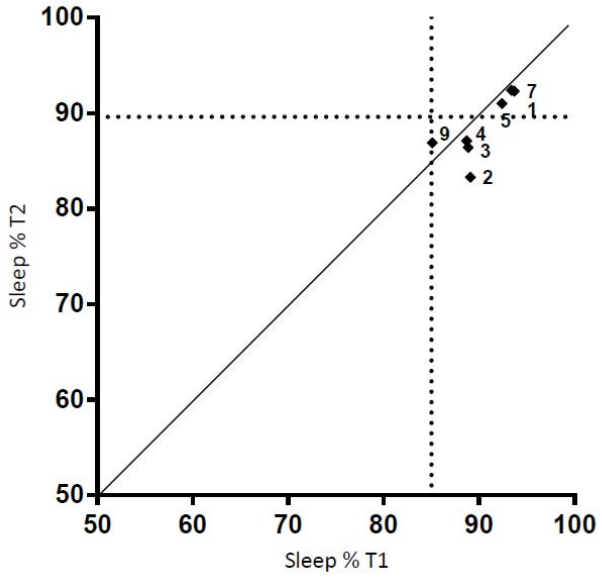

Figure 5.43. Comparison of sleep efficiency at night (sleep %) of carers between Times 1 and 2.

Including line of no change, group median at Time 1 = 89.6% (dotted lines). Note carers 6 and 8 are not represented due to missing actigraphy data.

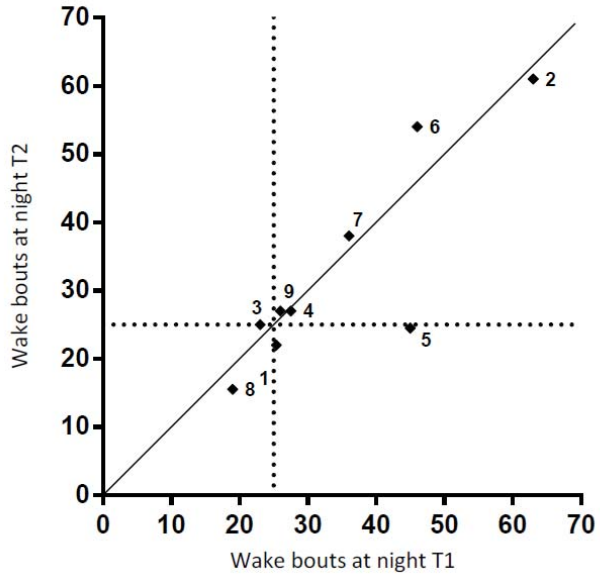

Figure 5.44. Comparison of number of wake bouts at night of PWD between Times 1 and 2.

Including line of no change, group median at Time 1 = 25 (dotted lines).

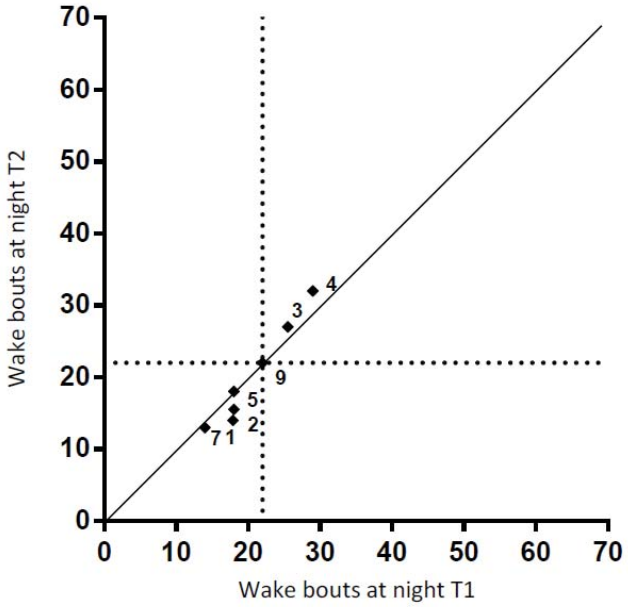

Figure 5.45. Comparison of number of wake bouts at night of carers between Times 1 and 2.

Including line of no change, group median at Time 1 = 22 (dotted lines). Note carers 6 and 8 are not represented due to missing actigraphy data.

Table 5.54

Ranked Position of Change of PWD's PSQI Scores Between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables

ID	PSQI	% sleep night	TST night	Wake time night	Awakenings night	TSTS day	SDI	Daily sleep rating	MMSE	QOL-AD, PWD	QOL-AD, carer	RMBPC memory frequency	RMBPC memory reaction	RMBPC depression frequency	RMBPC depression reaction	RMBPC disruption frequency	RMBPC disruption reaction
2	1.0	7.0∨	9.0∨	1.0	4.0	7.0∨	3.5	3.0†	8.0∨	5.0∨	4.0	3.5	7.0∨	-	-	8.0∨	4.0†
6	2.0	3.0	2.0	7.0∨	9.0∨	3.0	7.5∨	3.0†	4.5∨	6.0∨	3.0	9.0∨	-	5.5∨	-	5.0∨	-
1	4.0	4.0	4.0	3.0	3.0	5.0	7.5∨	3.0†	6.5∨	7.0∨	9.0∨	3.5	4.5∨	-	-	1.0	4.0 †
3	4.0	6.0	8.0∨	6.0	7.5∨	8.0∨	2.0	7.0∨	1.0	1.0	2.0	6.5∨	3.0∨	-	-	7.0∨	1.0
8	4.0	5.0	6.0∨	5.0	2.0	6.0†	-	8.0∨	2.0	4.0∨	7.0∨	6.5∨	1.5	5.5∨	4.0∨	9.0∨	8.0∨
4	6.0 †	2.0	7.0∨	2.0	5.0	4.0	5.5†	3.0†	3.0†	2.0	5.0†	8.0†	1.5	1.0	1.0	2.5	2.0
5	7.5∨	1.0	1.0	8.0∨	1.0	9.0∨	1.0	9.0∨	-	-	1.0	1.0	6.0∨	4.0∨	3.5 †	2.5	4.0 †
7	7.5∨	9.0∨	3.0	9.0 ∨	7.5∨	2.0	5.5†	3.0†	6.5∨	3.0	8.0∨	5.0†	8.0∨	3.0 †	3.5 †	5.0∨	7.0∨
9	9.0∨	8.0∨	5.0∨	4.0	6.0∨	1.0	3.5	3.0†	4.5∨	8.0∨	6.0∨	2.0	4.5∨	2.0	2.0	5.0∨	6.0∨

Ranked 1 (Most Improvement) to 9 (Least Improvement/Most Deterioration), † no change ∨deterioration in scale, - missing data

Table 5.55

Ranked Position of Change of PWD's % Sleep at Night Between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables

ID	% sleep night	TST night	Wake time night	Awakenings night	TSTS day	PSQI	SDI	Daily sleep rating	MMSE	QOL-AD, PWD	QOL-AD, carer	RMBPC memory frequency	RMBPC memory reaction	RMBPC depression frequency	RMBPC depression reaction	RMBPC disruption frequency	RMBPC disruption reaction
5	1.0	1.0	8.0∨	1.0	9.0∨	7.5∨	1.0	9.0∨	-	-	1.0	1.0	6.0∨	4.0∨	3.5†	2.5	4.0†
4	2.0	7.0∨	2.0	5.0	4.0	6.0†	5.5†	3.0	3.0†	2.0	5.0†	6.5∨	1.5	1.0	1.0	2.5	2.0
6	3.0	2.0	7.0∨	9.0∨	3.0	2.0	7.5∨	3.0	4.5∨	6.0∨	3.0	9.0∨	-	5.5∨	-	5.0∨	-
1	4.0	4.0	3.0	3.0	5.0	4.0	7.5∨	3.0	6.5∨	7.0∨	9.0∨	3.5	4.5∨	-	-	1.0	4.0†
8	6.0	6.0∨	5.0	2.0	6.0†	4.0	-	8.0∨	2.0	4.0∨	7.0∨	6.5∨	1.5	5.5∨	4.0∨	9.0∨	8.0∨
3	5.0	8.0∨	6.0	7.5∨	8.0∨	4.0	2.0	7.0∨	1.0	1.0	2.0	6.5∨	3.0∨	-	-	7.0∨	1.0
2	7.0∨	9.0∨	1.0	4.0	7.0∨	1.0	3.5	3.0	8.0∨	5.0∨	4.0	3.5	7.0∨	-	-	8.0∨	4.0†
9	8.0∨	5.0∨	4.0	6.0∨	1.0	9.0∨	3.5	3.0	4.5∨	8.0∨	6.0∨	2.0	4.5∨	2.0	2.0	5.0∨	6.0∨
7	9.0∨	3.0	9.0 ∨	7.5∨	2.0	7.5∨	5.5†	3.0	6.5∨	3.0	8.0∨	5.0V	8.0∨	3.0V	3.5V	5.0∨	7.0∨

Ranked 1 (Most Improvement) to 9 (Least Improvement/Most Deterioration), † = no change, ∨ = deterioration in scale, - = missing data

Table 5.56 shows the ranked position of change between actigraphy, questionnaire and diary variables between Times 1 and 2 for the carers. Improvement was determined by an increase in the following variables: total sleep time at night, percentage sleep efficiency at night, and the positive COPE score; and by a decrease in the remaining variables: Wake time at night, number of awakenings at night, total sleep time in the day, PSQI, daily diary rating of sleep quality, the negative COPE score, and both the HADS scores. The majority of carers showed deterioration within the key actigraphic variables, therefore the carers are presented in ranked order of improvement of the PSQI only. Table 5.56 shows that carers with greatest improvement to their sleep (ranks 1-3) were more likely to also to be amongst those with the greatest improvements to the number of wake bouts at night and depression scores. However there are no clear patterns of improvement amongst the group, and many showed deterioration across all measures.

Table 5.56

Ranked Position of Change of Carers PSQI scores between Times 1 and 2 with Regards to Other Actigraphy, Questionnaire, and Diary Variables.

ID	PSQI	% sleep night	TST night	Wake time night	Awakenings night	TSTS day	Daily sleep rating	HADS-Anxiety	HADS-Depression	COPE positive	COPE negative
2*	1.0	7.0 ∨	3.0	7.0 ∨	1.0	5.5†	3	3.5 ∨	2.0	4.5†	5.5†
5	2.0	4.0 ∨	7.0 ∨	1.0	2.0	5.5†	-	9.0∨	3.0	6.0∨	2.5
8	3.0	-	-	-	-	-	-	5.5∨	1.0	2.0	5.5†
1*	4.5	2.0 ∨	5.0 ∨	4.0 ∨	4.5 †	3.0 ∨	1	5.5∨	6.5∨	8.0∨	9.0∨
6*	4.5	-	-	-	-	-	6	5.5∨	9.0 ∨	8.0∨	5.5†
3	6.5†	6.0 ∨	6.0 ∨	6.0 ∨	6.0 ∨	5.5†	3	1.0	4.5∨	2.0∨	8.0∨
9	6.5†	1.0	1	2.0 ∨	4.5 ∨	1.0	5	2.0†	8.0∨	8.0∨	5.5†
7	8.0∨	3 ∨	2	3.0 ∨	3.0 ∨	5.5†	7.0 ∨	3.5	4.5∨	4.5†	2.5
4	-	2 ∨	4.0 ∨	5.0 ∨	7.0 ∨	2.0	3	5.5	6.5 ∨	2.0	1.0

Ranked 1 (Most Improvement) to 9 (Least Improvement/Most Deterioration)
* = carer of the PWD in top three ranks of most improved % sleep at night, † = no change, ∨ = deterioration in scale, - = missing data.

5.5 Summary of Findings: Study 4

In Study 4 an intervention package including BLT, therapeutic exercise, and sleep hygiene education was developed and trialled. Fifteen dyads consisting of a PWD and their family carer enrolled in the study; nine completed the six week trial. Case studies and grouped data showed that the majority of dyads found participating in the research and using the intervention feasible, informative and, in some cases, fun. Furthermore there was seldom any harm reported. Those who withdrew mostly did so because of loss of interest, unforeseen health changes, or moving into institutionalised care prior to completing the trial; rather than burden related to the research experience.

The first hypothesis for Study 4 was that PWD who took part in the intervention would have improved sleep at Time 2 compared to Time 1. Due to the smaller than anticipated sample size, this could not be statistically tested. Actigraphic data indicated that, compared to their carers, the PWD had poorer and more variable sleep timing and efficiency at both Times 1 and 2. In contrast, the self-rated PSQI sores of the PWD generally indicated less sleep disturbance than the carer group.

Six of the PWD who completed the trial had minor improvements in their sleep efficiency at night (0.6-3.6%) between Times 1 and 2. There were also cases indicating marked improvements in sleep duration and quality (e.g. PWD 5). These objective improvements were not clearly reflected in the subjective sleep ratings, measured by the PWDs' PSQI scores or carers' Sleep Disturbance Inventor (SDI) scores. Five of the PWD who completed the trial showed 1-3 points of improvement within their PSQI scores at Time 2.

The second hypothesis was that improved sleep of PWD would have positive secondary effects on dementia-related symptoms and quality of life at Time 2 compared to Time 1. Of the five PWD who had improved subjective sleep ratings at Time 2, two had improved scores on the MMSE and three had improvements to their quality of life as rated by their carer. It should be noted that, over the six week trial, several of the PWD showed deterioration with regards to their cognition and dementia-related behaviours. Six had reductions of 1-7 points in their MMSE score. Carers' reactions to dementia-related

behaviours and memory problems also increased across the trial for many cases (as indicated by the RMBPC).

The final hypotheses were that providing the sleep intervention package and improving the sleep of PWD would also improve the sleep of the carers at Time 2 compared to Time 1, and that this would have positive secondary effects on their mood and indicators of carer coping or burden. In general, carers who completed the trial had actigraphically good sleep, which remained stable between Times 1 and 2. Conversely, six out of the nine carers had PSQI scores that indicated disturbed sleep. Five had 1-5 points of improvement within their PSQI scores between Times 1 and 2. Of these five, three also had decreased ratings of depression at Time 2. The majority of carers were within the normal range for symptoms of anxiety and depression. The two who scored above the threshold for anxiety and depression at Time 1, remained so at Time 2. At Time 1, six of the carers were at increased risk for burden, as indicated by reduced scores regarding the positive impacts of caring on the COPE index. Of these six, only one showed some improvement at Time 2.

Improvements to the PWDs' sleep did not always translate into improved sleep, mood or coping of their carer. Of the three PWD and six carers who scored above the PSQI threshold for disturbed sleep at Time 1, two PWD and their carers reduced to within the normal range at Time 2 (Pairs 2 and 6).

The small heterogeneous sample limited the ability of the study to reliably measure the effectiveness of these interventions, or to identify the characteristics of PWD who were most likely to benefit. However the findings suggest that non-pharmacological interventions can be used successfully by some PWD living in the community, with positive outcomes.

6 DISCUSSION

The main aims of this thesis were to provide a better understanding of how sleep is affected for community-dwelling older people, PWD and their informal carers, as well as to address how to manage sleep problems for this population. The key questions posed were: "how are older New Zealanders sleeping?", "how do PWD and their carers personally experience sleep problems?", and "are non-pharmacological treatment options feasible and effective for community-dwelling PWD and their carers?" These were addressed via four studies, using a combination of quantitative and qualitative methods with community-dwelling older New Zealanders. The key findings, recommendations, and implications from this thesis are summarised in *Figure 6.1* and overleaf.

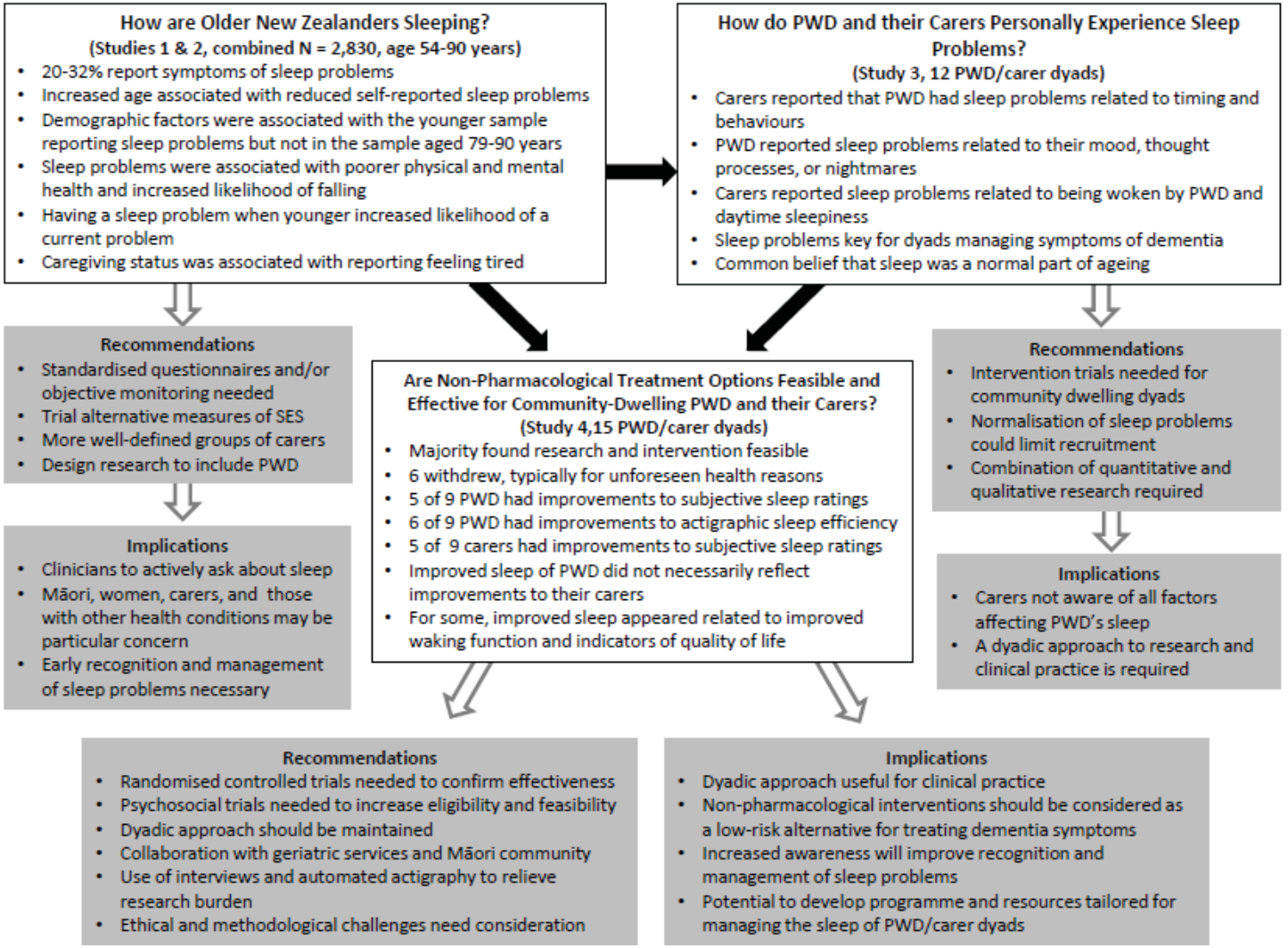

Figure 6.1. Key findings, recommendations for future research, and implications from the thesis.

6.1 Thesis Findings: Contributions to the Literature

6.1.1 How are Older New Zealanders Sleeping?

Studies 1 and 2 provide, to the best of the author's knowledge, the first estimates of the prevalence of sleep problems among older New Zealanders. These studies contribute to the growing body of knowledge addressing sleep across the lifespan in NZ. Findings corroborate relationships between sleep with physical and mental health (Foley, et al., 2004; K. Reid, et al., 2006), caregiving status (McCurry, et al., 2007), and risk of falling (Stone, et al., 2008). Most importantly, these studies indicate that the demographic factors associated with sleep problems in younger samples might not be the same for older New Zealanders, particularly for those aged 79 years or more.

Previous research with younger New Zealanders indicates a higher prevalence of sleep problems amongst Māori versus non-Māori, women versus men, with increasing socioeconomic deprivation, and with increasing age (between ages of 20-59 years, Paine & Gander, 2013; Paine, et al., 2005). The results of Study 2 were similar. Being Māori, female, or more socioeconomically deprived were independently associated with reporting symptoms of sleep problems amongst these New Zealanders aged 55-72 years (Gibson, et al., in press). However, increased age within this sample was associated in a *reduction* of reporting feeling tired or worn out. Furthermore, the sample aged 79 years or more in Study 1 reported *fewer* symptoms of sleep problems compared to the samples of New Zealanders aged less than 60 years.

Despite the fact that comorbidities associated with poor sleep become more prevalent with ageing (Buysse, et al., 1991; Ohayon, 2002; Ruiter, Vander Wal, et al., 2010), older participants in Studies 1 and 2 were less likely to report symptoms associated with poor sleep. This pattern has been noted in previous studies. In their sample of over 8,000 British participants aged 16-74 years, Arber et al. (2009) found that the likelihood of reporting sleep problems was reduced in female participants aged 65-74, compared to those aged 45-56 years (22.9% vs. 24.2%). Similarly, in a large survey of over 100,000 Americans (aged from 18 to more than 80), Grandner et al. (2012) reported decreased reports of sleep problems with those aged 80 or over compared to the 59-79 year olds. Reduced reports could be associated with the exceptional health of the study participants and a reduction of societal stressors associated with living into

advanced age. The findings from the focus groups presented in Study 3 and elsewhere (Gibson, et al., 2014; Venn & Arber, 2011, 2012) help to further explain this pattern. An apparent normalisation of sleep problems appears common and is likely to contribute to a reduction in self-reported sleep problems (Gibson, et al., 2014). With increased age and/or disease, sleep problems may not be considered as important by individuals or their family members and therefore may be less likely to be raised in clinical or research situations.

Unlike the previous studies of younger New Zealanders (Paine & Gander, 2013; Paine, et al., 2005), Study 1 found that Māori of advanced age appeared *less* likely to report sleep problems compared to non-Māori. This may be due to greater psychosocial wellbeing which is associated with being long-lived and having strong cultural engagement (Dyall, et al., 2014). Sex and socioeconomic status were not independent predictors of sleep problems for those aged 79 years or more. The reasons for these differences are unknown. In the younger sample of Arber et al. (2009), higher education and employment status were identified as independently associated with better sleep, whilst controlling for health and other demographic and socioeconomic factors. Alternative measures of socioeconomic position were explored when developing the logistic regression models for Study 1 (including educational level, previous occupation, and self-rated material and financial status). However, none of these variables improved the model fit compared to using NZDep alone. Another study, using the same cohort data as Study 1, found that demographic and socioeconomic factors were less closely related to health-related quality of life in advanced age (Dyall, et al., 2014). Sleep health might be better predicted by physical and mental health status for this age group. Future research incorporating standardised sleep questionnaires and/or objective monitoring would help to clarify these findings.

6.1.2 How do PWD and their Carers Personally Experience Sleep Problems?

The focus group findings presented in Study 3 are among the first published research of this kind. This study is unique in that it actively engaged community-dwelling PWD and their family carers in semi-structured conversations about their sleep. The themes reported were in agreement with previous reports gathered separately from carers or from PWD living in institutions (Arber & Venn, 2011; Ellmers, et al.,

2013; W. Martin & Bartlett, 2007; McCurry, et al., 1999; Simpson & Carter, 2013a), and reflect the physiological and psychological changes associated with ageing, dementia and caregiving (Chenoweth & Spencer, 1986; Klaffke & Staedt, 2006; McCurry, et al., 2007; Venn & Arber, 2012; Vitiello & Borson, 2001). The experiences and views of PWD with regard to their sleep provided new insights on the experience of what it is like to have dementia. These include descriptions by some PWD of how they cognitively struggle with getting to sleep and staying asleep, as well as the effects that nightmares and memory loss can have on their sleep and waking behaviour (Gibson, et al., 2014).

6.1.3 Are Non-Pharmacological Treatment Options Feasible and Effective for Community-Dwelling PWD and their Carers?

Previous research indicates that frequently prescribed medications for sleep disturbances have limited effectiveness and increased risks of negative side effects among older people (Glass, et al., 2005; Lee-Chiong & Harrington, 2008; McCarten, et al., 1995; Monane, et al., 1996; Salami, et al., 2011). Non-pharmacological therapies (including BLT, therapeutic exercise, and sleep hygiene education), used alone or in combination, can have positive effects on sleep. Benefits have been shown most consistently for younger adults with circadian rhythm disturbances, seasonal and non-seasonal depression, or insomnia (Chesson, 1999; Driver & Taylor, 2000; Mimeault & Morin, 1999; P. Montgomery & Dennis, 2003; Terman & Terman, 2005; Wirz-Justice, et al., 2008; Youngstedt & Freelove-Charton, 2005). Trials also indicate that PWD can benefit from such therapies, particularly BLT (Dowling, et al., 2005; Heyn, et al., 2004; King, et al., 1997; Lovell, 1995; McCurry, et al., 2005; Okumoto, 1998; Satlin, et al., 1992; Sloane, et al., 2007).

The research presented in Study 4 is important as interventions based on well-defined physiological and psychosocial processes were applied to people living in their own homes, which is steadily become a more common and preferable place for dementia care (Alzheimers New Zealand Incorporated, 2012). Community-based trials in the USA demonstrated that such interventions are feasible and have considerable potential (Colenda, et al., 1997; Friedman, et al., 2012; McCurry, et al., 2005; McCurry, et al., 2011). Study 4 builds on these studies and is, to the best of the author's knowledge, the first research of its kind to be conducted in NZ. It is also amongst the first community-based trials that

include both PWD and their carer in a case-study approach. McCurry et al. (2004) used case studies to illustrate the effects of a behavioural intervention. While carers were involved with the management of the PWD's sleep problems, their own sleep and health were not addressed in that study.

Comparing interventions in different studies.

To compare findings in different studies, it is first necessary to compare the intervention(s) used. In Table 6.1, Study 4 is compared to the previous community-based trials which included BLT (Colenda, et al., 1997; Friedman, et al., 2012; McCurry, et al., 2005; McCurry, et al., 2011). The majority of these used a light box. However, the intervention in Study 4 differed from these previous trials in that BLT was delivered in the morning, at a brighter intensity, and for a shorter duration. The exercise aspect of the intervention also had a recommended time frame unlike previous studies.

Delivering the interventions early in the day was considered potentially easier for PWD and carers, since the behaviour of PWD often tends to become more difficult towards evening, which is also a time of high care demands (evening meal and preparations for bed). Light exposure in the late morning would be predicted to advance the cycle of activity within the SCN pacemaker, and subsequently advance sleep timing (Khalsa, et al., 2003), while exercise around the middle of the day would be expected to have minimal phase-shifting effect (Buxton, et al., 2003). Independent of their phase shifting effects, regular exposure to bright light and exercise are expected to improve the stability of sleep timing and the quality of sleep via the reinforcing effects these time cues have on the activity within the SCN (Dowling, et al., 2005; Van Someren, Lijzenga, et al., 1997; Van Someren, et al., 1999). Participants in Study 4 also had the flexibility to choose to use any or all of the interventions.

Table 6.1

Comparison of Non-Pharmacological Interventions used in Study 4 with Previous Community-Based Trials

	Study 4	McCurry et al. (2005; 2011)	Friedman et al. (2012)	Colenda et al. (1997)
Light therapy device	DayLight, light box	SunRay, light box	SunBox, light box	Bio-brite Light Visor
Intensity of light	10,000 lux	2,500 lux	4,200 lux	2,000 lux
Duration of exposure	30 minutes	60 minutes	30 minutes	120 minutes
Timing of light	Within 9-11am	Within three hours of bedtime	Within 30 minutes of rising in the morning	Immediately after waking (7-9am)
Exercise type	Self-paced walking or senior fitness DVD	Self-paced walking	-	-
Duration of exercise	30 minutes	30 minutes	-	-
Timing of exercise	Within 11am-2pm	Not specified - during the day	-	-
Sleep education	Booklet with information about sleep, ageing, and dementia, plus sleep hygiene tips	Six sessions for developing and enforcing an individualised sleep plan	50-minute telephone session, manual with sleep hygiene instructions, and follow up telephone session	-
Duration of trial	Five weeks	Eight weeks	Two weeks	10 days

Comparing participants in different studies.

To compare findings from different studies, it is also necessary to consider the characteristics of the PWD studied. Table 6.2 shows that the PWD in Study 4 had similar degrees of cognitive impairment to previous community-based studies (as defined by the MMSE; Friedman, et al., 2012; Logsdon, et al., 2002; McCurry, et al., 2004; McCurry, et al., 2011; McCurry, et al., 2008; Tractenberg, et al., 2005) . However, in Study 4, the type of dementia was more variable than in previous studies and included AD, LBD, VaD, and MCI; rather than AD alone.

The memory-related symptoms of the PWD in Study 4 were rated by the carers as more frequent than but not as severe as those in the larger American validation study of the RMBPC (Teri, et al., 1992). However, the PWD in Study 4 had less frequent symptoms of depression or disruptive behaviours associated with dementia. This meant that their global frequency score was less than those in Teri et al.'s

sample (1992; mean = 30.1, SD = 11.1 vs. 33.6, SD = 16.0). These behaviours were also rated as less severe by the carers (mean global reaction score = 18.7, SD = 9.3 vs. 22.7, SD= 15.6). McCurry et al. (2006) used the RMBPC to help investigate which factors were associated with carers' reports of dementia-related sleep disturbance, amongst 46 dyads. Carers in that study rated the memory-related symptoms at the same level as in Study 4 (mean = 21, SD = 6.3 in both), however depression was more apparent in the sample of McCurry et al. (mean = 8.1, SD = 5.4 vs. 7.1, SD = 4.6). Disruptive behaviours were also more severe (mean = 8.0, SD = 4.8 vs. 4.7, SD = 4.1) than in Study 4.

The PWD in Study 4 had fewer comorbid health conditions that were considered to limit their activities, compared to the previous study by McCurry et al. (2011; mean = 1.3, SD= 1.1 vs. mean= 2.3, SD = 1.9). Just over a quarter of the PWD in Study 4 had a diagnosis of depression. This was less than expected. Cummings and McPherson (2001) reported a prevalence of 60% amongst people with VaD. The PWD in Study 4 also rated their quality of life more highly (mean = 38.5, SD = 5.9) than in previous community-based research using the same measure (mean = 35.6, SD = 5.7; McCurry, et al., 2006). Differences regarding the symptoms and health of PWD between studies could also be explained by the inclusion of participants with multiple types of dementia in Study 4.

Comparison of findings related to the PWD.

In general, the PWD in Study 4 had fewer sleep problems than previous studies. The PWD's mean PSQI score in Study 4 (5.1, SD = 2.9) is comparable to the sample of healthy Americans aged over 80 years of Buysse et al.'s study (1991; mean = 4.8, SD = 3.0). The prevalence of cases defined as poor sleepers by this scale was also similar between these studies (33.0% vs. 32%). However, previous estimates of problem sleep among British adults aged over 75 years (Venn & Arber, 2009) and amongst samples of PWD (Boeve, 2008; Vitiello & Borson, 2001) suggest a prevalence of 40-60%. Therefore the PSQI scores were lower than expected in Study 4. The PWD in Study 4 were less likely to have symptoms of sleep apnoea reported by their carer (26.7%) compared to previous estimates among PWD (40-60%; Ancoli-Israel, Klauber, et al., 1991; Hoch, et al., 1986). The PWD in Study 4were also less likely to report using a sleeping medication than the American PWD in the study of McCurry et al. (2005; 13%

vs.30%).Instead, the use of sleeping medications was the same as amongst older (aged ≥65 years) British adults (Venn & Arber, 2009).

As in previous studies (McCurry, et al., 2006; Tractenberg, et al., 2003), the sleep disturbances that carers in Study 4 were most likely to report on the SDI were that their partner with dementia was getting up to use the bathroom, sleeping excessively in the daytime, and/or waking others at night. However, Study 4 carers indicated lower frequency and severity of these behaviours than in the other studies. Therefore the global SDI scores at Time 1 were lower in Study 4 (mean = 0.6, SD = 1.2) compared to McCurry et al. (2011; mean = 1.1, SD = 0.2) or Tractenberg et al. (2003; mean = 3.6, SD = 2.2). Sundowning behaviours have been highlighted as a particular feature of dementia-related sleep disturbances (Bachman & Rabins, 2006; Khachiyants, et al., 2011; Klaffke & Staedt, 2006). Among the participants in the study of Tractenberg et al., almost 70% reported sundowning behaviours at least once a week ("wandering, pacing or getting involved with inappropriate activities at night", Tractenberg, et al., 2003, p. 334). These symptoms were rated as the most distressing for carers. In the study of McCurry et al. (2006), 39% of carers reported such problems three or more times per week. By comparison, among Study 4 participants, just 7% reported these activities occurring once or more per week.

The actigraphic sleep data of the PWD in Study 4 are compared to previous trials including community-based PWD in Table 6.2. Although the PWD in Study 4 had more objective awakenings recorded at night at Time 1 compared to the previous trials, they had longer total sleep duration at night, and less total time awake at night. Therefore the sleep efficiency of the PWD in Study 4 was greater than in previous trials, indicating they had better sleep at baseline.

The Study 4 case studies illustrate the value of considering both quantitative and qualitative information. In some cases, the combined information suggested improvements in sleep and waking function after trialling the intervention. In other cases, the rapid dementia-related deterioration masked any possible improvements or prevented participants from being able to complete the trial. For example, Pairs 2 and 5 were affected by the most severe cognitive impairment and sleep disturbances. Carers 2 and 5 were amongst the most dedicated to the research protocol and reported using the light box with their spouses as consistently as possible. Adam's (PWD 5) PSQI score deteriorated across the trial, however his

SDI score improved at Time 2. His wife, Claire, reported a marked improvement in his daytime alertness and functioning. His actigraphy results showed amongst the greatest improvement between Times 1 and 2. While Claire's actigraphic sleep did not change much between Times 1 and 2, her subjective sleep ratings improved and she reported sleeping better as she was less disturbed by Adam. Conversely, Jack's (PWD 2) actigraphy records indicated minor improvements to the fragmentation and variability of his sleep timing. His wife reported some subjective improvements. However, he also had a substantial increase of dementia-related behaviours. Therefore, for dyad 2, any potential improvements in waking function that might have resulted from improved sleep were less apparent.

Table 6.2

Comparison of Study 4 Sample and Actigraphic Sleep Data with Previous Community-Based Trials ^

	Study 4	McCurry et al. (2011)	McCurry et al. (2005)	Friedman et al. (2012)	Singer et al. (2003)
Sample size (N)	15	132	36	59	157
Dementia type	Mixed	AD†	AD†	Mixed	AD†
MMSE	18 (6)	19 (6)	12 (8)	22 (5)	14 (9)
Total sleep time, night (minutes)	488 (57)	460 (15)	462 (114)	434 (106)	351 (83)
Sleep efficiency, night (%)	83 (11)	79 (2)	82 (12)	73 (17)	69 (11)
Wake time, night	106 (68)	121 (11)	N/A	124 (98)	162 (59)
Number of awakenings, night	30 (17)	15 (1)	11 (10)	-	-
Total sleep time, day (minutes)	42 (25)	162 (16)*	84 (84)	-	151 (92)

^ all data represents rounded means (and standard deviations), † possible or probable AD
* includes periods of daytime "inactivity", - item not reported

Comparing characteristics of carers in different studies.

The sample of carers in Study 4 had relatively good sleep (by actigraphy) compared to some other groups of dementia carers. For example, carers in the study of Friedman et al. (2012) had an average sleep efficiency of 73% (SD = 17%) whereas those in Study 4 had an average of 88% (SD = 5%) at Time 1. Despite this, on the PSQI, 66% of Study 4 carers reported symptoms of sleep disturbance at Time 1. This is comparable to PSQI findings from previous studies with dementia carers (McCurry, et al., 2007; McCurry & Teri, 1995; Peng & Chang, 2013; Von Känel, et al., 2010), and indicates greater sleep

disturbances than among non-carers, or more broadly defined caregivers such as those in Study 2 and elsewhere (Gibson, et al., in press; Maher & Green, 2002). Discrepancies between subjective and objective sleep data have also been found in other samples of older carers (R. Moore, et al., 2011; Rowe, et al., 2008; Von Känel, et al., 2014) and are likely to be associated with the psychosocial impact of caring on the symptoms of insomnia.

The carers in Study 4 reported more symptoms of depression (using the HADS) than the sample of New Zealanders of advanced age in Study 1 (using the Geriatric Depression Scale; 13% vs. 8-10% respectively). However, only one carer had a diagnosis of depression in Study 4. The NZ dementia carers in the study of Roude et al. (2006) had approximately double the prevalence of anxiety and depression symptoms reported via the General Health Questionnaire (D. Goldberg et al., 1997). The Study 4 carers also differed from Roud et al.'s with regards to carer burden. Both groups reported feeling similar levels of overall support on the COPE index (McKee, et al., 2003). However, 16% of the carers in the study of Roud et al. had raised scores concerning the negative impact of caring and 7% had low positive value scores, whereas none of the carers in Study 4 had raised negative impact scores, but 47% had low positive value scores. The difference in these samples could be explained by the positive correlation Roud et al. found between scores of depression and anxiety with the scores regarding negative aspects of coping. All of the carers in Study 4 who had reduced positive coping scores also had raised PSQI scores, indicating poor sleep. The multifaceted nature of sleep disturbances and caregiving for older people suggests that accurately recognising and managing carers' sleep problems can be challenging (McCurry, et al., 2008). Further research using the COPE Index as a tool for understanding the nature of carers' sleep problems is warranted.

The mixed findings in Study 4 are concordant with previous research with PWD living in institutions or the community, using both cross-sectional studies and RCTs (Brown, et al., 2013; Forbes, 2009). Inconclusive results concerning the treatment of dementia-related sleep problems have been attributed to several factors. These include variability in comorbid diseases and demographic factors among PWD, as well as the fluctuating nature of their sleep and dementia-related behaviours. As detailed in Table 6.1 and 6.2, comparisons between studies are also confounded by differences in the measures of sleep and

the characteristics of the interventions trialled. The timing of an intervention in the course of the dementia-related deterioration , and factors necessary for stable sleep timing (including damage to the eye, pineal gland, and SCN), are also expected to affect the effectiveness of chronotherapies (Ancoli-Israel, Martin, et al., 2003; Liu, et al., 2000; Liu, et al., 1999; Valenti, 2004; Weinert, 2000; Wu, et al., 2007). For example, Ohashi et al. (1999) found that BLT trialled with older nursing home residents with and without AD was only successful in supressing the expression of melatonin during the daytime amongst those without AD.

6.2 Limitations and Recommendations

The limitations in Studies 1-3 have been presented in previous chapters. Therefore the following section considers the limitations of Study 4 and the broader limitations of the thesis as a whole. Being a pilot study, one of the aims of Study 4 was to understand the methodological challenges of conducting dementia-related research in the NZ community.

6.2.1 Recruitment Issues

The recruitment processes required for Study 4 were considered the greatest barrier to conducting this research. Despite being aware that conducting dementia-related research would be difficult, the time and effort that was required to recruit the 15 pairs for Study 4 was much greater than expected. With an estimated 1.1% of the NZ population having dementia, there are approximately 2,200 PWD in the Wellington region (Alzheimers New Zealand Incorporated, 2012). Although this estimate includes PWD living in institutions, the pool of potential participants in the Wellington region was still considered sufficient to achieve the target sample of 30-45 dyads over one year. *Figure 6.2* illustrates the distal and proximal factors identified as affecting recruitment, engagement, and retention of participants in the present research and corroborated elsewhere (Bartlett & Martin, 2002; Berghmans & Ter Meulen, 1995; Goldsmith, 1996; McKeown, et al., 2010; Whitebird, et al., 2011).

In retrospect, much of the recruitment effort was associated with building relationships with staff at the locations that were assisting with recruitment. Establishing a level of trust with those gatekeeping the access to potential participants is important. Once the research was underway, exposed more in the

media, and gathering momentum, the researcher's intentions and experience appeared to be better recognised and considered more *bona fide*. It was noted that PWD and/or carers were underrepresented at the social groups of older people where presentations and advertising was taking place. The limited social presence of this population has been associated with the stigma of dementia, physical and mental barriers to leaving the house and taking part in social situations, as well as high caregiving demands (Brækhus, Øksengård, Engedal, & Laake, 1998; Brannelly, 2011; Thommessen et al., 2002). Such limits make this a particularly challenging group to access and offer support.

Recognised factors for improving recruitment include: having endorsement from support workers or healthcare professionals, having an established track record as a researcher, and being associated with a dementia or geriatric clinic. Previous research involving PWD living in the community has generally recruited more successfully via healthcare professionals (e.g., McCurry, et al., 2011; C. Singer, et al., 2003). Future research would benefit from a formal collaboration between the SWRC and a geriatric outpatients clinic.

While there was interest in Study 4 within the dementia community, engaging people enough to take part was the challenge. The main reasons dyads did not enrol in the research were: the PWD not knowing or denying their status with regards to dementia or sleep problems, the rapid progression of dementia-related symptoms, and anxiety surrounding the time or work involved with taking part. Those who did take part seldom reported research burden. However due to the requirement for information sheets to be comprehensive, and the inclusion of unusual devices such as the Actiwatches and the light box, the protocol may have appeared more daunting than it was to those who eventually participated. By comparison, recruitment for the focus groups presented in Study 3 was much more straightforward and successful.

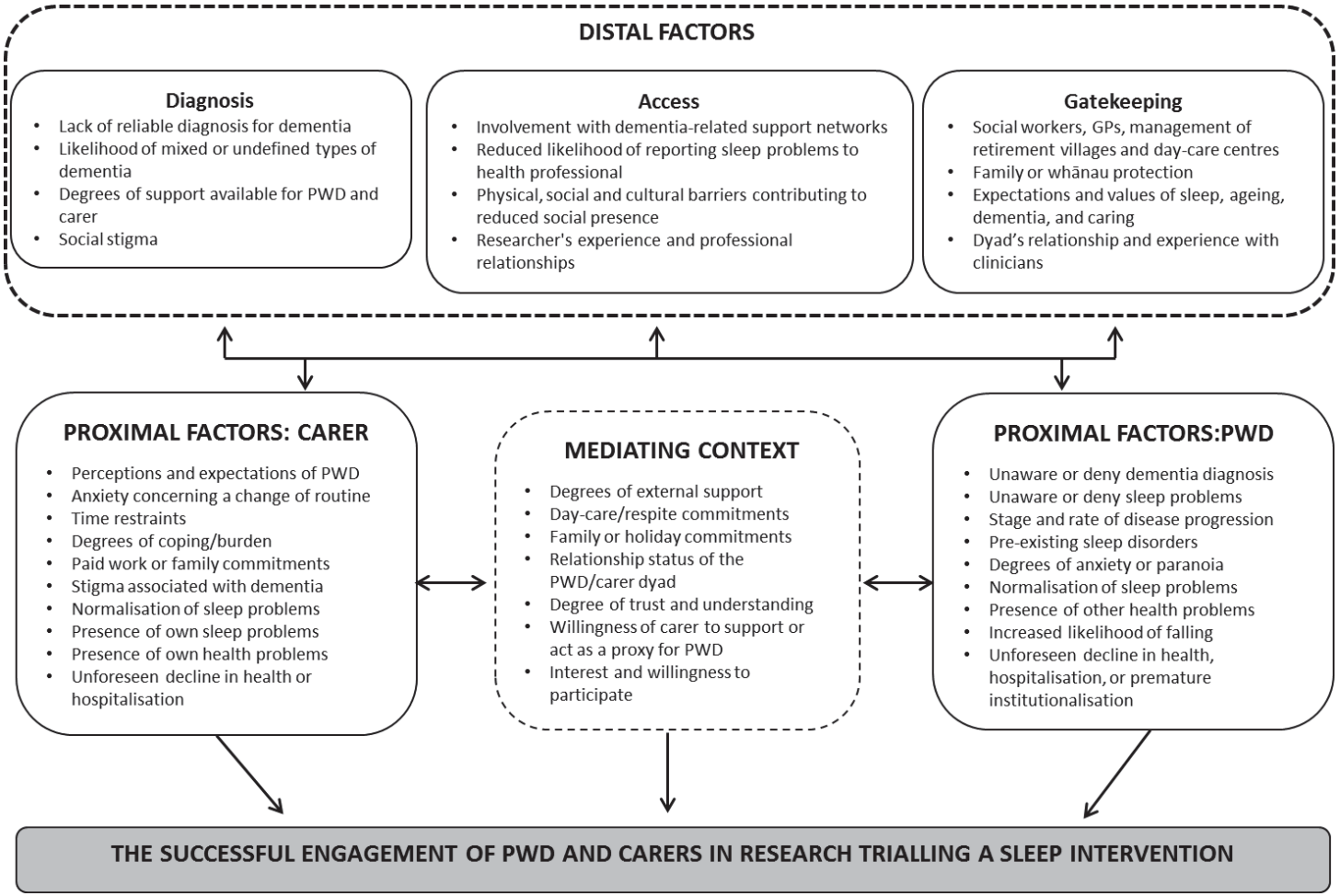

Figure 6.2. Conceptual framework showing the distal and proximal factors and the mediating context associated with the successful recruitment, engagement, and retention of PWD and carers in research.

A limitation of the Study 4 recruitment process was that potential participants who were sent study information by staff at local organisations were invited to make contact with the researcher if they were interested in participating. It may have been more effective to seek permission for the researcher to actively contact potential participants, so they could be reminded about the research opportunity, and ask questions at an earlier stage. The tactics of actively following up potential participants, as well as using multiple forms of recruitment, have been deemed useful for engaging larger samples of older New Zealanders, such as those of advanced age recruited into the longitudinal cohort presented in Study 1 (Dyall, et al., 2013).

Previous research also reports substantial challenges with recruitment of PWD and carers living in the community (Carr et al., 2010; Dowling & Wiener, 1997; Marx, Cohen-Mansfield, & Guralnik, 2003; Murphy, et al., 2007). For example, in a study proposing EEG for people with AD versus depression, Williams et al. (1988) reported a ratio of initial inquires to actual enrolment of 9:1. They noted that people with milder forms of AD and males with severe depression were particularly difficult to recruit. In their less invasive protocol, McCurry et al. (2011) reported that of the 535 PWD/carer pairs who initially inquired to take part in their sleep intervention trial, 32% declined to participate. A further 232 were excluded based on eligibility criteria. In contrast, for Study 4 it was necessary to relax the initial eligibility criteria to include family carers who were younger than 65 years and not spouses, and to include PWD with all types dementia, which increased the variability in the sample.

6.2.2 Sample Biases

Dementia status.

Study 4 included people with various forms of dementia, whilst previous studies focused on just AD (Colenda, et al., 1997; McCurry, et al., 2005; McCurry, et al., 2011; C. Singer, et al., 2003; Tractenberg, et al., 2003). This could account for some of the differences in results between studies. Four of the PWD in Study 4 had MMSE scores within the normal range for cognitive functioning, and four had undefined dementia or MCI. This probably contributed to the variability in RMBPC scores. Although these indicated impaired memory for all PWD, the depression and behavioural symptoms of dementia were less common among the PWD in Study 4 than among a larger sample of PWD (Teri, et al., 1992). Given that

AD is more frequently associated with sundowning and circadian rhythm disturbances, and that some of the hypotheses were based on the neurophysiological changes associated with AD (Vitiello & Borson, 2001; Wu & Swaab, 2007), ideally the sample would have included only people with AD, and of around the same age and stage of disease. The diagnostic pathway for AD is complex and unreliable, and many PWD have multiple forms of dementia (Gurland & Toner, 1983; Jellinger, 2006), making it difficult to recruit a homogenous sample. In addition, excluding people without a definite diagnosis would have further reduced the target population and the final sample size, as well as marginalised PWD who might have been seeking help with their sleep.

As an alternative approach, future research could consider opening up the eligibility criteria to include any older people with sleep problems. It is anticipated that this approach, together with strategic marketing through dementia-related organisations, would facilitate recruitment of a larger sample of PWD. The participants could then be analysed as a sub-sample and compared with older participants without dementia. Recruitment and consent information could thus avoid the use of dementia-related terminology. For some dyads it was the anxiety around continuously raising their dementia diagnosis that was the greatest barrier and ethical risk with regards to taking part.

Sleep status.

Previous research in the field stresses the importance of screening for sleep problems of PWD during the recruitment process (McCurry, et al., 2011; P. Montgomery & Dennis, 2004; Tractenberg, et al., 2003; van der Ploeg & O'Connor, 2014). Using brief questionnaires to record sleep problems may not be a reliable way to screen, due to issues with validity (see section 6.2.4). The exclusion of participants based on objective sleep monitoring was not within the budget of the research. It also raises an ethical dilemma of where to refer dyads who are seeking help with their sleep, but who do not have sleep problems identified by objective measurement. Tractenberg et al. (2005) suggest that the subjective perception of sleep problems should also be taken into account. The present research supported this person-centred approach. However this meant that more people were enrolled who had reasonably good sleep, but an interest in taking part or improving their sleep in general. This would have contributed to the relatively mild degree of sleep disturbance in the Study 4 participants compared to previous studies.

Health status.

When trialling a sleep-related treatment it is usual to screen participants out on the basis of comorbid health conditions, particularly other primary sleep disorders (P. Montgomery & Dennis, 2004). When including PWD in such research, it is very difficult to exclude participants on such a rigid basis, and by doing so the research sample becomes less representative of the population of interest. The most common comorbidities among PWD in Study 4 were high blood pressure and heart disease. Over a quarter of the sample also had diagnosed lung disease, depression, or arthritis. Such conditions are consistently associated with wakefulness at night and daytime sleepiness (Ancoli-Israel, Klauber, et al., 1991; Cummings & McPherson, 2001; Foley, et al., 2004; Foley, et al., 1995; Hoch, et al., 1986; Phillips & Ancoli-Israel, 2001; Wolkove, et al., 2007). The concurrent physical and mental health issues associated with ageing and dementia need to be considered for each PWD and their carer. This supports the argument for a single-case approach for research and clinical management of sleep problems in this population (Blampied, 2001; Van't Leven, et al., 2013; Vitiello & Borson, 2001).

The interventions trialled in Study 4 were mostly targeted towards the chronobiological changes that are common with dementia which have a physiological basis. These interventions may also have improved symptoms of mood disorders. However, the sleep disturbances associated with OSA, pain, or other health conditions were not expected to improve. This could explain the limited and mixed effectiveness of chronotherapies on the sleep disturbances of PWD. A much larger study would be required in order to either exclude participants or statistically control for primary sleep disorders and comorbidities associated with secondary sleep disorders.

Screening or controlling for the effects of medications on sleep is also recommended but challenging for research with older people, particularly PWD. The majority of the participants were taking medications that potentially had negative side effects on sleep, and 40% of the PWD were taking more than five medications, indicating polypharmacy (Jyrkkä, et al., 2009). With increased age and stages of dementia, the use of multiple medications increases. Instead of excluding people taking medications, it was therefore considered more appropriate to document medications and identify any substantial changes between Times 1 and 2, particularly in the use of psychotropic medications or sleeping pills.

Demographic status.

Previous NZ research, including Studies 1 and 2, indicates that Māori generally have poorer sleep health than non-Māori, except possibly among those of advanced age (Gibson, et al., in press; Mihaere, et al., 2009; Paine & Gander, 2013; Paine, et al., 2005). Studies 3 and 4 could not address differences by ethnicity because of their pilot nature and the difficulties around recruiting equal numbers of Māori and non-Māori dyads. As illustrated by Study 1, a much larger study with different recruitment strategies for Māori would be needed to address whether the interventions trialled in Study 4 would have differential benefits for Māori and non-Māori dyads.

Improving dementia care for Māori and Pacific people is a particular focus in the Alzheimers New Zealand's Dementia Strategy (2010, 2012). Kiata, Kerse and Dixon (2005) found that older Māori are less likely to be represented in institutionalised care compared to non-Māori, and more likely to be cared for by their whānau at home, including direct family members as well as the wider local community acting as informal carers. Māori are also under-represented in membership of organisations such as Alzheimers Wellington. Reduced access to formal support has been associated with communication barriers, disrespect, mistrust, as well as socioeconomic factors. This makes Māori informal or whānau carers more likely to experience caregiver burden or stress (Collins & Wilson, 2008; Jorgensen, et al., 2010; Vitaliano, et al., 1991). These factors mean that older Māori are likely to be a particularly important but challenging group to access and recruit into dementia-related research. Future research would benefit from collaboration with a Māori research team and active engagement with the Māori community, to facilitate research participation and support Māori PWD and carers.

Previous research with younger and healthier older adults has shown differences in sleep health by sex and SES (Arber, et al., 2009; Bixler, et al., 1979; Gibson, et al., in press; Ohayon, 2002; Paine, et al., 2004). The pilot nature of Studies 3 and 4 meant that it was not feasible to recruit structured samples balanced for male and female PWD and across the NZ Deprivation Index. However, Study 1 showed that these factors were not significantly associated with reporting sleep problems among people of advanced age once health status was taken into account ($p = 0.06$ and 0.30 respectively). Likewise, in previous samples of PWD, dementia severity has been found to be more strongly associated with objective or subjective

sleep status than demographic factors (Moe, et al., 1995; Tractenberg, et al., 2003). Therefore dementia status should remain the primary focus.

6.2.3 Ethical Considerations for Inclusion and Retention of Participants

Previous research has highlighted that retaining PWD in research can be challenging (Bartlett & Martin, 2002; Harris & Dyson, 2001; McCurry, et al., 2011). Study 4 prepared for this by attempting to recruit more participants than needed to detect meaningful statistical differences. However, as discussed above, the planned sample size was not achieved. The power of this already small sample was further limited by an expected attrition rate of 40%. Typically these participants withdrew due to unforeseen circumstances involving sudden changes to health leading to hospitalisation and/or moving into residential care. Those who withdrew due to lack of interest tended to be dyads who had fewer sleep problems at Time 1 and less cognitive impairment compared to those who completed the trial. The effects of paranoia and anxiety concerning research participation were evident in Pair 14's case study (see *Figure 6.2*).

A strength of Study 4 was that strategies were put in place to ethically include and retain PWD. These included alternative consent processes, gauging continued consent, and actively including PWD in data collection. In order to achieve this, a more flexible approach to data collection and use of the intervention was sometimes required. This could have reduced the comparability of data across dyads.

The method of completing questionnaires depended on the cognitive ability of the PWD. Some were able to read, understand and complete the questionnaire alone, with a little clarification from the carer; others completed the questionnaire in an interview style, with the researcher asking questions and completing the paperwork while they read along; while in instances of severe cognitive impairment, the carer would answer the majority of the questions on behalf of the PWD or complete the paperwork for them. This approach has been adopted in previous research with people with mild-moderate AD (Moran, et al., 2005). While this maintains a person-centred protocol and facilitates the retention of participants, differences between self-reporting versus proxy-reporting may have affected the internal validity of the data (Cotrell & Schulz, 1993; McCurry, et al., 2006; Tractenberg, et al., 2005). To help counter this, for each participant, the same method of data collection was used at Times 1 and 2.

It is recommended that future research adopts an interview style of collecting questionnaire data for both PWD and carers, unless the PWD is unable to answer verbally for themselves. This appeared to be the preferred and most personal method in Study 4. Using a consistent protocol for both PWD and carers should improve data reliability and participants are also treated the same irrespective of having dementia. Interviews might also reduce the high proportion of missing and anomalous data in carers' questionnaires compared to those of the PWD. This could also avoid researchers probing for answers after data collection, which could be considered unreliable and unethical.

The time of day that participants completed data collection varied between dyads. This was dependent on individual routines and personal preferences. Flexibility was available to make the research more convenient to the participants. Time of day is considered to have an impact on performance, particularly when coupled with sleep deprivation (Moe, et al., 1995; Naismith, et al., 2010; Williamson & Friswell, 2011). This could account for some of the variance between participants concerning their perceptions of sleep and psychosocial factors, as well as their cognitive functioning scores. To minimise these effects, each pair were visited at approximately the same time of day at Times 1 and 2. However, carers completed most of their questionnaires in their own time, so time of day was not controlled. The use of interviews instead of questionnaires would enable better control for such effects.

There was an ethical concern about removing the light box and exercise DVD at the end of the trial in order to loan it to the next dyad. At the end of the one-year data collection period, the equipment was offered back on loan to all study participants. Many dyads' circumstances had changed so the offer was not widely accepted.

6.2.4 Methods, Protocol, and Compliance

Subjective sleep ratings.

It could be argued that the inconclusive results of Study 4 were due to limited room for improvement, as the PWD had relatively good baseline sleep compared to previous samples (Friedman, et al., 2012; McCurry, et al., 2011; Tractenberg, et al., 2003). McCurry et al. (2011) had a similar finding with regards to carers' reports of dementia-related sleep disturbances using the SDI. Their low baseline

score (mean = 1.1, SD = 0.2) also left limited room for improvement, although there was a 0.6 point decrease in scores after the trial (SD = 0.2), this was non-significant (p = 0.23). These authors considered the actigraphic measures as more useful to measure change. The reliability of proxy reports about the sleep of PWD is also questionable (Gibson, et al., 2014; McCurry, et al., 2006).

Providing the forum for PWD to subjectively report information about their sleep was a strength of Studies 3 and 4. This should be retained in future dementia-related research. Allowing PWD, when possible, to complete their own PSQI gave them a voice in the research. Within the 15 pairs, 33% of the PWD and 73% of the carers scored above the PSQI threshold indicating disturbed sleep. However, actigraphic sleep of PWD generally indicated poorer sleep compared to their subjective ratings, and compared to carers' actigraphic sleep. Boddy et al. (2007) reported that PWD with Parkinson's disease or LBD were more likely to have raised PSQI results than people with AD. Possible reasons include the particular movement-related symptoms of the former dementia types as well as profound short-term memory loss with AD. This highlights the importance of either controlling for dementia status or using a within-subjects design.

Studies 1-3 and others indicate a tendency for older people to underreport and/or normalise sleep problems (Gibson, et al., in press; Gibson, et al., 2014; Grandner, et al., 2012; Venn & Arber, 2012). This tendency might be reflected in the low rate of reporting sleep disturbances among the PWD in Study 4. Further validation trials of sleep measures for completion by PWD and/or carers are warranted.

Actigraphy.

Overall, participants were compliant with the actigraphy protocol. The majority of PWD tolerated wearing the Actiwatch well, with only three taking the watch off intentionally or in their sleep. Participants did not report any other problems with wearing it. However, the event marker button was seldom used, particularly by PWD, who often relied on the carer to press it for them. This issue has been reported in other trials (e.g., C. Singer, et al., 2003) and means that the diary, light, and activity data are relied on for manually scoring actigraphy periods. Some participants found maintaining a diary to be a challenge, particularly if the PWD was attempting to complete it for themselves, or if additional

information was being documented in error. This reflects the importance of clarifying that participants understand the study requirements so as to avoid unnecessary work or confusion.

Consistent with previous trials, participants were instructed to wear the Actiwatch for seven nights at Time 1 and Time 2 (Friedman, et al., 2012; McCurry, et al., 2005; McCurry, et al., 2011), which was the maximum possible within the timeframe for Study 4 and equipment constraints. Van Someren (2007) recommends that at least 14 nights of actigraphic recording are collected to gain a more reliable measure of sleep. It was initially considered that a longer protocol may have been overwhelming and would make the study less appealing to potential participants. In the Singer et al. (2003) trial of supplemental melatonin, they successfully recorded actigraphy from over 150 PWD across a 10-week protocol. However carers' compliance with the sleep diaries across this time was poor, possibly due to stress. Likewise the case studies presented here indicated that, if a simpler diary was developed, wearing the Actiwatch for longer periods may not be such an issue.

The present research used manual scoring of actigraphic rest periods and software-defined variables for sleep timing and efficiency categorised by day and night. This is comparable to the methods used in McCurry et al. (2005;2011) and Friedman (2012), and made it possible to define 'good' and 'poor' sleepers as previously described (Yesavage, et al., 2003). However, these traditional sleep variables are not consistently able to detect the effects of potential treatments and rely on participants completing sleep diaries. Some carers in Study 4 commented on how difficult it could be to document their partner's daytime naps due to their high frequency and short length. For example, PWD 4 had such fitful napping that his carer could not keep track. Consequently his daytime sleep data could not be analysed using the Study 4 protocol. Likewise, periods of time awake at night were not always documented by carers, due to them not necessarily being aware of such episodes.

Actigraphic sleep is inferred from inactivity. Therefore improvements to actigraphic variables are most reliably interpreted as a reduction in night time *restlessness* rather than increased sleep per se. Due to the nature of actigraphy, together with unreliable diaries, it is likely that sleep duration and efficiency within the manually-defined rest intervals was overestimated. This has been previously reported in studies comparing actigraphy to polysomnography, which find that actigraphy is not as reliable for detecting wake

time within time in bed (Pollak, et al., 2001; Signal, et al., 2005; C. Singer, et al., 2003). The reliability of actigraphy for recognising wake further reduces in populations with insomnia or very fragmented sleep (Ancoli-Israel, Cole, et al., 2003; Littner, et al., 2003; Sivertsen, et al., 2006; Stone & Ancoli-Israel, 2011). However, due to the challenges of polysomnographic recording with PWD, actigraphy is still the preferred method for objectively monitoring sleep in this group.

It is recommended that future research considers using automated scoring of actigraphy data, so carers do not need to complete sleep diaries. Automated scoring is less reliable for calculating the traditional sleep variables such as those presented in Study 4 and elsewhere (Friedman, et al., 2012; McCurry, et al., 2005; McCurry, et al., 2011; C. Singer, et al., 2003). However, this form of data analysis may be more useful for plotting the interdaily variability and intradaily stability of PWDs' sleep/wake patterns. Van Someren et al. (1999) and others (Harper, 2010; Higgins, et al., 2010) stress the usefulness of these variables for representing the fragmentation of sleep, which is a key marker of dementia-related sleep problems and caregiving. Automated actigraphy scoring might also provide more accurate sleep propensity curves. Those presented here show assumed sleep, which was derived from the manually scored times in bed. Automated scoring might capture restlessness and sleep fragmentation more reliably, however more days of actigraphy recoding would be required. This is recommended for future studies seeking to use actigraphy to obtain objective sleep data from PWD.

Previous research using polysomnography with PWD led to the assumption that increased sleep duration or consolidation would translate into improved proportions of SWS and REM sleep stages (Prinz, Peskind, et al., 1982; Prinz, Vitaliano, et al., 1982; Vitiello & Borson, 2001). It was also assumed that improved sleep would facilitate cognitive processing, because of the associations between these sleep stages and memory and brain plasticity that have been recorded in healthy younger adults (Plihal & Born, 1997; C. Smith, et al., 2004; Stickgold, 2005). These are tenuous links, particularly for PWD who have so many confounding factors. More sophisticated methods are required to test such hypotheses, including polysomnography and tests of declarative and non-declarative memory. Incorporating such methods would be expensive, challenging, and invasive, therefore raising additional ethical challenges. Although the majority of Study 4 participants reported no difficulty with completing the questionnaires and tests, some

found the process somewhat clinical and threatening (e.g., Georgina of Pair14). The PWD may be less likely to personally benefit from taking part in more rigorous protocols. Future research needs to carefully consider which aspects of the protocol are necessary or superfluous for gaining the information required to improve the lives of PWD.

The intervention.

Compliance with the intervention was assessed by self-reported diary entries as well as comparing the light and activity data from the Actiwatches within the prescribed time frames for light exposure and exercise. Diary entries were not always consistent between or within participants and may not reliably reflect their activities if they were completed in retrospect. Research conducted in institutions has the advantage of multiple staff who can document compliance. However, community-based trials typically rely on reports from the family carer (McCurry, et al., 2003; McCurry, et al., 2005).

In order to compare light exposure between Times 1 and 2, the total amount of light (lux) recorded between 9-11am was compared. It should be noted that the sample's baseline light exposure was more than 2,000 lux across this two-hour interval, which is high compared to previous research, particularly with PWD living in institutions (Van Someren, Kessler, et al., 1997). Having a reasonable baseline of light exposure could help explain the reduced effectiveness of the intervention for some of these dyads, compared to those living in darker institutions. Previous studies have typically reported the amount of time PWD are exposed to light over a particular threshold (e.g. 1,000 or 2,000 lux) across the entire day (Campbell, et al., 1988; Higgins, et al., 2010; Sanchez, Ge, and Zee., as cited in McCurry, 2000, p. 613). This form of analyses would make comparisons between studies more reliable in the future.

The light exposure data recorded from the Actiwatch was used to compare Baseline (Time 1) and Time 2. Actigraphy data were not collected for the first four weeks of the trial, so compliance data is lacking for these weeks. The reliability of using a wrist worn device to measure bright light exposure is questionable. The light sensor is not positioned to accurately reflect light received at the eye. Previous research has indicated that Actiwatches are just as accurate at recording ambient light compared to at the eye level (Campbell, et al., 1988; Okudaira, Kripke, & Webster, 1983). However, it is possible that the light sensor was obstructed by clothing. This might explain the inconsistencies between carers' reports and

the records of light exposure in Study 4. Future research should consider alternative measures of compliance that are more valid and reduce the amount of work the carers need to invest in the protocol. For example, in the more recent RCT of McCurry et al. (2011) a light sensor was worn around the neck, which faced the light box during therapy periods. Higgins et al. (2010) used a headband-mounted light monitor, and Most et al. (2010) propose to use a sensor on the light box itself to monitor use.

In their review, Vitiello and Borson (2001) stated that BLT might be "impractical or too burdensome and expensive for use in community-dwelling patients" (p. 791). Modifications to light fittings which increase the ambient light in living areas are less intrusive. However the expense and practicality of such an installation in an individual's home versus institutions is clearly a factor. The majority of Study 4 participants reported using the light box or receiving bright natural light exposure on as many days as they could. In Study 4, none of the dyads reported that using the light box was difficult. Some commented that they had limited space in which to set the light up, and one or two of the PWD had issues with keeping their eyes open or staying seated in front of the light, however these factors did not appear to affect their desire to use it or compliance. A strength of Study 4 was the use of light boxes that had a modern design, facilitating more comfortable and effective use (Wirz-Justice, et al., 2008). The Day-Light submits a higher intensity of light than devices used in previous research (Colenda, et al., 1997; Friedman, et al., 2012; McCurry, et al., 2005; McCurry, et al., 2011), and thus it was prescribed for a shorter period, reducing the potential for discomfort or burden.

The trials took place across a year. No assessments were made for differences in light exposure and compliance with the season of data collection. However, a larger trial might benefit by controlling for this, with the expectation that BLT might be of more benefit during the winter. As yet, there is no consensus around recommended timing or intensity of BLT for PWD. This is due to the variable nature of dementia-related sleep disturbances and the other intrinsic, extrinsic, and mediating factors reviewed in Chapter 2. Alternatives are needed to the complex, highly-controlled laboratory protocols generally used to accurately measure circadian timing (Harper, 2010). A trial taking place in the Netherlands proposes to measure temperature, cortisol, and actigraphy of community-dwelling participants who have mild-moderate cognitive impairment, including early stage AD (scoring ≤14 on the MMSE). Measures will be

taken over 24-hours every six months for two years, during a trial of morning and evening light therapy for treating symptoms of depression and sleep disturbance (Most, et al., 2010). If successful, this study would offer a valuable new approach for circadian research with older people with cognitive impairments.

Activity counts measured by the Actiwatch between 11am-1pm were compared between Times 1 and 2 as an indicator of compliance with the exercise component of the intervention. Although this was useful to gauge compliance in the prescribed timeframe, many of the dyads in Study 4 reported performing their exercises at different times of day. This was usually the case for those who already had a routine involving such activities. This might explain why only 44% showed any increase in activity counts within the allotted time. Future studies would benefit from comparing activity counts across the full day instead of (or as well as) within the prescribed times, to more accurately measure changes.

The exercise DVD was rated as more challenging to use than the light box. Many dyads preferred the walking option, however this meant that compliance was often weather-dependent. Although the DVD programme was targeted for older and less able people, some of the dyads reported that the exercises became confusing or frustrating for the PWD due to lack of coordination and pace. Conversely, others found it a fun activity to do together. Future research could consider developing an exercise programme specifically targeted towards PWD, at an individual or dyadic level, in order to provide a more appropriate indoor option for therapeutic exercise.

Poor long-term compliance with interventions involving exercise, even with specialised programmes for nursing home residents with dementia, have been related to reduced willingness to take part and the behavioural symptoms of dementia (Rolland et al., 2007). In their community-based trials, McCurry et al. (2005; 2010; 2011) aimed to increase the number of days and the amount of time spent walking. Although walking significantly increased in the first weeks of the trial, compliance with the protocol declined after two and six months, in association with increasing cognitive impairment, depression, and behavioural symptoms of dementia. Dyads in which the carer was the PWD's spouse and was less stressed were also significantly more likely to be complaint across time. As with Study 4, reasons given for poor compliance noted by McCurry et al. (2010) were related to environmental factors and physical health.

Interventions which increase physical activity of PWD have been shown to have positive effects on not only sleep but also cognition, mood, wellbeing, functional abilities, strength, and balance (Eggermont & Scherder, 2006). Eggermont and Scherder's review highlights the importance of a dyadic approach for implementing activity-based treatments for dementia. Arkin (1999, 2003) proposes the potential of pairing PWD with an external exercise partner, rather than relying solely on the family carer to enforce exercises. In these studies, a student-led exercise programme was demonstrated to be a successful and cost-effective method of supervising and motivating community-based PWD to take part in appropriate exercises, whilst relieving some carer burden. Such a programme would require careful planning and surveillance, but is a promising option. Methods such as passive body heating are also being investigated as a way of mimicking the thermoregulatory effects that exercise has on the body (Driver & Taylor, 2000; Van Someren, 2000). Dorsey et al. (1996) found that hot bath has a similar effect to exercise for delaying sleep onset and increasing the proportion of SWS among female insomniacs aged 60-72 years. These results suggest that this could be an appropriate and effective alternative for people with disturbed sleep who are more physically disabled.

The choices available to Study 4 participants and the community setting would have increased the variability in exposure to light and other time cues, both within and between PWD. For example, participants who chose to exercise outside would have been exposed to additional light compared to those who were less able and exercised indoors. Likewise, those who were more socially active would have more exposure to social-mediated time cues, which are less well understood than timed light or exercise. Applying a controlled experimental protocol such as can be achieved in laboratory or institutionalised settings is methodologically challenging and raises ethical issues with this population. Individual's activities were considered likely to remain reasonably consistent across the trial and participants were asked to record any unusual activities or events in their dairies. This issue adds to the argument that research incorporating case studies or at least a within-subjects design is essential.

The effectiveness of the sleep hygiene booklet used in Study 4 was more difficult to measure. Some participants reported that it was informative and they had made changes to their routines or lifestyle. However, specific feedback was often lacking. Previous research has taken a more active approach to

enforcing behavioural changes associated with sleep (Friedman, et al., 2012; McCurry, et al., 2003; McCurry, et al., 2005; McCurry, et al., 2011). Future research might benefit from also promoting specific sleep hygiene goals and measuring individuals' achievement and adherence. However, the additional work that might be required of carers in such a protocol needs consideration. McCurry et al. (2011) found that carers reported greater research effort in a protocol combining face-to-face sleep hygiene sessions with BLT and exercise. Furthermore, the additional effects for the sleep of PWD from active sleep hygiene over BLT alone were clinically negligible. The potential of using behavioural modifications, such as used by parents with children who have sleeping problems, are also being considered for PWD and carer dyads (Gallagher, Odenheimer, & Kunik, 2011). For example, gradual extinction of the carer's presence or time spent soothing could help PWD to re-learn self-settling techniques. However, such techniques are also likely to create additional burden on carers (at least at first) and might only be realistic for people with mild forms of dementia.

Sleep hygiene could be particularly useful for carers' own sleep. Friedman et al. (2012) incorporated the Blake-Gomez Sleep Hygiene Questionnaire (Blake & Gómez, 1998) in their community-based trial of BLT with sleep hygiene education versus dim light with sleep hygiene education ($p = <0.05$). This helped to measure behavioural modifications related to sleep. They found that the carers in both conditions had an increase in healthy sleep behaviours. Both groups showed improvements to sleep measures (wake after sleep onset, sleep efficiency, and reported symptoms of insomnia), with a possible mediating effect of reduced symptoms of depression. McCurry et al. (1998) found that a multifaceted behavioural treatment improved the sleep of carers with regards to PSQI scores and sleep efficiency. However, in their study, measures of mood and burden remained unchanged. This research indicates the potential that sleep hygiene information alone can have for dementia carers. Future research should emphasise the administration and monitoring of this aspect of the intervention for community-dwelling dyads. Refresher sessions may be necessary to improve durability of the key messages and strategies over time (P. Montgomery, 2002; P. Montgomery & Dennis, 2004).

Using a more flexible approach increased the possibility of a Hawthorne effect (Wickstrom & Bendix, 2000). During Study 4, there were three or four home visits from a researcher, as well as weekly

telephone calls. Increased attention, novelty value of the light box and DVD, and the social nature of data collection, may have contributed to a short-term improvement in subjective questionnaire ratings. Some consider that participation effects might not be as great amongst PWD compared to other populations (e.g. cancer patients), due to the poor prognosis of dementia and lack of successful treatments (Iliffe, et al., 2008). However, such effects have been previously reported in a RCT of ginkgo biloba for PWD. McCarney et al. (2007) found that more intensive follow-up procedures were associated with increased scores of cognitive functioning compared to those with minimal follow up. The Hawthorne effect is a complicated limitation for dementia-related research. Typically, further follow-up studies would be used in order to measure continued effects after the main research protocol. However, the rapidly changing nature of dementia-related diseases makes this less possible or reliable.

6.2.5 Recommendations

Study 4 was exploratory and the findings were expected to be complex, due to the nature of the population. The findings and limitations described above provide a foundation for future research. Two approaches are recommended for trialling therapies for managing dementia-related sleep problems for older New Zealander: a biomedical approach and a psychosocial approach.

A biomedical approach.

The accepted standard for demonstrating that non-pharmacological interventions are effective for PWD and their carers would be to conduct an RCT. This approach would require greater funding, time, and staff than was available in Study 4. The research would need to be conducted through multiple centres, health-care professionals, and regions of NZ in order to achieve the necessary sample size. Mixed methods would be helpful to examine differences between subjective, proxy, and objective sleep measures (Higgins, et al., 2010; Tractenberg, et al., 2005). An RCT would enable the use of repeated-measures analysis of variance to compare participants' data between time points, while controlling for possible confounding factors.

While it is appealing to propose such research, previous reasonably-powered RCTs in institutional and community settings have still had inconclusive results (Brown, et al., 2013; Forbes,

2009). For example, Singer et al. (2003) used a multi-centred and medical approach to recruit community-dwelling Americans with AD and their carers into a trial using two doses of Melatonin versus placebo. They successfully recruited the target sample of 50 participants in each arm, however results were equivocal. They concluded that a larger sample would be required for a reliable trial. Likewise, McCurry et al. (2011) successfully recruited 132 dyads and found that their interventions had a positive effect on the wake time at night of the PWD, but also concluded that more participants would be required to reliably compare which aspect of the intervention (i.e., BLT, exercise, sleep hygiene, or a combination of all three) is most effective.

Inconclusive results for dementia-related sleep therapies (pharmacological and non-pharmacological) are often associated with high variance within the group, particularly in the type and stage of dementia, the extent of sleep problems, comorbidities, and medication use. Sleep and dementia symptoms have also been found to fluctuate substantially within individual PWD. This makes them a less reliable group to compare the influence of treatments between times (Eeles, 2006). Very large sample sizes would be required to be able to control for all these different sources of variance.

Since sleep timing is so variable, attention should be paid to attempting to measure circadian phase (e.g., via core body temperature) in order to accurately prescribe light at the most effective time of day for each individual (Harper, et al., 2001; Satlin, et al., 1995). This quantitative approach is scientifically desirable for determining the effectiveness of interventions to improve sleep (Brown, et al., 2013; Forbes, 2009). However, such studies are considered less appropriate for an older population affected by dementia (Clarke & Keady, 2002; W. Martin & Bartlett, 2007; Wilkinson, 2002). Based on the experiences of recruitment, sampling, and retaining participants in Study 4, the ability to apply more rigorous exclusion criteria and protocols would likely be challenging. Furthermore, overly long or demanding protocols could deny some PWD the opportunity to trial a therapy which, even for just a short window of time, they might find beneficial. It could be more ethical to make such interventions available to all.

A psychosocial approach.

Limited numbers of people with cognitive impairment or formal caregiving duties engaged in the longitudinal and empirically designed research presented in Studies 1 and 2. Older people affected by dementia are a unique and variable group who are difficult to access and recruit into research, which creates challenges for rigorous quantitative study design and statistical approaches. The final sample in Study 4 was too small and variable for reliable statistical analysis. Considering the variability among PWD in Study 4, the original target sample of 30-45 dyads might still have been insufficient to find significant statistical differences in measures of sleep and waking function between Time 1 and Time 2. Instead, the data was presented on a case-by-case basis. Ultimately this provided a more comprehensive understanding of each dyad's experiences and stories. These factors may have been be overlooked or over-generalised by using inferential statistics. This thesis indicates that the subjective and qualitative reports of community-dwelling PWD and their carers offer a rich adjunct or potentially an alternative to complex and burdensome studies using objective measures.

It is recommended that future research embarks on more single-case psychosocial research (Blampied, 1999, 2001). Combining quantitative with qualitative methods can provide rich case studies, including multiple observations across experimental phases for the PWD (or dyad). This design would allow for some causal inferences to be made regarding independent variables and changes with the intervention. A strength of using a single-case design is that the research can be conducted at the level of the dyad, which is becoming the recommended process for understanding sleep disturbances and trialling interventions amongst PWD (Kotronoulas, et al., 2013; Van't Leven, et al., 2013). A single-case approach also allows for the uniqueness of the participant to be recognised. The results are more applicable to the participants than generalised group results.

To improve participation and to keep dementia-related research more person-centred, future trials of sleep interventions should focus more on the dyad and incorporate qualitative as well as quantitative methods. For example, this could include subjective ratings or the individual's dialogue regarding sleep and affect, combined with automated scoring of actigraphic data. The findings from Study 3 and others (Arber & Venn, 2011; Ellmers, et al., 2013; W. Martin & Bartlett, 2007; Simpson & Carter,

2013a; Venn & Arber, 2011, 2012) suggest that semi-structured interviews reveal the sociological experience of sleep, which is relevant to each individual and dyad. Such sociological inquiry may be key to understanding the sleep of older people: The "biographical relays between continuity and change, constitute a major vantage point for future medical sociological research on health, ageing and the life course" (Bury, 2000; summarised by S. Williams, 2005, p. 90). A key finding from previous research is that sleep disturbances contribute to PWD being admitted into institutionalised care (Hope, et al., 1998; Pollak & Perlick, 1991). While objective sleep measures are useful to confirm relief of clinically-defined sleep disruptions, it is argued that the PWD/carer dyad also need to experience subjective improvements to sleep, waking function, as well as their sociological wellbeing in order to be able to extend their time living together at home.

Psychosocial models for dementia care and research are person-centred and focus on the adaptation of the psychosocial and/or material environments of PWD (Finnema, Dröes, Ribbe, & Van Tilburg, 2000; Kitwood, 1989). Adopting such an approach is considered useful for supporting PWD and carers in implementing an intervention for improving their sleep, during a time in which they are also coping and adapting to the additional consequences of dementia. It is desirable to reach dyads affected by dementia before sleep becomes too problematic, and before behaviours are developed which contribute to perpetuating sleep problems that are more difficult to resolve (Spielman, et al., 1987). Increased awareness via dissemination and resource development could empower individuals to assess their own sleep and that of their partner, develop goals for improving sleep, as well help dispel the common belief that sleep problems are a normal and acceptable part of ageing and dementia.

6.3 Implications

Considered together, the results from the studies presented in this thesis confirm that with ageing, dementia, and caregiving come particular sleep disruptions. However, such sleep disruptions might be underreported by this population. This is a particular concern for PWD living in the community, due to the associations between sleep and cognitive functioning, health, mood, behaviour, as well as caregiving relationships, and quality of life (Altena, et al., 2010; Hauri, 1997; Jimenez, et al., 1989; Lichstein, et al., 2001; Vitiello & Borson, 2001; Wolkove, et al., 2007). It is important to treat sleep problems of PWD to

help prevent exacerbation of dementia-related symptoms, functional decline, and premature admission to long-term institutionalised care (Ancoli-Israel, Klauber, Gillin, Campbell, & Hofstetter, 1994; Pollak & Perlick, 1991; Song, et al., 2010).

6.3.1 Research Implications

The current National Dementia Strategy for NZ identifies the need for research to improve the quality of dementia care practices. Psychosocial approaches to delaying institutionalisation have been highlighted as key for reducing the economic and social impact of dementia (Alzheimers New Zealand Incorporated, 2008, 2010). The present research fits within this strategy by contributing to the knowledge and management of age and dementia-related sleep problems of PWD and their carers living in the community.

The research presented in Studies 3 and 4 highlights the ability to successfully include PWD in the research processes from planning to completion. It is a unique example of how a person-centred approach can be applied to dementia-related sleep research. This has implications for future research and dementia care, which is and should be moving into a more holistic and person-centred philosophy (Downs, 1997; Innes, 2009; McKeown, et al., 2010; T. Moore & Hollett, 2003; Wilkinson, 2002). The psychosocial considerations outlined above highlight the potential value of testing a hybrid research approach. This could increase the chances of helping people at a time when they need it, whilst also formally assessing the benefits from trials of non-pharmacological interventions. This would involve PWD and their carers being offered the interventions as a primary care option, with data collection limited to short-form questionnaires administered by interview and/or actigraphy without diaries.

The methodological and ethical challenges highlighted in this thesis prompted a fifth study which is not presented here, given the scope of Studies 1-4. Interviews with 11 academics and clinicians who have experience with dementia-related research were conducted around the biannual Alzheimers New Zealand conference in 2012. Nine of the participants were NZ based. Interviews were semi-structured and addressed their experience of methodological and ethical challenges with conducting their research, as well as how they overcame them. This project was met with much enthusiasm from the participants, who

overall reported similar struggles to those reported in Study 4. Many seemed to appreciate a forum to discuss their research experience. Likewise the interviews provided external support and advice for the researcher during the final stages of data collection. These interviews have been transcribed and thematic analysis will be used to highlight the key barriers to conducting research with PWD, and recommendations to help overcome them. The results from these analyses, together with the specific ethical and methodological challenges encountered in Studies 3 and 4, will be disseminated via publication in order to help inform future dementia-related research both locally and internationally.

6.3.2 Clinical implications.

The clinical implications of Study 4 are somewhat speculative due to the limitations mentioned above. However, the case studies revealed that using BLT and/or therapeutic exercise with sleep hygiene education is feasible for PWD and carers at home, with limited burden. Given the insufficient options for treating dementia, it is recommended that these low-risk interventions are considered by healthcare professionals and support workers for managing the sleep and wellbeing of PWD. They could be implemented either as an alternative or in conjunction with pharmacological treatments.

At this time, specific recommendations cannot be made and healthcare professionals are left to make clinical decisions about prescribing such interventions. However, given the reduced chance of adverse side effects compared to pharmacological options (Cloyd & Conway, 2008; Glass, et al., 2005; Krystal, 2009; Wirz-Justice, et al., 2008) and older people's resistance to using sleeping medications (Venn, et al., 2013), these are a promising, non-invasive option. People with dementia are a complex and vulnerable group. Therefore the management of dementia-related sleep problems requires a dyadic approach which considers the multiple factors that could be contributing to sleep problems and their successful treatment (Kotronoulas, et al., 2013).

Sleep disturbances have been related to more severe waking symptoms of dementia (Eeles, 2006; Vitiello & Borson, 2001). While it is difficult to differentiate between irreversible neuropathological cognitive decline and the impairments associated with sleep deprivation, in theory, treating sleep problems could have a positive impact on other symptoms. It should be noted that some PWD in Study 4 did appear

to have benefitted from these interventions, regardless of statistical significance. Furthermore, there appears to be no harm in attempting such treatments, so long as the PWD and their carer do not find the process too demanding.

The case studies presented in Study 4 indicate that healthcare professionals need to be mindful of the dyad when treating symptoms related to dementia. A more holistic psychosocial approach is recommended with regards to people close to the PWD, in conjunction with the individual. For example, specific treatment plans could be designed for each dyad dependent on the specific sleep problems, sleep timing, as well as other factors affecting their sleep and potential compliance with the intervention. Greater emotional support for caregivers or extended family may also be necessary. The workload and burden associated with providing care for PWD were identified as barriers to carers taking part. For example, the possibility of changing their daily routine was daunting to some despite the potential for those changes to improve the situation. A protocol where support is offered throughout the process is necessary in order to address any questions or problems as they arise.

A holistic approach sits comfortably within culturally sensitive healthcare. For example, Māori communities encourage the inclusion of the wider whānau in health-related consultations and in caring for people with disabilities (Collins & Wilson, 2008; Cram, Smith, & Johnstone, 2003; Kidd, Gibbons, Lawrenson, & Johnstone, 2010). This is important as more Māori people are expected to be presenting with dementia in the future (Alzheimers New Zealand Incorporated, 2012). Therefore, clinicians need to be mindful of strategies to manage their symptoms in an ethical and appropriate manner.

Identifying dyads that might benefit from non-pharmacological interventions and reaching them at the right time for them clinically and psychologically is an important consideration and challenge. The results from Studies 1 and 2 and others (Arber, et al., 2009; Grandner, et al., 2012; Venn & Arber, 2012) indicate that the likelihood of self-reporting sleep problems could change with age, possibly due to altered perceptions regarding sleep and ageing. The focus groups in Study 3 revealed that some dyads lacked awareness regarding the extent of each other's sleep problems. Previous studies have also identified that carers might not report sleep problems related to the PWD in their care unless their own sleep is disrupted (Tractenberg, et al., 2005). Reasons for PWD waking their carer in the night are multifactorial

and need exploring when designing treatment plans (McCurry, et al., 1999). The symptoms that carers are less aware of are more likely to be normalised or downplayed. Carers who are more critical of their partner's behaviour have also been identified as more likely to give inaccurate reports of their sleep, compared to actigraphy data (McCurry, et al., 2006).

Studies 3 and 4 also indicated that daytime sleeping could be underreported by older people. Through conducting interviews with community-dwelling, 65-95 year-old, problem sleepers, Venn and Arber (2011) found that 50% of older people resisted rather than accepted daytime sleep. Reasons for resistance were related to negative connotations associated with being inactive and of being old. Such resistance is likely to contribute to reduced reports of daytime sleeping. Study 3 indicated that some carers would sacrifice an opportunity to sleep in order to get household chores done. Carers could be even less likely to accept or report daytime sleeping if they associate the activity with guilt or wasting time that could be spent on chores or care-providing activities. Conversely, those who accept napping might not report such activities due to considering it a normal part of the ageing process (Venn & Arber, 2011). Together these findings indicate that using simple questionnaires alone may not be sufficient to gauge sleeping habits and problems amongst older people, PWD, and carers.

Older people have been identified as unlikely to talk to their doctor about sleep problems, due to the expectation that sleeping medications could be prescribed against their will (Venn & Arber, 2012; Venn, et al., 2013). Healthcare professionals are therefore encouraged to take the time to talk to their older clients about sleep before symptoms are likely to become more severe and less easy to manage. Prompting PWD to talk openly about their sleep, rather than relying solely on proxy reports or objective measures, could be useful for developing more targeted and successful interventions. This could also be more meaningful for measuring efficacy compared to tools such as the SDI used in the present and previous studies. A more recently developed questionnaire for PWD to report on their own sleep (Manni, Sinforiani, Zucchella, Terzaghi, & Rezzani, 2013) could be used to guide consultations to assess the PWD's perceptions of their sleep fragmentation, with the guidance of their carer. Taking the time for such conversations in clinical practice is also likely to help carers to appreciate the experience of the person in their care and facilitate their support processes. The dementia and caregiving experiences are dynamic.

Therefore levels of stress, coping, and reports of sleep disturbances are likely to vary (Simpson & Carter, 2013b). There is a clinical need to keep reassessing and offering sleep-related support to PWD and carers throughout the disease process.

6.3.3 Community implications.

More immediate implications from the present research are the development of resources for use via Alzheimers Wellington. The 10 light boxes used in Study 4 have been offered to Alzheimers Wellington as a koha (donation). New aims include establishing a programme for PWD to be able to borrow the light boxes from them in conjunction with the SWRC and their GP's support. A brief interview or questionnaire could be used to audit effects. Resources addressing sleep hygiene and exercise for promoting healthy sleep will also be redeveloped. These resources could be made available at a broader level in order to help raise community awareness with regards to sleep changes with ageing and dementia. This will help PWD and carers to be better prepared for the physiological and psychosocial changes that could affect their sleep, and give them access to strategies that could help.

Results from Study 3 have been summarised in the form of a pamphlet, in newsletters, and local presentations, in order to raise awareness of the importance of sleep for this population. The results from Study 4 will also be summarised this way and disseminated to members of Alzheimers Wellington as well as via other organisations and healthcare professionals associated with elder care. Findings from all four studies have been presented at national and international scientific conferences. Sharing the results in this form contributes to wider knowledge and awareness regarding the nature of sleep problems of older people and PWD.

6.4 Conclusion

Sleeping is a vital state that we all experience and need on a daily basis. The experience of restorative and satisfying sleep depends not only on physiological processes, but also on health factors and psychosocial elements. People who sleep too little, have fragmented sleep, or attempt sleep at times out of step with their internal circadian timing system, are more likely to experience cognitive impairment, symptoms of mood disorders, poorer health and have more accidents. Increasing age has consistently been

associated with increased risk of sleep disorders. Prior to this thesis, there was limited information available on the sleep-health of older New Zealanders. Studies 1 and 2 confirm that sleep problems are associated with poorer physical and mental health among older New Zealanders. The demographic risk factors for poorer sleep health among younger New Zealanders (notably socioeconomic deprivation and being Māori) may change or become less significant with advanced age. Underreporting sleep problems is likely to be an issue among older people.

Diseases causing dementia are more common with ageing and are often associated with symptoms of disturbed sleep. This is due to the accelerated degeneration of the areas and networks within the brain necessary for healthy sleeping, as well as psychosocial factors. Sleep disturbances can be challenging for informal carers whose sleep is also affected. This creates a situation in which the symptoms and effects of dementia can be exacerbated, and living safely and happily at home together becomes more difficult. Life expectancy is increasing, and so the proportion of people affected by dementia is also rising. Therefore, it is increasingly important to understand how sleep changes with ageing, dementia, and caregiving, and to offer possible therapies while PWD are still living at home.

People with dementia and family carers were under-represented in Studies 1 and 2, as well as in previous longitudinal and epidemiological research. The impersonal nature of large, quantitative studies makes representing PWD's voices methodologically and ethically challenging. In response to this, more qualitative and person-centred research was designed. The focus group conversations in Study 3 explored the dyadic themes, addressing the diverse nature of sleep problems experienced by PWD and carers living in the NZ community. The desire to manage dementia-related sleep problems was highlighted, however the tendency to under-report or normalise sleep problems was still evident.

Medications are often prescribed to treat sleeping problems for older people and PWD, despite the possibility of negative side effects, uncertainty over long-term efficacy, and older people's resistance towards medicating sleeping problems. Therefore, the final study trialled non-pharmacological interventions for community-dwelling PWD and their carers. Case study results indicated that, for some PWD, positive effects can be experienced with regards to sleep and dementia-related behaviours. Carers' sleep and mood may also be improved. The case-study design allowed for factors affecting the feasibility

and success of the interventions to be explored. This contributes to the current literature with regards to theory, research and practice. It is recommended that future community-based trails of non-pharmacological interventions should focus more on psychosocial methods and protocols that reduce research burden and are more person-centred.

In the meantime, it is recommended that PWD, carers, and healthcare professionals are made aware of the importance and impact of dementia-related sleep problems. Increased knowledge and awareness should translate into sleep being more formally addressed during consultations with researchers, clinicians, or social workers. This might increase opportunities for non-pharmacological treatments to be trialled and accepted, empowering PWD and carers to manage their own sleep.

In summary, this body of research offers new knowledge regarding sleep, ageing and dementia. This, together with the potential of non-pharmacological interventions, serves to inform future research as well as education and treatment options. These implications provide hope for improving the sleeping and waking experience of older people affected by dementia.

7 REFERENCES

Achermann, P., & Borbély, A. A. (2011). Sleep homeostasis and models of sleep regulation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 431-444). St. Louis, MO: Saunders, Elsiver Inc.

Alessi, C. A., Martin, J. L., Webber, A. P., Kim, E. C., Harker, J. O., & Josephson, K. R. (2005). Randomized, controlled trial of a nonpharmacological intervention to improve abnormal sleep/wake patterns in nursing home residents. *Journal of the American Geriatrics Society, 53*(5), 803-810.

Alessi, C. A., Yoon, E., Schnelle, J. F., Al-Samanai, N. R., & Cruise, P. A. (1999). A randomized trial of a combined physical activity and environmental intervention in nursing home residents: Do sleep and agitation improve? *Journal of the American Geriatrics Society, 47*(7), 784-791.

Alpass, F., Pond, R., Stephens, C., Stevenson, B., Keeling, S., & Towers, A. (2013). The influence of ethnicity and gender on caregiver health in older New Zealanders. *Journals of Gerontology - Series B Psychological Sciences and Social Sciences, 68*(5), 783-793.

Alpass, F., Towers, A., Stephens, C., Fitzgerald, E., Stevenson, B., & Davey, J. (2007). Independence, well-being, and social participation in an aging population. *Annals of the New York Academy of Sciences, 1114*, 241-250.

Altena, E., Ramautar, J. R., Van Der Werf, Y. D., & Van Someren, E. J. W. (2010). Do sleep complaints contribute to age-related cognitive decline? *Progress In Brain Research, 185*(C), 181-205.

Altshuler, K. Z., Barad, M., & Golfarb, A. I. (1963). A survey of dreams in the aged. II. Noninstitutionalized subjects. *Archives of General Psychiatry, 8*, 33-37.

Alzheimers New Zealand Incorporated. (2008). Dementia economic impact report. Retrieved from http://www.alzheimers.org.nz/reports-and-statistics/

Alzheimers New Zealand Incorporated. (2010). National dementia strategy 2010-2015. Retrieved from http://www.nzdoctor.co.nz/media/261405/national%20dementia%20strategy%20alzheimers%20new%20zealand.pdf

Alzheimers New Zealand Incorporated. (2012). Updated dementia economic impact report, 2011, New Zealand. Retrieved from http://www.alzheimers.org.nz/reports-and-statistics/

American Academy of Sleep Medicine. (2005). *The international classification of sleep disorders - Second edition (ICSD-2).* Chicago IL: American Academy of Sleep Medicine.

Ancoli-Israel, S., Amatniek, J., Ascher, S., Sadik, K., & Ramaswamy, K. (2005). Effects of galantamine versus donepezil on sleep in patients with mild to moderate Alzheimer disease and their caregivers: A double-blind, head-to-head, randomized pilot study. *Alzheimer Disease and Associated Disorders, 19*(4), 240-245.

Ancoli-Israel, S., Clopton, P., Klauber, M. R., Fell, R., & Mason, W. (1997). Use of wrist activity for monitoring sleep/wake in demented nursing- home patients. *Sleep, 20*(1), 24-27.

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep, 26*(3), 342-392.

Ancoli-Israel, S., Klauber, M. R., Butters, N., Parker, L., & Kripke, D. F. (1991). Dementia in institutionalized elderly: Relation to sleep apnea. *Journal of the American Geriatrics Society, 39*(3), 258-263.

Ancoli-Israel, S., Klauber, M. R., Gillin, J. C., Campbell, S. S., & Hofstetter, C. R. (1994). Sleep in non-institutionalized Alzheimer's disease patients. *Aging Clinical and Experimental Research, 6*(6), 451-458.

Ancoli-Israel, S., Klauber, M. R., Jones, D. W., Kripke, D. F., Martin, J., Mason, W., . . . Fell, R. (1997). Variations in circadian rhythms of activity, sleep, and light exposure related to dementia in nursing-home patients. *Sleep, 20*(1), 18-23.

Ancoli-Israel, S., Kripke, D. F., Klauber, M. R., Mason, W. J., Fell, R., & Kaplan, O. (1991). Periodic limb movements in sleep in community-dwelling elderly. *Sleep, 14*(6), 496-500.

Ancoli-Israel, S., Martin, J. L., Gehrman, P. R., Shochat, T., Corey-Bloom, J., Marler, M., . . . Levi, L. (2003). Effect of light on agitation in institutionalized patients with severe Alzheimer disease. *American Journal of Geriatric Psychiatry, 11*(2), 194-203.

Ancoli-Israel, S., Martin, J. L., Kripke, D. F., Marler, M., & Klauber, M. R. (2002). Effect of light treatment on sleep and circadian rhythms in demented nursing home patients. *Journal of the American Geriatrics Society, 50*(2), 282-289.

Anthony, J. C., LeResche, L., & Niaz, U. (1982). Limits of the 'mini-mental state' as a screening test for dementia and delirium among hospital patients. *Psychological Medicine, 12*(2), 397-408.

Arber, S., Bote, M., & Meadows, R. (2009). Gender and socio-economic patterning of self-reported sleep problems in Britain. *Social Science and Medicine, 68*(2), 281-289.

Arber, S., & Venn, S. (2011). Caregiving at night: Understanding the impact on carers. *Journal of Aging Studies, 25*(2), 155-165.

Arkin, S. M. (1999). Elder rehab: A student-supervised exercise program for Alzheimer's patients. *Gerontologist, 39*(6), 729-735.

Arkin, S. M. (2003). Student-led exercise sessions yield significant fitness gains for Alzheimer's patients. *American Journal of Alzheimer's Disease and other Dementias, 18*(3), 159-170.

Attride-Stirling, J. (2001). Thematic networks: An analytic tool for qualitative research. *Qualitative Research, 1*(3), 385-405.

Avidan, A. Y. (2005). Sleep disordered breathing in the geriatric patient population. In M. P. Mattson (Ed.), *Sleep and aging* (pp. 79-111). Amsterdam: Elsivier.

Avidan, A. Y. (2007). Sleep disturbances in dementia and other neurodegenerative disorders. In A. Culebras (Ed.), *Sleep disorders and neurologic diseases* (pp. 315-336). New York: Informa Healthcare USA.

Bachman, D., & Rabins, P. (2006). "Sundowning" and other temporally associated agitation states in dementia patients. *Annual Reveiw of Medicine, 57*, 499-511.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research, 53*(3), 737-740.

Baddeley, A. (2012). Working memory: Theories, models, and controversies. *Annual Review of Psychology, 63*, 1-29.

Baddeley, A., Bressi, S., Sala, S. D., Logie, R., & Spinnler, H. (1991). The decline of working memory in Alzheimer's disease. A longitudinal study. *Brain, 114*(6), 2521-2542.

Baehr, E. K., Eastman, C. I., Revelle, W., Olson, S. H. L., Wolfe, L. F., & Zee, P. C. (2003). Circadian phase-shifting effects of nocturnal exercise in older compared with young adults. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 284*(6), 53-56.

Baikie, E. (2002). The impact of dementia on marital relationships. *Sexual and Relationship Therapy, 17*(3), 289-299.

Baldwin, C. (Ed.). (2011). *Personhood, personalism and dementia: A journey of becoming*. London: Jessica Kingsley Publishers.

Ballard, C., Creese, B., Corbett, A., & Aarsland, D. (2011). Atypical antipsychotics for the treatment of behavioral and psychological symptoms in dementia, with a particular focus on longer term outcomes and mortality. *Expert Opinion on Drug Safety, 10*(1), 35-43.

Ballard, C., Holmes, C., McKeith, I., Neill, D., O'Brien, J., Cairns, N., . . . Perry, R. (1999). Psychiatric morbidity in dementia with Lewy bodies: A prospective clinical and neuropathological comparative study with Alzheimer's disease. *American Journal of Psychiatry, 156*(7), 1039-1045.

Ballard, C., Waite, J., & Birks, J. (2012). Atypical antipsychotics for aggression and psychosis in Alzheimer's disease (review). *The Cochrane Library*. Retrieved from http://www.update-software.com/pdf/CD003476.pdf

Bamford, C., & Bruce, E. (2002). Sucesses and challenges in using focus groups with older people with dementia. In H. Wilkinson (Ed.), *The perspectives of people with dementia: Research methods and motivations* (pp. 139-164). London, England: Jessica Kingsley Publishers.

Barclay, N. L., Eley, T. C., Buysse, D. J., Rijsdijk, F. V., & Gregory, A. M. (2010). Genetic and environmental influences on different components of the Pittsburgh Sleep Quality Index and their overlap. *Sleep, 33*(5), 659-668.

Barker, W. W., Luis, C. A., Kashuba, A., Luis, M., Harwood, D. G., Loewenstein, D., . . . Duara, R. (2002). Relative frequencies of Alzheimer disease, Lewy body, vascular and frontotemporal dementia, and hippocampal sclerosis in the State of Florida Brain Bank. *Alzheimer Disease and Associated Disorders, 16*(4), 203-212.

Barry, H. C., & Eathorne, S. W. (1994). Exercise and aging: Issues for the practitioner. *Medical Clinics of North America, 78*(2), 357-376.

Barter, G., & Cormack, M. (1996). The long-term use of benzodiazepines: Patients' views, accounts and experiences. *Family Practice, 13*(6), 491-497.

Bartlett, H., & Martin, W. (2002). Ethical issues in dementia care research. In H. Wilkinson (Ed.), *The Perspectives of People with Dementia: Research Methods and Motivations* (pp. 47-62). London, England: Jessica Kingsley Publishers.

Baskys, A., & Hou, A. C. (2007). Vascular dementia: Pharmacological treatment approaches and perspectives. *Clinical Interventions in Aging, 2*(3), 327-335.

Bastien, C. H., LeBlanc, M., Carrier, J., & Morin, C. M. (2003). Sleep EEG power spectra, insomnia, and chronic use of benzodiazepines. *Sleep, 26*(3), 313-317.

Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the insomnia severity index as an outcome measure for insomnia research. *Sleep Medicine, 2*(4), 297-307.

Bazargan, M. (1996). Self-reported sleep disturbance among African-American elderly: The effects of depression, health status, exercise, and social support. *International Journal of Aging & Human Development, 42*(2), 143-160.

Benedetti, F., Barbini, B., Colombo, C., & Smeraldi, E. (2007). Chronotherapeutics in a psychiatric ward. *Sleep Medicine Reviews, 11*(6), 509-522.

Berghmans, R. L. P., & Ter Meulen, R. H. J. (1995). Ethical issues in research with dementia patients. *International Journal of Geriatric Psychiatry, 10*(8), 647-651.

Bixler, E. O., Kales, A., Soldatos, C. R., Kales, J. D., & Healey, S. (1979). Prevalence of sleep disorders in the Los Angeles metropolitan area. *American Journal of Psychiatry, 136*(10), 1257-1262.

Bixler, E. O., Kales, A., Vela-Bueno, A., Jacoby, J. A., Scarone, S., & Soldatos, C. R. (1982). Nocturnal myoclonus and nocturnal myoclonic activity in a normal population. *Research Communications in Chemical Pathology and Pharmacology, 36*(1), 129-140.

Bixler, E. O., Vgontzas, A. N., Ten Have, T., Tyson, K., & Kales, A. (1998). Effects of age on sleep apnea in men. I. Prevalence and severity. *American Journal of Respiratory and Critical Care Medicine, 157*(1), 144-148.

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: An updated literature review. *Journal of Psychosomatic Research, 52*(2), 69-77.

Black, B. S., Kass, N. E., Fogarty, L. A., & Rabins, P. V. (2007). Informed consent for dementia research: The study enrollment encounter. *IRB Ethics and Human Research, 29*(4), 7-14.

Blake, D. D., & Gómez, M. H. (1998). A scale for assessing sleep hygiene: preliminary data. *Psychological Reports, 83*(3 Pt 2), 1175-1178.

Blampied, N. M. (1999). A legacy neglected: Restating the case for single-case research in cognitive-behaviour therapy. *Behaviour Change, 16*(2), 89-104.

Blampied, N. M. (2001). The third way: Single-case research, training, and practice in clinical psychology. *Australian Psychologist, 36*(2), 157-163.

Bliwise, D. L. (1993). Sleep in normal aging and dementia. *Sleep, 16*(1), 40-81.

Bliwise, D. L. (1994). What is sundowning? *Journal of the American Geriatrics Society, 42*(9), 1009-1011.

Bliwise, D. L. (2004). Sleep disorders in Alzheimer's disease and other dementias. *Clinical Cornerstone, 6*(1), 16-28.

Bliwise, D. L. (2006). Periodic leg movements in sleep and restless legs syndrome: Considerations in geriatrics. *Sleep Medicine Clinics, 1*(2), 263-271.

Bliwise, D. L. (2011a). Normal aging. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 27-41). St. Louis, MO: Saunders, Elsiver Inc.

Bliwise, D. L. (2011b). Sleep in independently living and institutionalized elderly. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 1551-1562). St. Louis, MO: Saunders, Elsiver Inc.

Bliwise, D. L., Ansari, F. P., Straight, L. B., & Parker, K. P. (2005). Age changes in timing and 24-hour distribution of self-reported sleep. *American Journal of Geriatric Psychiatry, 13*(12), 1077-1082.

Bliwise, D. L., Watts, R. L., Watts, N., Rye, D. B., Irbe, D., & Hughes, M. (1995). Disruptive nocturnal behavior in Parkinson's disease and Alzheimer's disease. *Journal of Geriatric Psychiatry and Neurology, 8*(2), 107-110.

Bliwise, D. L., Yesavage, J. A., & Tinklenberg, J. R. (1992). Sundowning and rate of decline in mental function in Alzheimer's disease. *Dementia, 3*(5-6), 335-341.

Bliwise, D. L., Yesavage, J. A., Tinklenberg, J. R., & Dement, W. C. (1989). Sleep apnea in Alzheimer's disease. *Neurobiology of Aging, 10*(4), 343-346.

Boddy, F., Rowan, E. N., Lett, D., O'Brien, J. T., McKeith, I. G., & Burn, D. J. (2007). Subjectively reported sleep quality and excessive daytime somnolence in Parkinson's disease with and without dementia, dementia with Lewy Bodies and Alzheimer's disease. *International Journal of Geriatric Psychiatry, 22*(6), 529-535.

Boeve, B. F. (2008). Update on the diagnosis and management of sleep disturbances in dementia. *Sleep Medicine Clinics, 3*(3), 347-360.

Boivin, D. B., Czeisler, C. A., Dijh, D. J., Duffy, J. F., Folkard, S., Minors, D. S., . . . Waterhouse, J. M. (1997). Complex interaction of the sleep-wake cycle and circadian phase modulates mood in healthy subjects. *Archives of General Psychiatry, 54*(2), 145-152.

Bonanni, E., Maestri, M., Tognoni, G., Fabbrini, M., Nucciarone, B., Manca, M. L., . . . Murri, L. (2005). Daytime sleepiness in mild and moderate Alzheimer's disease and its relationship with cognitive impairment. *Journal of Sleep Research, 14*(3), 311-317.

Bonelli, S. B., Ransmayr, G., Steffelbauer, M., Lukas, T., Lampl, C., & Deibl, M. (2004). L-dopa responsiveness in dementia with Lewy bodies, Parkinson disease with and without dementia. *Neurology, 63*(2), 376-378.

Booth, T., & Booth, W. (1996). Sounds of silence: Narrative research with inarticulate subjects. *Disability and Society, 11*(1), 55-69.

Borlase, B. J., Gander, P. H., & Gibson, R. H. (2013). Effects of school start times and technology use on teenagers' sleep: 1999-2008. *Sleep and Biological Rhythms, 11*(1), 46-54.

Braak, H., & Braak, E. (1991). Neuropathological stageing of Alzheimer-related changes. *Acta Neuropathologica, 82*(4), 239-259.

Braak, H., Thal, D. R., Ghebremedhin, E., & Del Tredici, K. (2011). Stages of the pathologic process in Alzheimer disease: Age categories from 1 to 100 years. *Journal of Neuropathology and Experimental Neurology, 70*(11), 960-969.

Bradford, C. A. (2004). *Basic ophthalmology* (8 ed.). San Fransiscon, CA: American Academy of Ophthalmology.

Brækhus, A., Øksengård, A. R., Engedal, K., & Laake, K. (1998). Social and depressive stress suffered by spouses of patients with mild dementia. *Scandinavian Journal of Primary Health Care, 16*(4), 242-246.

Brainard, G. C., Hanifin, J. R., Greeson, J. M., Byrne, B., Glickman, G., Gerner, E., & Rollag, M. D. (2001). Action spectrum for melatonin regulation in humans: Evidence for a novel circadian photoreceptor. *Journal of Neuroscience, 21*(16), 6405-6412.

Brannelly, T. (2011). Sustaining citizenship: People with dementia and the phenomenon of social death. *Nursing Ethics, 18*(5), 662-671.

Braskie, M. N., Klunder, A. D., Hayashi, K. M., Protas, H., Kepe, V., Miller, K. J., . . . Thompson, P. M. (2010). Plaque and tangle imaging and cognition in normal aging and Alzheimer's disease. *Neurobiology of Aging, 31*(10), 1669-1678.

Braun, V., & Clarke, V. (2006). Using thematic analysis in psychology. *Qualitative Research in Psychology, 3*(2), 77-101.

Bravo, G., Paquet, M., & Dubois, M. (2003). Opinions regarding who should consent to research on behalf of an older adult suffering from dementia. *Dementia, 2*, 49-65.

Bray, A. (1998). *Research involving people with intellectual disabilitites: Issues of informed consent and participation*. Dunedin, New Zealand: Donald Beasley Institute.

Brickell, K. L., Leverenz, J. B., Steinbart, E. J., Rumbaugh, M., Schellenberg, G. D., Nochlin, D., . . . Bird, T. D. (2007). Clinicopathological concordance and discordance in three monozygotic twin pairs with familial Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry, 78*(10), 1050-1055.

Brickell, K. L., Steinbart, E. J., Rumbaugh, M., Payami, H., Schellenberg, G. D., Van Deerlin, V., . . . Bird, T. D. (2006). Early-onset Alzheimer disease in families with late-onset alzheimer disease: A potential important subtype of familial Alzheimer disease. *Archives of Neurology, 63*(9), 1307-1311.

Brill, P. A., Drimmer, A. M., Morgan, L. A., & Gordon, N. F. (1995). The feasibility of conducting strength and flexibility programs for elderly nursing home residents with dementia. *Gerontologist, 35*(2), 263-266.

Brouwer, W. B. F., Van Exel, N. J. A., & Stolk, E. A. (2005). Acceptability of less than perfect health states. *Social Science and Medicine, 60*(2), 237-246.

Brown, C. A., Berry, R., Tan, M. C., Khoshia, A., Turlapati, L., & Swedlove, F. (2013). A critique of the evidence base for non-pharmacological sleep interventions for persons with dementia. *Dementia, 12*(2), 210-237.

Brunton, M., Fouché, C., & Jordan, C. (2007). When privilege turns to duty: Balancing work-life commitments as an unpaid caregiver. In M. Waring & C. Fouché (Eds.), *Managing mayhem: Work-life balance in New Zealand* (pp. 175-199). Wellington, New Zealand: Dunmore Books Ltd.

Brusco, L. I., Fainstein, I., Marquez, M., & Cardinali, D. P. (1999). Effect of melatonin in selected populations of sleep-disturbed patients. *Biological Signals and Receptors, 8*(1-2), 126-131.

Brzezinski, A., Vangel, M. G., Wurtman, R. J., Norrie, G., Zhdanova, I., Ben-Shushan, A., & Ford, I. (2005). Effects of exogenous melatonin on sleep: A meta-analysis. *Sleep Medicine Reviews, 9*(1), 41-50.

Buckles, V. D., Powlishta, K. K., Palmer, J. L., Coats, M., Hosto, T., Buckley, A., & Morris, J. C. (2003). Understanding of informed consent by demented individuals. *Neurology, 61*(12), 1662-1666.

Buscemi, N., Vandermeer, B., Hooton, N., Pandya, R., Tjosvold, L., Hartling, L., . . . Vohra, S. (2005). The efficacy and safety of exogenous melatonin for primary sleep disorders: A meta-analysis. *Journal of General Internal Medicine, 20*(12), 1151-1158.

Buscemi, N., Vandermeer, B., Hooton, N., Pandya, R., Tjosvold, L., Hartling, L., . . . Baker, G. (2006). Efficacy and safety of exogenous melatonin for secondary sleep disorders and sleep disorders accompanying sleep restriction: Meta-analysis. *British Medical Journal, 332*(7538), 385-388.

Busse, A., Hensel, A., Gühne, U., Angermeyer, M. C., & Riedel-Heller, S. G. (2006). Mild cognitive impairment: Long-term course of four clinical subtypes. *Neurology, 67*(12), 2176-2185.

Buxton, O. M., Lee, C. W., L'Hermite-Balériaux, M., Turek, F. W., & Van Cauter, E. (2003). Exercise elicits phase shifts and acute alterations of melatonin that vary with circadian phase. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 284*(3 53-3), 53-53.

Buysse, D. J., Reynolds, I. C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193-213.

Buysse, D. J., Reynolds Iii, C. F., Monk, T. H., Hoch, C. C., Yeager, A. L., & Kupfer, D. J. (1991). Quantification of subjective sleep quality in healthy elderly men and women using the Pittsburgh Sleep Quality Index (PSQI). *Sleep, 14*(4), 331-338.

Buzsáki, G. (1998). Memory consolidation during sleep: A neurophysiological perspective. *Journal of Sleep Research, Supplement, 7*(1), 17-23.

Campbell, S. S., Kripke, D. F., Gillin, J. C., & Hrubovcak, J. C. (1988). Exposure to light in healthy elderly subjects and Alzheimer's patients. *Physiology and Behavior, 42*(2), 141-144.

Campbell, S. S., Terman, M., Lewy, A. J., Dijk, D. J., Eastman, C. I., & Boulos, Z. (1995). Light treatment for sleep disorders: consensus report. V. Age-related disturbances. *Journal of Biological Rhythms, 10*(2), 151-154.

Cardinali, D. P., Brusco, L. I., Liberczuk, C., & Furio, A. M. (2002). The use of melatonin in Alzheimer's disease. *Neuroendocrinology Letters, 23*(1), 20-23.

Cardinali, D. P., & Karasek, M. (2010). Melatonin, aging, and Alzheimer's disease. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 97-106). Cambridge, England: Cambridge University Press.

Carpenter, B., Strauss, M., & Patterson, M. B. (1996). Sleep disturbances in community-dwelling patients with Alzheimer's disease. *Clinical Gerontologist, 16*(2), 35-49.

Carpenter, J., & Andrykowski, M. A. (1998). Psychometric evaluation of the Pittsburgh Sleep Quality Index. *Journal of Psychosomatic Research, 45*(1), 5-13.

Carr, S. A., Davis, R., Spencer, D., Smart, M., Hudson, J., Freeman, S., . . . Jicha, G. A. (2010). Comparison of recruitment efforts targeted at primary care physicians versus the community at large for participation in Alzheimer disease clinical trials. *Alzheimer Disease and Associated Disorders, 24*(2), 165-170.

Carrier, J. (2010). Possible mechanisms and cosequences of age-related changes in middle years of life. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 22-33). Cambridge, England: Cambridge University Press.

Carrier, J., Viens, I., Poirier, G., Robillard, R., Lafortune, M., Vandewalle, G., . . . Filipini, D. (2011). Sleep slow wave changes during the middle years of life. *European Journal of Neuroscience, 33*(4), 758-766.

Carskadon, M. A., & Dement, W. C. (2011). Normal human sleep: An overview *Principle and pracrtice of sleep medicine* (5 ed., pp. 16-26). St Louis, MO: Elsevier Inc.

Carvalho-Bos, S. S., Riemersma-van Der Lek, R. F., Waterhouse, J., Reilly, T., & Van Someren, E. J. W. (2007). Strong association of the rest-activity rhythm with well-being in demented elderly women. *American Journal of Geriatric Psychiatry, 15*(2), 92-100.

Caselli, R. J. (2008). Dementia: Risk factors and genetics. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology of the older adult* (Vol. 2, pp. 296-302). Philadelphia, PA: Wolters Kluwer: Lippincott Williams & Wilkins.

Castro, C. M., Lee, K. A., Bliwise, D. L., Urizar, G. G., Woodward, S. H., & King, A. C. (2009). Sleep patterns and sleep-related factors between caregiving and non-caregiving women. *Behavioral Sleep Medicine, 7*(3), 164-179.

Charlson, M. E., Pompei, P., Ales, K. A., & MacKenzie, C. R. (1987). A new method of classifying prognostic comorbidity in longitudinal studies: Development and validation. *Journal of Chronic Diseases, 40*(5), 373-383.

Chen, J. C., Borson, S., & Scanlan, J. M. (2000). Stage-specific prevalence of behavioral symptoms in Alzheimer's disease in a multi-ethnic community sample. *American Journal of Geriatric Psychiatry, 8*(2), 123-133.

Chenoweth, B., & Spencer, B. (1986). Dementia: The experience of family caregivers. *The Gerontologist, 26*(3), 267-272.

Chesson, A., L., Littner, Michael., Davila, David., Anderson, MacDowell., Grigg-Damberger., Hartse, Kristyna., Johnsen, Stephen., Wise, Merrill. (1999). Practice parameter for the use of light therapy in the treatment of sleep disorders. *Sleep 22*(5), 641-660.

Chiu, Y. C., Lee, Y. N., Wang, P. C., Chang, T. H., Li, C. L., Hsu, W. C., & Lee, S. H. (2014). Family caregivers' sleep disturbance and its associations with multilevel stressors when caring for patients with dementia. *Aging and Mental Health, 18*(1), 92-101.

Choi, O., & Irwin, M. R. (2008). Insomnia in aging. In A. Y. Avidan & C. Alessi (Eds.), *Geriatric sleep medicine* (pp. 89-112). New York, NY: Informa Healthcare USA.

Clare, L. (2004). Awareness in early-stage Alzheimer's disease: A review of methods and evidence. *British Journal of Clinical Psychology, 43*(2), 177-196.

Clarke, C. L., & Keady, J. (2002). Getting down to brass tacks: A discussion of data collection with people with dementia. In H. Wilkinson (Ed.), *The perspectives of people with dementia: Research methods and motivations* (pp. 25-46). London, England: Jessica Kingsley Publishers.

Cloyd, J. C., & Conway, J. M. (2008). Age-related changes in pharmacokinetics, drug interations, and adverse effects. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology of the older adult* (Vol. 2, pp. 40-53). Philadelphia, PA: Wolters Kluwer: Lippincott Williams & Wilkins.

Cohen-Mansfield, J. (2003). Consent and refusal in dementia research: Conceptual and practical considerations. *Alzheimer Disease and Associated Disorders, 17*(1), 17-25.

Cohen-Mansfield, J., Garfinkel, D., & Lipson, S. (2000). Melatonin for treatment of sundowning in elderly persons with dementia - A preliminary study. *Archives of Gerontology and Geriatrics, 31*(1), 65-76.

Colenda, C. C., Cohen, W., McCall, W. V., & Rosenquist, P. B. (1997). Phototherapy for patients with Alzheimer disease with disturbed sleep patterns: Results of a community-based pilot study. *Alzheimer Disease and Associated Disorders, 11*(3), 175-178.

Collins, A., & Wilson, G. (2008). Māori and informal caregiving: A background paper prepared for the National Health Committee. Retrieved from http://nhc.health.govt.nz/publications/nhc-publications-pre-2011/m%C4%81ori-and-informal-caregiving-background-paper-prepared

Collop, N. (2006). Polysomnography. In T. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 973-976). Hoboken, NJ: John Wiley and Sons Inc.

Connell, C. M., & Gibson, G. D. (1997). Racial, ethnic, and cultural differences in dementia caregiving: Review and analysis. *Gerontologist, 37*(3), 355-364.

Cooke, J. R., Liu, L., Natarajan, L., He, F., Marler, M., Loredo, J. S., . . . Ancoli-Israel, S. (2006). The effect of sleep-disordered breathing on stages of sleep in patients with Alzheimer's disease. *Behavioral Sleep Medicine, 4*(4), 219-227.

Cooke, J. R., Loredo, J. S., Liu, L., Marler, M., Corey-Bloom, J., Fiorentino, L., . . . Ancoli-Israel, S. (2006). Acetylcholinesterase inhibitors and sleep architecture in patients with Alzheimer's disease. *Drugs and Aging, 23*(6), 503-511.

Cornwall, J., & Davies, J. (2004). Impact of population ageing in New Zealand on the demand for health and disability support services, and workforce implications: A background paper completed for the Ministry of Health in June 2003 Retrieved from https://www.health.govt.nz/system/files/documents/publications/cornwallanddavey.pdf

Cotrell, V., & Schulz, R. (1993). The perspective of the patient with Alzheimer's disease: A neglected dimension of dementia research. *Gerontologist, 33*(2), 205-211.

Cram, F., Smith, L., & Johnstone, W. (2003). Mapping the themes of Maori talk about health. *New Zealand Medical Journal, 116*(1170)

Creese, J., Bédard, M., Brazil, K., & Chambers, L. (2008). Sleep disturbances in spousal caregivers of individuals with Alzheimer's disease. *International Psychogeriatrics, 20*(1), 149-161.

Crespo, M., López, J., & Zarit, S. H. (2005). Depression and anxiety in primary caregivers: A comparative study of caregivers of demented and nondemented older persons. *International Journal of Geriatric Psychiatry, 20*(6), 591-592.

Culberson, J. W., & Ziska, M. (2008). Prescription drug misuse/abuse in the elderly. *Geriatrics, 63*(9), 22-26.

Cummings, J. L., & McPherson, S. (2001). Neuropsychiatric assessment of Alzheimer's disease and related dementias. *Aging Clinical and Experimental Research, 13*(3), 240-246.

Cummings, J. L., Mega, M., Gray, K., Rosenberg-Thompson, S., Carusi, D. A., & Gornbein, J. (1994). The neuropsychiatric inventory: Comprehensive assessment of psychopathology in dementia. *Neurology, 44*(12), 2308-2314.

Cupidi, C., Realmuto, S., Lo Coco, G., Cinturino, A., Talamanca, S., Arnao, V., . . . Lo Coco, D. (2012). Sleep quality in caregivers of patients with Alzheimers disease and Parkinsons disease and its relationship to quality of life. *International Psychogeriatrics, 24*(11), 1827-1835.

Czeisler, C. A., & Buxton, O. M. (2011). The human circadian timing system and sleep-wake regulation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 402-419). St. Louis, MO: Saunders, Elsiver Inc.

Czeisler, C. A., Duffy, J. F., Shanahan, T. L., Brown, E. N., Mitchell, J. F., Rimmer, D. W., . . . Kronauer, R. E. (1999). Stability, precision, and near-24-hour period of the human circadian pacemaker. *Science, 284*(5423), 2177-2181.

Czeisler, C. A., Dumont, M., Duffy, J. F., Steinberg, J. D., Richardson, G. S., Brown, E. N., . . . Ronda, J. M. (1992). Association of sleep-wake habits in older people with changes in output of circadian pacemaker. *Lancet, 340*(8825), 933-936.

Dagan, Y. (2007). Diseases don't sleep at night. *Harefuah, 146*(7), 537-538.

Danermark, B., Ekström, M., Jakobsen, L., & Karlsson, J. (2002). *Explaining society: Critical realism in the social sciences*. London, England: Routledge.

Dauvilliers, Y. (2007). Insomnia in patients with neurodegenerative conditions. *Sleep Medicine, 8*(4), 27-34.

Day, K., Carreon, D., & Stump, C. (2000). The therapeutic design of environments for people with dementia: A review of the empirical research. *Gerontologist, 40*(4), 397-416.

De Jonghe, A., Korevaar, J. C., Van Munster, B. C., & De Rooij, S. E. (2010). Effectiveness of melatonin treatment on circadian rhythm disturbances in dementia. Are there implications for delirium? A systematic review. *International Journal of Geriatric Psychiatry, 25*(12), 1201-1208.

Deimling, G. T., & Bass, D. M. (1986). Symptoms of mental impairment among elderly adults and their effects on family caregivers. *Journals of Gerontology, 41*(6), 778-784.

DermNet NZ. (2014). Melatonin. *Treatments* Retrieved April 2, 2013, from http://www.dermnetnz.org/treatments/melatonin.html

Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., Houck, P. R., . . . Reynolds Iii, C. F. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine, 65*(1), 63-73.

Dewing, J. (2002). From ritual to relationship: A person-centred approach to consent in qualitative research with older people who have a dementia. *Dementia, 1*, 157-171.

Dewing, J. (2007). Participatory research: A method for process consent with persons who have dementia. *Dementia, 6*(1), 11-25.

Dijk, D. J., Duffy, J. F., & Czeisler, C. A. (2001). Age-related increase in awakenings: Impaired consolidation of nonREM sleep at all circadian phases. *Sleep, 24*(5), 565-577.

Dijk, D. J., Duffy, J. F., Kiel, E., Shanahan, T. L., & Czeisler, C. A. (1999). Ageing and the circadian and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *Journal of Physiology, 516*(2), 611-627.

Dijk, D. J., & Edgar, D. M. (1999). Circadian and homeostatic control of wakefulness and sleep. In F. W. Turek & P. C. Zee (Eds.), *Regulation of sleep and circadian rhythms* (pp. 111-147). New York, NY: Marcel Decker.

Domhoff, G. W. (2001). A new neurocognitive theory of dreams. *Dreaming, 11*(1), 13-33.

Dorsey, C. M., Lukas, S. E., Teicher, M. H., Harper, D., Winkelman, J. W., Cunningham, S. L., & Satlin, A. (1996). Effects of passive body heating on the sleep of older female insomniacs. *Journal of Geriatric Psychiatry and Neurology, 9*(2), 83-90.

Dos Santos Moraes, W. A., Poyares, D. R., Guilleminault, C., Ramos, L. R., Ferreira Bertolucci, P. H., & Tufik, S. (2006). The effect of donepezil on sleep and REM sleep EEG in patients with Alzheimer disease: A double-blind placebo-controlled study. *Sleep, 29*(2), 199-205.

Dowling, G. A., Burr, R. L., Van Someren, E. J. W., Hubbard, E. M., Luxenberg, J. S., Mastick, J., & Cooper, B. A. (2008). Melatonin and bright light treatment for rest-activity disruption in institutionalised patients with Alzheimer's disease. *Jounral of American Geriatrics Society, 56*, 239-256.

Dowling, G. A., & Mastick, J. (2010). Effects of light on the elderly. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 423-430). Cambridge, England: Cambridge University Press.

Dowling, G. A., Mastick, J., Hubbard, E. M., Luxenberg, J. S., & Burr, R. L. (2005). Effect of timed bright light treatment for rest-activity disruption in institutionalized patients with Alzheimer's disease. *International Journal of Geriatric Psychiatry, 20*(8), 738-743.

Dowling, G. A., & Wiener, C. L. (1997). Roadblocks encountered in recruiting patients for a study of sleep disruption in Alzheimer's disease. *Journal of Nursing Scholarship, 29*(1), 59-64.

Downs, M. (1997). The emergence of the person in dementia research. *Ageing and Society, 17*(5), 597-607.

Driver, H. S., & Taylor, S. R. (2000). Exercise and sleep. *Sleep Medicine Reviews, 4*(4), 387-402.

Duffy, J. F., & Czeisler, C. A. (2009). Effect of Light on Human Circadian Physiology. *Sleep Medicine Clinics, 4*(2), 165-177.

Duffy, J. F., Dijk, D. J., Klerman, E. B., & Czeisler, C. A. (1998). Later endogenous circadian temperature nadir relative to an earlier wake time in older people. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 275*(5), 44-45.

Duffy, J. F., Willson, H. J., Wang, W., & Czeisler, C. A. (2009). Healthy older adults better tolerate sleep deprivation than young adults: Brief reports. *Journal of the American Geriatrics Society, 57*(7), 1245-1251.

Duffy, J. F., & Wright, K. P., Jr. (2005). Entrainment of the human circadian system by light. *Journal of Biological Rhythms, 20*(4), 326-338.

Duffy, J. F., Zeitzer, J. M., Rimmer, D. W., Klerman, E. B., Dijk, D. J., & Czeisler, C. A. (2002). Peak of circadian melatonin rhythm occurs later within the sleep of older subjects. *American Journal of Physiology - Endocrinology and Metabolism, 282*(2), 45-42.

Durrence, H. H., & Lichstein, K. L. (2006). The sleep of African Americans: A comparative review. *Behavioral Sleep Medicine, 4*(1), 29-44.

Dyall, L., Kepa, M., Hayman, K., Teh, R., Moyes, S., Broad, J. B., & Kerse, N. (2013). Engagement and recruitment of Māori and non-Māori people of advanced age to LiLACS NZ. *Australian and New Zealand Journal of Public Health, 37*(2), 124-131.

Dyall, L., Kēpa, M., Teh, R., Mules, R., Moyes, S., Wham, C., . . . Kerse, N. (2014). Cultural and social factors and quality of life of Māori in advanced age: Te puāwaitanga o ngā tapuwae kia ora tonu. Life and living in advanced age: A cohort study in New Zealand (LiLACS NZ). *New Zelaand Medical Journal, 127*(1393), 62-79.

Ebly, E. M., Hogan, D. B., & Fung, T. S. (1997). Potential adverse outcomes of psychotropic and narcotic drug use in Canadian seniors. *Journal of Clinical Epidemiology, 50*(7), 857-863.

Eeles, E. (2006). Sleep and its management in dementia. *Reviews in Clinical Gerontology, 16*(1), 59-70.

Eggermont, L. H. P., & Scherder, E. J. A. (2006). Physical activity and behaviour in dementia: A review of the literature and implications for psychosocial intervention in primary care. *Dementia, 5*(3), 411-428.

Ellmers, T., Arber, S., Luff, R., Eyers, I., & Young, E. (2013). Factors affecting residents' sleep in care homes. *Nursing Older People, 25*(8), 29-32.

Elmståhl, S., Stenberg, I., Annerstedt, L., & Ingvad, B. (1998). Behavioral disturbances and pharmacological treatment of patients with dementia in family caregiving: A 2-year follow-up. *International Psychogeriatrics, 10*(3), 239-252.

Enderlin, C., & Richards, K. C. (2006). Sleep, exercise, and sports. In T. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 947-952). Hoboken, New Jersey: John Wiley & Sons Inc.

Exum, M. E., Phelps, B. J., Nabers, K. E., & Osborne, J. G. (1993). Sundown syndrome: Is it reflected in the use of PRN medications for nursing home residents? *Gerontologist, 33*(6), 756-761.

Falsetti, A. E. (2000). Risperidone for control of agitation in dementia patients. *American Journal of Health-System Pharmacy, 57*(9), 862-870.

Farrer, L. A., Cupples, L. A., Haines, J. L., Hyman, B., Kukull, W. A., Mayeux, R., . . . Van Duijn, C. M. (1997). Effects of age, sex, and ethnicity on the association between apolipoprotein E genotype and Alzheimer disease: A meta-analysis. *Journal of the American Medical Association, 278*(16), 1349-1356.

Fazio, S. (2013). The individual is the core-and the key-to the person centered care. *Generations, 37*(3), 16-22.

Feher, E. P., Mahurin, R. K., Inbody, S. B., Crook, T. H., & Pirozzolo, F. J. (1991). Anosognosia in Alzheimer's disease. *Neuropsychiatry, Neuropsychology and Behavioral Neurology, 4*(2), 136-146.

Fellows, L. K. (1998). Competency and consent in dementia. *Journal of the American Geriatrics Society, 46*(7), 922-926.

Ferrari, E., Cravello, L., Falvo, F., Barili, L., Solerte, S. B., Fioravanti, M., & Magri, F. (2008). Neuroendocrine features in extreme longevity. *Experimental Gerontology, 43*(2), 88-94.

Figueiro, M. G. (2008). A proposed 24 h lighting scheme for older adults. *Lighting Research and Technology, 40*, 153-160.

Figueiro, M. G., Lesniak, N. Z., & Rea, M. S. (2011). Implications of controlled short-wavelength light exposure for sleep in older adults. *BioMed Central: Research Notes, 4*, 334.

Finelli, L. A., Baumann, H., Borbély, A. A., & Achermann, P. (2000). Dual electroencephalogram markers of human sleep homeostasis: Correlation between theta activity in waking and slow-wave activity in sleep. *Neuroscience, 101*(3), 523-529.

Finnema, E., Dröes, R. M., Ribbe, M., & Van Tilburg, W. (2000). A review of psychosocial models in psychogeriatrics: Implications for care and research. *Alzheimer Disease and Associated Disorders, 14*(2), 68-80.

Fisk, M., & Wigley, V. (2000). Accessing and interviewing the oldest old in care homes. *Quality in Ageing – Policy, Practice and Research, 1*(1), 27-33.

Fitzpatrick, A. L., Kuller, L. H., Lopez, O. L., Kawas, C. H., & Jagust, W. (2005). Survival following dementia onset: Alzheimer's disease and vascular dementia. *Journal of the Neurological Sciences, 229-230*, 43-49.

Fleishman, J. A., Selim, A. J., & Kazis, L. E. (2010). Deriving SF-12v2 physical and mental health summary scores: A comparison of different scoring algorithms. *Quality of Life Research, 19*(2), 231-241.

Fogel, S. M., & Smith, C. T. (2006). Learning-dependent changes in sleep spindles and Stage 2 sleep. *Journal of Sleep Research, 15*(3), 250-255.

Fogel, S. M., & Smith, C. T. (2011). The function of the sleep spindle: A physiological index of intelligence and a mechanism for sleep-dependent memory consolidation. *Neuroscience and Biobehavioral Reviews, 35*(5), 1154-1165.

Foley, D. J., Ancoli-Israel, S., Britz, P., & Walsh, J. (2004). Sleep disturbances and chronic disease in older adults: Results of the 2003 National Sleep Foundation Sleep in America Survey. *Journal of Psychosomatic Research, 56*(5), 497-502.

Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: An epidemiologic study of three communities. *Sleep, 18*(6), 425-432.

Foley, D. J., Vitiello, M. V., Bliwise, D. L., Ancoli-Israel, S., Monjan, A. A., & Walsh, J. K. (2007). Frequent napping is associated with excessive daytime sleepiness, depression, pain, and nocturia in older adults: Findings from the National Sleep Foundation 2003 sleep in America poll. *American Journal of Geriatric Psychiatry, 15*(4), 344-350.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). 'Mini mental state'. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*(3), 189-198.

Fonareva, I., Amen, A. M., Zajdel, D. P., Ellingson, R. M., & Oken, B. S. (2011). Assessing sleep architecture in dementia caregivers at home using an ambulatory polysomnographic system. *Journal of Geriatric Psychiatry and Neurology, 24*(1), 50-59.

Fontana Gasio, P., Kräuchi, K., Cajochen, C., Van Someren, E., Amrhein, I., Pache, M., . . . Wirz-Justice, A. (2003). Dawn-dusk simulation light therapy of disturbed circadian rest-activity cycles in demented elderly. *Experimental Gerontology, 38*(1-2), 207-216.

Forbes, D., Culum, I., Lichka, A.R., Morgan, D.G., Peacock, S., Forbes, J., Forbes, S. (2009). Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia (review). *The Chochrane Collaboration,* (4). Retrieved from http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD003946.pub3/pdf

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders: An opportunity for prevention? *Journal of the American Medical Association, 262*(11), 1479-1484.

Francis, P. T., Palmer, A. M., Snape, M., & Wilcock, G. K. (1999). The cholinergic hypothesis of Alzheimer's disease: A review of progress. *Journal of Neurology Neurosurgery and Psychiatry, 66*(2), 137-147.

Frank, E., Sidor, M. M., Gamble, K. L., Cirelli, C., Sharkey, K. M., Hoyle, N., . . . Hasler, B. P. (2013). Circadian clocks, brain function, and development. *Annals of the New York Academy of Sciences, 1306*(1), 43-67.

Friedman, L., Spira, A. P., Hernandez, B., Mather, C., Sheikh, J., Ancoli-Israel, S., . . . Zeitzer, J. M. (2012). Brief morning light treatment for sleep/wake disturbances in older memory-impaired individuals and their caregivers. *Sleep Medicine, 13*(5), 546-549.

Fuller, P. M., Gooley, J. J., & Saper, C. B. (2006). Neurobiology of the sleep-wake cycle: Sleep architecture, circadian regulation, and regulatory feedback. *Journal of Biological Rhythms, 21*(6), 482-493.

Funkhouser, A. T., Hirsbrunner, H.-P., Cornu, C., & Bahro, M. (1999). Dreams and dreaming among the elderly: An overview. *Aging and Mental Health, 3*(1), 10-20.

Furio, A. M., Brusco, L. I., & Cardinali, D. P. (2007). Possible therapeutic value of melatonin in mild cognitive impairment: A retrospective study. *Journal of Pineal Research, 43*(4), 404-409.

Gagnon, J. F., Bédard, M. A., Fantini, M. L., Petit, D., Panisset, M., Rompré, S., . . . Montplaisir, J. (2002). REM sleep behavior disorder and REM sleep without atonia in Parkinson's disease. *Neurology, 59*(4), 585-589.

Gais, S., Mölle, M., Helms, K., & Born, J. (2002). Learning-dependent increases in sleep spindle density. *Journal of Neuroscience, 22*(15), 6830-6834.

Gallagher-Thompson, D., Brooks Iii, J. O., Bliwise, D., Leader, J., & Yesavage, J. A. (1992). The relations among caregiver stress, 'sundowning' symptoms, and cognitive decline in Alzheimer's disease. *Journal of the American Geriatrics Society, 40*(8), 807-810.

Gallagher, K. S., Odenheimer, G., & Kunik, M. E. (2011). Treating sleep problems in dementia caregivers based on parent-child interventions. *American Journal of Alzheimer's Disease and other Dementias, 26*(5), 366-372.

Gallicchio, L., & Kalesan, B. (2009). Sleep duration and mortality: A systematic review and meta-analysis. *Journal of Sleep Research, 18*(2), 148-158.

Gander, P. H. (2003). *Sleep in the 24-hour society*. Lower Hutt, New Zealand: The Open Polytechnic of New Zealand.

Gander, P. H., Marshall, N. S., Harris, R. B., & Reid, P. (2005a). The Epworth Sleepiness Scale: Influence of age, ethnicity, and socioeconomic deprivation. Epworth Sleepiness scores of adults in New Zealand. *Sleep, 28*(2), 249-253.

Gander, P. H., Marshall, N. S., Harris, R. B., & Reid, P. (2005b). Sleep, sleepiness and motor vehicle accidents: A national survey. *Australian and New Zealand Journal of Public Health, 29*(1), 16-20.

Garfinkel, D., Laudon, M., Nof, D., & Zisapel, N. (1995). Improvement of sleep equality in elderly people by controlled-release melatonin. *Lancet, 346*(8974), 541-544.

Garratt, S., & Hamilton-Smith, E. (1995). *Rethinking dementia: An Australian approach*. Melbourne, Australia: Ausmed Publications.

Gehrman, P. R., Connor, D. J., Martin, J. L., Shochat, T., Corey-Bloom, J., & Ancoli-Israel, S. (2009). Melatonin fails to improve sleep or agitation in double-blind randomized placebo-controlled trial of institutionalized patients with Alzheimer disease. *American Journal of Geriatric Psychiatry, 17*(2), 166-169.

Gehrman, P. R., & Goonerante, N. S. (2010). Non-pharmacological treatment of insomnia in the elderly: Cognitive behaviour therapies. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 384-393). Cambridge, England: Cambridge University Press.

Gehrman, P. R., Marler, M., Martin, J. L., Shochat, T., Corey-Bloom, J., & Ancoli-Israel, S. (2004). The timing of activity rhythms in patients with dementia is related to survival. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 59*(10), 1050-1055.

Gehrman, P. R., Martin, J. L., Shochat, T., Nolan, S., Corey-Bloom, J., & Ancoli-Israel, S. (2003). Sleep-disordered breathing and agitation in institutionalized adults with Alzheimer disease. *American Journal of Geriatric Psychiatry, 11*(4), 426-433.

Geula, C., Nagykery, N., Nicholas, A., & Wu, C. K. (2008). Cholinergic neuronal and axonal abnormalities are present early in aging and in Alzheimer disease. *Journal of Neuropathology and Experimental Neurology, 67*(4), 309-318.

Gibson, R. H., Elder, D., & Gander, P. (2012). Actigraphic sleep and developmental progress of one-year-old infants. *Sleep and Biological Rhythms, 10*(2), 77-83.

Gibson, R. H., Gander, P., & Elder, D. (2012). Factors differentiating infants identified by parents as problem sleepers, and those that are not. *Sleep and Biological Rhythms, 10*(1), 46-52.

Gibson, R. H., Gander, P. H., Alpass, F., & Stephens, C. (in press). The effect of caregiving status on the sleep of older New Zealanders. *Australasian Journal on Ageing*

Gibson, R. H., Gander, P. H., & Jones, L. M. (2014). Understanding the sleep problems of people with dementia and their family caregivers. *Dementia: The International Journal of Social Research and Practice, 13*(3), 348 - 363.

Glass, J., Lanctôt, K. L., Herrmann, N., Sproule, B. A., & Busto, U. E. (2005). Sedative hypnotics in older people with insomnia: Meta-analysis of risks and benefits. *British Medical Journal, 331*(7526), 1169-1173.

Goldberg, D., Gater, R., Sartorius, N., Ustun, T. B., Piccinelli, M., Gureje, O., & Rutter, C. (1997). The validity of two versions of the GHQ in the WHO study of mental illness in general health care. *Psychological Medicine, 27*(1), 191-197.

Goldberg, L., & Williams, P. (1988). *A user's guide to the General Health Questionnaire*. Windsor, England: NFER-Nelson.

Goldsmith, M. (1996). *Hearing the voice of people with dementia: Oppotunitites and obstacles*. London, England: Jessica Kingsly Publishers.

Goodhead, A., & McDonald, J. (2007). *Informal caregivers literature review: A report for the National Health Committee*. Wellington, New Zealand: Health Services Research Centre, Victoria University of Wellington.

Gooley, J., & Saper, C. B. (2011). Anatomy of the mamillian circadian system. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 376-389). St. Louis, MO: Saunders, Elsiver Inc.

Grandner, M. A., Martin, J. L., Patel, N. P., Jackson, N. J., Gehrman, P. R., Pien, G., . . . Gooneratne, N. S. (2012). Age and sleep disturbances among American men and women: Data from the U.S. Behavioral Risk Factor Surveillance System. *Sleep, 35*(3), 395-406.

Grandner, M. A., Patel, N. P., Gehrman, P. R., Xie, D., Sha, D., Weaver, T., & Gooneratne, N. (2010). Who gets the best sleep? Ethnic and socioeconomic factors related to sleep complaints. *Sleep Medicine, 11*(5), 470-478.

Grandner, M. A., Patel, N. P., Jean-Louis, G., Jackson, N., Gehrman, P. R., Perlis, M. L., & Gooneratne, N. S. (2013). Sleep-related behaviors and beliefs associated with race/ethnicity in women. *Journal of the National Medical Association, 105*(1), 4-15.

Greve, M., & O'Connor, D. (2005). A survey of Australian and New Zealand old age psychiatrists' preferred medications to treat behavioral and psychological symptoms of dementia (BPSD). *International Psychogeriatrics, 17*(2), 195-205.

Groves, W. C., Brandt, J., Steinberg, M., Warren, A., Rosenblatt, A., Baker, A., & Lyketsos, C. G. (2000). Vascular dementia and Alzheimer's disease: Is there a difference? A comparison of symptoms by disease duration. *Journal of Neuropsychiatry and Clinical Neurosciences, 12*(3), 305-315.

Guardiola-Lemaitre, B., & Quera-Salva, M. A. (2011). Melatonin and the regulation of sleep and circadian rhythms. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 420-440). St. Louis: Saunders, Elsiver Inc.

Gulmann, N. C., & Korsgaard, S. (1976). Psychiatric side effects of levodopa treatment. *Ugeskrift for Laeger, 138*(18), 1105-1107.

Gurland, B., & Toner, J. (1983). Differentiating dementia from nondementing conditions. *Advances in Neurology, 38*, 1-17.

Hamilton, M. (1967). Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology, 6*(4), 278-296.

Hancock, P., & Larner, A. J. (2009). Diagnostic utility of the Pittsburgh Sleep Quality Index in memory clinics. *International Journal of Geriatric Psychiatry, 24*(11), 1237-1241.

Harper, D. G. (2010). Sleep and circadian rhythm distrubances in Alzheimer's disease. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 214-226). Cambridge, England: Cambridge University Press.

Harper, D. G., Stopa, E. G., McKee, A. C., Satlin, A., Fish, D., & Volicer, L. (2004). Dementia severity and Lewy bodies affect circadian rhythms in Alzheimer disease. *Neurobiology of Aging, 25*(6), 771-781.

Harper, D. G., Stopa, E. G., McKee, A. C., Satlin, A., Harlan, P. C., Goldstein, R., & Volicer, L. (2001). Differential circadian rhythm disturbances in men with Alzheimer disease and frontotemporal degeneration. *Archives of General Psychiatry, 58*(4), 353-360.

Harper, D. G., Volicer, L., Stopa, E. G., McKee, A. C., Nitta, M., & Satlin, A. (2005). Disturbance of endogenous circadian rhythm in aging and Alzheimer disease. *American Journal of Geriatric Psychiatry, 13*(5), 359-368.

Harris, R., & Dyson, E. (2001). Recruitment of frail older people to research: Lessons learnt through experience. *Journal of Advanced Nursing, 36*(5), 643-651.

Hauri, P. J. (1997). Cognitive deficits in insomnia patients. *Acta Neurologica Belgica, 97*(2), 113-117.

Hayman, K. J., Kerse, N., Dyall, L., Kepa, M., Teh, R., Wham, C., . . . Jatrana, S. (2012). Life and living in advanced age: A cohort study in New Zealand -Te puawaitanga o nga tapuwae kia ora tonu, LiLACS NZ: Study protocol. *BioMed Central: Geriatrics, 12*(33). Retrieved from http://www.scopus.com/inward/record.url?eid=2-s2.0-84863006945&partnerID=40&md5=fce6607bab68af3447fd52a58b278c9d

Health and Disability Commissioner. (1996). Code of health and disability services consumers' rights regulation. *The act and code* Retrieved August 7, 2013, from http://www.hdc.org.nz/the-act--code/the-code-of-rights/the-code-(full)

Hellström, I., Nolan, M., Nordenfelt, L., & Lundh, U. (2007). Ethical and methodological issues in interviewing persons with dementia. *Nursing Ethics, 14*(5), 608-619.

Hening, W. A., Allen, R. P., Earley, C. J., Picchietti, D. L., & Silber, M. H. (2004). An update on the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder. *Sleep, 27*(3), 560-583.

Herljevic, M., Middleton, B., Thapan, K., & Skene, D. J. (2005). Light-induced melatonin suppression: Age-related reduction in response to short wavelength light. *Experimental Gerontology, 40*(3), 237-242.

Herrmann, C. (1997). International experiences with the hospital anxiety and depression scale - A review of validation data and clinical results. *Journal of Psychosomatic Research, 42*(1), 17-41.

Heyman, A., Peterson, B., Fillenbaum, G., & Pieper, C. (1997). Predictors of time to institutionalization of patients with Alzheimer's disease: The CERAD experience, Part XVII. *Neurology, 48*(5), 1304-1309.

Heyn, P., Abreu, B. C., & Ottenbacher, K. J. (2004). The effects of exercise training on elderly persons with cognitive impairment and dementia: A meta-analysis. *Archives of Physical Medicine and Rehabilitation, 85*(10), 1694-1704.

Higgins, P. A., Hornick, T. R., & Figueiro, M. G. (2010). Rest-activity and light exposure patterns in the home setting: A methodological case study. *American Journal of Alzheimer's Disease and other Dementias, 25*(4), 353-361.

Hoch, C. C., Reynolds Iii, C. F., Kupfer, D. J., Houck, P. R., Berman, S. R., & Stack, J. A. (1986). Sleep-disordered breathing in normal and pathologic aging. *Journal of Clinical Psychiatry, 47*(10), 499-503.

Hoch, C. C., Reynolds Iii, F., Nebes, R. D., Kupfer, D. J., Berman, S. R., & Campbell, D. (1989). Clinical significance of sleep-disordered breathing in Alzheimer's disease. Preliminary data. *Journal of the American Geriatrics Society, 37*(2), 138-144.

Hofman, M. A., & Swaab, D. F. (1994). Alterations in circadian rhythmicity of the vasopressin-producing neurons of the human suprachiasmatic nucleus (SCN) with aging. *Brain Research, 651*(1-2), 134-142.

Hoogendijk, W. J. G., Van Someren, E. J. W., Mirmiran, M., Hofman, M. A., Lucassen, P. J., Zhou, J. N., & Swaab, D. F. (1996). Circadian rhythm-related behavioral disturbances and structural hypothalamic changes in Alzheimer's disease. *International Psychogeriatrics, 8*(3), 245-252.

Hope, T., Keene, J., Gedling, K., Fairburn, C. G., & Jacoby, R. (1998). Predictors of institutionalization for people with dementia living at home with a carer. *International Journal of Geriatric Psychiatry, 13*(10), 682-690.

Hoque, R., & Chesson A. L, J. (2010). Pharmacologically induced/exacerbated restless legs syndrome, periodic limb movements of sleep, and REM behavior disorder/REM sleep without atonia: Literature review, qualitative scoring, and comparative analysis. *Journal of Clinical Sleep Medicine, 6*(1), 79-83.

House, J. S., Lepkowski, J. M., Kinney, A. M., Mero, R. P., Kessler, R. C., & Herzog, A. R. (1994). The social stratification of aging and health. *Journal of Health and Social Behavior, 35*(3), 213-234.

Hubbard, G., Downs, M. G., & Tester, S. (2003). Including older people with dementia in research: Challenges and strategies. *Aging and Mental Health, 7*(5), 351-362.

Iliffe, S., Manthorpe, J., Warner, J., Drennan, V., Goodman, C., Rait, G., & Kharicha, K. (2008). Making progress in psychosocial research in dementia. *Dementia, 7*(2), 167-174.

Inglis, F. (2002). The tolerability and safety of cholinesterase inhibitors in the treatment of dementia. *International Journal of Clinical Practice, Supplement*(127), 45-63.

Ingram, C. D., Ciobanu, R., Coculescu, I. L., Tanasescu, R., Coculescu, M., & Mihai, R. (1998). Vasopressin neurotransmission and the control of circadian rhythms in the suprachiasmatic nucleus. *Progress In Brain Research, 119*, 351-364.

Innes, A. (2009). *Dementia studies:A social science perspective*. London, England: Sage.

Jack, C. R., Jr., Petersen, R. C., Xu, Y. C., O'Brien, P. C., Smith, G. E., Ivnik, R. J., . . . Kokmen, E. (1999). Prediction of AD with MRI-based hippocampal volume in mild cognitive impairment. *Neurology, 52*(7), 1397-1403.

Jacobs, D., Ancoli-Israel, S., Parker, L., & Kripke, D. F. (1989). Twenty-four-hour sleep-wake patterns in a nursing home population. *Psychology and Aging, 4*(3), 352-356.

Jean-Louis, G., Von Gizycki, H., & Zizi, F. (1998). Melatonin effects on sleep, mood, and cognition in elderly with mild cognitive impairment. *Journal of Pineal Research, 25*(3), 177-183.

Jellinger, K. A. (2006). Clinicopathological analysis of dementia disorders in the elderly - An update. *Journal of Alzheimer's Disease, 9*(3), 61-70.

Jewett, M. E., Kronauer, R. E., & Czeisler, C. A. (1994). Phase-amplitude resetting of the human circadian pacemaker via bright light: A further analysis. *Journal of Biological Rhythms, 9*(3-4), 295-314.

Jewett, M. E., Rimmer, D. W., Duffy, J. F., Klerman, E. B., Kronauer, R. E., & Czeisler, C. A. (1997). Human circadian pacemaker is sensitive to light throughout subjective day without evidence of transients. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 273*(5 42-5), 1800-1809.

Jimenez, C. J., Perez, T. A., Prieto, F. S., & Navia-Osorio, P. M. (1989). Behavioural habits and affective disorders in old people. *Journal of Advanced Nursing, 14*(5), 356-364.

Johnson, M. P., Duffy, J. F., Dijk, D. J., Ronda, J. M., Dyal, C. M., & Czeisler, C. A. (1992). Short-term memory, alterness and performance: A reappraisal of their relationship to body temperature. *Journal of Sleep Research, 1*(1), 24-29.

Jorgensen, D., Parsons, M., Jacobs, S., & Arksey, H. (2010). The New Zealand informal caregivers and their unmet needs. *New Zealand Medical Journal, 123*(1317), 9-16.

Jorm, A. F., & Jolley, D. (1998). The incidence of dementia: A meta-analysis. *Neurology, 51*(3), 728-733.

Judge, K. S., Menne, H. L., & Whitlatch, C. J. (2010). Stress process model for individuals with dementia. *Gerontologist, 50*(3), 294-302.

Jyrkkä, J., Enlund, H., Korhonen, M. J., Sulkava, R., & Hartikainen, S. (2009). Patterns of drug use and factors associated with polypharmacy and excessive polypharmacy in elderly persons: Results of the kuopio 75 study: A cross-sectional analysis. *Drugs and Aging, 26*(6), 493-503.

Kavanau, J. L. (2001). Memory failures, dream illusions and mental malfunction. *Neuropsychobiology, 44*(4), 199-211.

Kawas, C., Gray, S., Brookmeyer, R., Fozard, J., & Zonderman, A. (2000). Age-specific incidence rates of Alzheimer's disease: The Baltimore longitudinal study of aging. *Neurology, 54*(11), 2072-2077.

Kawinska, A., Dumont, M., Selmaoui, B., Paquet, J., & Carrier, J. (2005). Are modifications of melatonin circadian rhythm in the middle years of life related to habitual patterns of light exposure? *Journal of Biological Rhythms, 20*(5), 451-460.

Kerr, D., Wilkinson, H., & Cunningham, C. (2008). *Supporting older people in care homes at night*. York, England: Joseph Rowntree Foundation.

Kesselring, A., Krulik, T., Bichsel, M., Minder, C., Beck, J. C., & Stuck, A. E. (2001). Emotional and physical demands on caregivers in home care to the elderly in Switzerland and their relationship to nursing home admission. *European Journal of Public Health, 11*(3), 267-273.

Khachiyants, N., Trinkle, D., Son, S. J., & Kim, K. Y. (2011). Sundown syndrome in persons with dementia: An update. *Psychiatry Investigation, 8*(4), 275-287.

Khalsa, S. B. S., Jewett, M. E., Cajochen, C., & Czeisler, C. A. (2003). A phase response curve to single bright light pulses in human subjects. *Journal of Physiology, 549*(3), 945-952.

Kiata, L., Kerse, N., & Dixon, R. (2005). Residential care workers and residents: The New Zealand story. *New Zealand Medical Journal, 118*(1214). Retrieved from URL: http://www.nzma.org.nz/journal/118-1214/1445/

Kidd, J., Gibbons, V., Lawrenson, R., & Johnstone, W. (2010). A whanau ora approach to health care for Maori. *Journal of Primary Health Care, 2*(2), 163-164.

Kim, J., Tofade, T. S., & Peckman, H. (2009). Caring for the elderly in an inpatient setting: Managing Insomnia and polypharmacy. *Journal of Pharmacy Practice, 22*(5), 494-506.

King, A. C., Oman, R. F., Brassington, G. S., Bliwise, D. L., & Haskell, W. L. (1997). Moderate-intensity exercise and self-rated quality of sleep in older adults: A randomized controlled trial. *Journal of the American Medical Association, 277*(1), 32-37.

Kirkman, A. M. (2006). Dementia in the news: The media coverage of Alzheimer's disease. *Australasian Journal on Ageing, 25*(2), 74-79.

Kitabayashi, Y., Ueda, H., Tsuchida, H., Yamashita, T., Narumoto, J., & Fukui, K. (2006). Donepezil-induced nightmares in mild cognitive impairment. *Psychiatry and Clinical Neurosciences, 60*(1), 123-124.

Kitwood, T. (1989). Brian, mind and dementia: with particular reference to Alzheimer's disease. *Ageing and Society, 9*(1), 1-15.

Kitwood, T. (1997). *Dementia reconsidered: The person comes first*. Buckingham, England: Open University Press.

Kitwood, T., & Bredin, K. (1992). Toward a theory of dementia care: Personhood and wellbeing. *Ageing and Society, 12*, 269-287.

Klaffke, S., & Staedt, J. (2006). Sundowning and circadian rhythm disorders in dementia. *Acta Neurologica Belgica, 106*(4), 168-175.

Knopman, D. S., Parisi, J. E., Boeve, B. F., Cha, R. H., Apaydin, H., Salviati, A., . . . Rocca, W. A. (2003). Vascular dementia in a population-based autopsy study. *Archives of Neurology, 60*(4), 569-575.

Ko, C. H., & Takahashi, J. S. (2006). Molecular components of the mammalian circadian clock. *Human Molecular Genetics, 15*(2), 271-277.

Kolker, D. E., Vitaterna, M. H., Fruechte, E. M., Takahashi, J. S., & Turek, F. W. (2004). Effects of age on circadian rhythms are similar in wild-type and heterozygous Clock mutant mice. *Neurobiology of Aging, 25*(4), 517-523.

Kondratova, A. A., & Kondratov, R. V. (2012). The circadian clock and pathology of the ageing brain. *Nature Reviews Neuroscience, 13*(5), 325-335.

Korczyn, A. D., & Halperin, I. (2009). Depression and dementia. *Journal of the Neurological Sciences, 283*(1-2), 139-142.

Kotronoulas, G., Wengström, Y., & Kearney, N. (2013). Sleep and sleep-wake disturbances in care recipient-caregiver dyads in the context of a chronic illness: A critical review of the literature. *Journal of Pain and Symptom Management, 45*(3), 579-594.

Kripke, D. F. (2000). Chronic hypnotic use: Deadly risks, doubtful benefit. *Sleep Medicine Reviews, 4*(1), 5-20.

Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry, 59*(2), 131-136.

Krueger, R. A. (1994). *Focus groups: A practical guide for applied research* (2nd ed.). Thousand Oaks, CA: Sage.

Krystal, A. D. (2009). A compendium of placebo-controlled trials of the risks/benefits of pharmacological treatments for insomnia: The empirical basis for U.S. clinical practice. *Sleep Medicine Reviews, 13*(4), 265-274.

Kubitz, K. A., Landers, D. M., Petruzzello, S. J., & Han, M. (1996). The effects of acute and chronic exercise on sleep: A meta-analytic review. *Sports Medicine, 21*(4), 277-291.

Kuhn, D., Edelman, P., & Fulton, B. R. (2005). Daytime sleep and the threat to well-being of persons with dementia. *Dementia, 4*(2), 233-247.

Kukull, W. A., Brenner, D. E., Speck, C. E., Nochlin, D., Bowen, J., McCormick, W., . . . Larson, E. B. (1994). Causes of death associated with Alzheimer disease: Variation by level of cognitive impairment before death. *Journal of the American Geriatrics Society, 42*(7), 723-726.

Kushida, C. A., Chang, A., Gadkary, C., Guilleminault, C., Carrillo, O., & Dement, W. C. (2001). Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep-disordered patients. *Sleep Medicine, 2*(5), 389-396.

Lack, L., & Wright, H. (1993). The effect of evening bright light in delaying the circadian rhythms and lengthening the sleep of early morning awakening insomniacs. *Sleep, 16*(5), 436-443.

Lack, L., Wright, H., Kemp, K., & Gibbon, S. (2005). The treatment of early-morning awakening insomnia with 2 evenings of bright light. *Sleep, 28*(5), 616-623.

Lader, M. (1986). The use of hypnotics and anxiolytics in the elderly. *International Clinical Psychopharmacology, 1*(4), 273-283.

Landolt, H. P. (2008). Sleep homeostasis: A role for adenosine in humans? *Biochemical Pharmacology, 75*(11), 2070-2079.

Landolt, H. P., & Borbély, A. A. (2001). Age-dependent changes in sleep EEG topography. *Clinical Neurophysiology, 112*(2), 369-377.

Landolt, H. P., Dijk, D. J., Achermann, P., & Borbély, A. A. (1996). Effect of age on the sleep EEG: Slow-wave activity and spindle frequency activity in young and middle-aged men. *Brain Research, 738*(2), 205-212.

Laurin, D., Verreault, R., Lindsay, J., MacPherson, K., & Rockwood, K. (2001). Physical activity and risk of cognitive impairment and dementia in elderly persons. *Archives of Neurology, 58*(3), 498-504.

Lawton, M. P. (1983). The varieties of wellbeing. *Experimental Aging Research, 9*(2), 65-72.

Lawton, M. P. (1991). A multidimensional view of quality of life in frail elders. In J. E. Birren, J. E. Lubben, J. C. Rowe & Deutchman (Eds.), *The Concept and Measurement of Quality of Life in the Frail Elderly* (pp. 3-11). San Diego, CA: Academic Press, Inc. Harcourt Brace Jovanovich.

Lawton, M. P. (1997). Assessing quality of life in Alzheimer disease research. *Alzheimer Disease and Associated Disorders, 11*(6), 91-99.

Lawton, M. P., Moss, M., & Duhamel, L. M. (1995). The quality of daily life among elderly care receivers. *Journal of Applied Gerontology, 14*(2), 150-171.

Lawton, M. P., Winter, L., Kleban, M. H., & Ruckdeschel, K. (1999). Affect and quality of life objective and subjective. *Journal of Aging and Health, 11*(2), 169-198.

Lee-Chiong, T., & Harrington, J. (2008). Pharmacologic therapy of chronic insomnia in older adults. In A. Y. Avidan & C. Alessi (Eds.), *Geriatric sleep medicine* (pp. 113-139). New York, NY: Informa Healthcare USA.

Lee, D. (2008). Health-related quality of life in dementia caregiving: Relationships with sleep quality and daytime sleepiness. *Primary Care and Community Psychiatry, 13*(3), 119-125.

Lee, D., Morgan, K., & Lindesay, J. (2007). Effect of institutional respite care on the sleep of people with dementia and their primary caregivers. *Journal of the American Geriatrics Society, 55*(2), 252-258.

Lee, J., Bliwise, D. L., Ansari, F. P., Goldstein, F. C., Cellar, J. S., Lah, J. J., & Levey, A. I. (2007). Daytime sleepiness and functional impairment in Alzheimer disease. *American Journal of Geriatric Psychiatry, 15*(7), 620-626.

Lewis, G., & Wessely, S. (1990). Comparison of the general health questionnaire and the hospital anxiety and depression scale. *British Journal of Psychiatry, 157*, 860-864.

Li, F., Fisher, K. J., Harmer, P., Irbe, D., Tearse, R. G., & Weimer, C. (2004). Tai chi and self-rated quality of sleep and daytime sleepiness in older adults: A randomized controlled trial. *Journal of the American Geriatrics Society, 52*(6), 892-900.

Lichstein, K. L., Durrence, H. H., Riedel, D. W., Taylor, D. J., & Bush, A. J. (2004). *Epideiology of sleep: Age, gender, and ethnicity*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). Quantitative criteria for insomnia. *Behaviour Research and Therapy, 41*(4), 427-445.

Lichstein, K. L., Heith Durrence, H., Riedel, B. W., & Bayen, U. J. (2001). Primary versus secondary insomnia in older adults: Subjective sleep and daytime functioning. *Psychology and Aging, 16*(2), 264-271.

Lichstein, K. L., Taylor, D. J., McCrae, C. S., & Ruiter, M. E. (2011). Insomnia:Epidemiology and risk factors. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 827-838). St. Louis, MO: Saunders, Elsiver Inc.

Lindstorm, H. A., Smyth, K. A., Sami, S. A., Dawson, N. V., Patterson, M. B., Bohinc, J. H., . . . Whitehouse, P. J. (2006). Medication use to treat memory loss in dementia. *Dementia, 5*(1), 27-50.

Liperoti, R., Pedone, C., & Corsonello, A. (2008). Antipsychotics for the treatment of Behavioral and Psychological Symptoms of Dementia (BPSD). *Current Neuropharmacology, 6*(2), 117-124.

Littner, M., Kushida, C. A., Anderson, M., Bailey, D., Berry, R. B., Davila, D. G., . . . Johnson, S. F. (2003). Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: An update for 2002 - An American academy of sleep medicine report. *Sleep, 26*(3), 337-341.

Liu, R. Y., Zhou, J. N., Hoogendijk, W. J. G., Van Heerikhuize, J., Kamphorst, W., Unmehopa, U. A., . . . Swaab, D. F. (2000). Decreased vasopressin gene expression in the biological clock of Alzheimer disease patients with and without depression. *Journal of Neuropathology and Experimental Neurology, 59*(4), 314-322.

Liu, R. Y., Zhou, J. N., Van Heerikhuize, J., Hofman, M. A., & Swaab, D. F. (1999). Decreased melatonin levels in postmortem cerebrospinal fluid in relation to aging, Alzheimer's disease, and apolipoprotein E-ε4/4 genotype. *Journal of Clinical Endocrinology and Metabolism, 84*(1), 323-327.

Lockley, S. W., Evans, E. E., Scheer, F. A., Brainard, G. C., Czeisler, C. A., & Aeschbach, D. (2006). Short-wavelength sensitivity for the direct effects of light on alertness, vigilance, and the waking electroencephalogram in humans. *Sleep, 29*(2), 161-168.

Loewen, A. H. S., Poulin, M. J., & Hanly, P. J. (2010). Sleep apnea in the elderly. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 3-21). Cambridge, England: Cambridge University Press.

Logsdon, R. G., & Albert, S. M. (1999). Assessing quality of life in Alzheimer's disease: Conceptual and methodological issues. *Journal of Mental Health and Aging, 5*(1), 3-6.

Logsdon, R. G., Gibbons, L. E., McCurry, S. M., & Teri, L. (1999). Quality of life in Alzheimer's Disease: Patient and caregiver reports. *Journal of Mental Health and Aging, 5*(1), 21-32.

Logsdon, R. G., Gibbons, L. E., McCurry, S. M., & Teri, L. (2002). Assessing quality of life in older adults with cognitive impairment. *Psychosomatic Medicine, 64*(3), 510-519.

Logsdon, R. G., Teri, L., McCurry, S. M., Gibbons, L. E., Kukull, W. A., & Larson, E. B. (1998). Wandering: A significant problem among community-residing individuals with Alzheimer's disease. *Journals of Gerontology - Series B Psychological Sciences and Social Sciences, 53*(5), 294-299.

Loomis, A. L., Harvey, E. N., & Hobart, G. A. (1937). Cerebral states during sleep, as studied by human brain potentials. *Journal of Experimental Psychology, 21*(2), 127-144.

Lopez, O. L., Becker, J. T., Chang, Y. F., Sweet, R. A., DeKosky, S. T., Gach, M. H., . . . Kuller, L. H. (2012). Incidence of mild cognitive impairment in the Pittsburgh Cardiovascular Health Study-Cognition Study. *Neurology, 79*(15), 1599-1606.

Lovell, B. B. (1995). Effect of bright light treatment on agitated behavior in institutionalized elderly subjects. *Psychiatry Research, 57*(1), 7-12.

Lucetti, C., Logi, C., Del Dotto, P., Berti, C., Ceravolo, R., Baldacci, F., . . . Bonuccelli, U. (2010). Levodopa response in dementia with lewy bodies: A 1-year follow-up study. *Parkinsonism and Related Disorders, 16*(8), 522-526.

Lyketsos, C. G., Lopez, O., Jones, B., Fitzpatrick, A. L., Breitner, J., & Dekosky, S. (2002). Prevalence of neuropsychiatric symptoms in dementia and mild cognitive impairment: Results from the cardiovascular health study. *Journal of the American Medical Association, 288*(12), 1475-1483.

Maher, J., & Green, H. (2002). *Carers 2000*. London, England: The National Stationary Office.

Mahowald, M. W., & Schenck, C. H. (2000). Diagnosis and management of parasomnias. *Clinical Cornerstone, 2*(5), 48-54.

Malamut, B. L., & Ryan, L. M. (2008). Dementia: Behavioral and congitive aspects. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology of the older adult* (Vol. 2, pp. 318-338). Philadelphia, PA: Wolters Kluwer Health: Lippincott Williams & Wilkins.

Manni, R., Sinforiani, E., Zucchella, C., Terzaghi, M., & Rezzani, C. (2013). A sleep continuity scale in Alzheimer's disease: Validation and relationship with cognitive and functional deterioration. *Neurological Sciences, 34*(5), 701-705.

Manson, N. C., & O'Neill, O. (2007). *Rethinking informed consent in bioethics*. Cambridge, England: Cambridge University Press.

Marks, G. A. (2006). The neurobiology of sleep. In T. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 11-18). Hoboken, NJ: John Wiley and Sons Inc.

Martin, J. L., Marler, M. R., Harker, J. O., Josephson, K. R., & Alessi, C. A. (2007). A multicomponent nonpharmacological intervention improves activity rhythms among nursing home residents with disrupted sleep/wake patterns. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 62*(1), 67-72.

Martin, J. L., Webber, A. P., Alam, T., Harker, J. O., Josephson, K. R., & Alessi, C. A. (2006). Daytime sleeping, sleep disturbance, and circadian rhythms in the nursing home. *American Journal of Geriatric Psychiatry, 14*(2), 121-129.

Martin, W., & Bartlett, H. (2007). The social significance of sleep for older people with dementia in the context of care. *Sociological Research Online, 12*(5). Retrieved from Sociological Research Online website: http://www.socresonline.org.uk/12/5/11.html>

Marx, M. S., Cohen-Mansfield, J., & Guralnik, J. M. (2003). Recruiting community-dwelling elderly at risk for physical disability into exercise research. *Journal of Aging and Physical Activity, 11*(2), 229-241.

Massey University. (2010). Code of ethical conduct for research, teaching and evaluations involving human participants Retrieved from http://www.massey.ac.nz/massey/research/research-ethics/human-ethics/code-ethical-conduct.cfm

McCall, W. V., Erman, M., Krystal, A. D., Rosenberg, R., Scharf, M., Zammit, G. K., & Wessel, T. (2006). A polysomnography study of eszopiclone in elderly patients with insomnia. *Current Medical Research and Opinion, 22*(9), 1633-1642.

McCarley, R. W. (2004). Mechanisms and models of REM sleep control. *Archives Italiennes de Biologie, 142*(4), 429-467.

McCarney, R., Warner, J., Iliffe, S., Van Haselen, R., Griffin, M., & Fisher, P. (2007). The Hawthorne Effect: A randomised, controlled trial. *BioMed Central: Medical Research Methodology, 7*, 30-38.

McCarten, J. R., Kovera, C., Maddox, M. K., & Cleary, J. P. (1995). Triazolam in Alzheimer's disease: Pilot study on sleep and memory effects. *Pharmacology, Biochemistry and Behavior, 52*(2), 447-452.

McCurry, S. M. (2000). Treatment of sleep disturbance in Alzeimer's disease. *Sleep Medicine Reviews, 4*(6), 603-628.

McCurry, S. M., & Ancoli-Israel, S. (2003). Sleep dysfunction in Alzheimer's disease and other dementias. *Current Treatment Options in Neurology, 5*(3), 261-272.

McCurry, S. M., Gibbons, L. E., Logsdon, R. G., Vitiello, M., & Teri, L. (2003). Training caregivers to change the sleep hygiene practices of patients with dementia: The NITE-AD project. *Journal of the American Geriatrics Society, 51*(10), 1455-1460.

McCurry, S. M., Gibbons, L. E., Logsdon, R. G., Vitiello, M. V., & Teri, L. (2005). Nighttime insomnia treatment and education for Alzheimer's disease: A randomized, controlled trial. *Journal of the American Geriatrics Society, 53*(5), 793-802.

McCurry, S. M., Gibbons, L. E., Logsdon, R. G., Vitiello, M. V., & Teri, L. (2009). Insomnia in caregivers of persons with dementia: Who is at risk and what can be done about It? *Sleep Medicine Clinics, 4*(4), 519-526.

McCurry, S. M., Logsdon, R. G., Teri, L., Gibbons, L. E., Kukull, W. A., Bowen, J. D., . . . Larson, E. B. (1999). Characteristics of sleep disturbance in community-dwelling Alzheimer's disease patients. *Journal of Geriatric Psychiatry and Neurology, 12*(2), 53-59.

McCurry, S. M., Logsdon, R. G., Teri, L., & Vitiello, M. V. (2007). Sleep disturbances in caregivers of persons with dementia: Contributing factors and treatment implications. *Sleep Medicine Reviews, 11*(2), 143-153.

McCurry, S. M., Logsdon, R. G., Vitiello, M. V., & Teri, L. (1998). Successful behavioral treatment for reported sleep problems in elderly caregivers of dementia patients: A controlled study. *Journals of Gerontology - Series B Psychological Sciences and Social Sciences, 53*(2), 122-129.

McCurry, S. M., Logsdon, R. G., Vitiello, M. V., & Teri, L. (2004). Treatment of sleep and nighttime disturbances in Alzheimer's disease: A behavior management approach. *Sleep Medicine, 5*(4), 373-377.

McCurry, S. M., Pike, K. C., Logsdon, R. G., Vitiello, M. V., Larson, E. B., & Teri, L. (2010). Predictors of short- and long-term adherence to a daily walking program in persons with Alzheimers disease. *American Journal of Alzheimer's Disease and other Dementias, 25*(6), 505-512.

McCurry, S. M., Pike, K. C., Vitiello, M. V., Logsdon, R. G., Larson, E. B., & Teri, L. (2011). Increasing walking and bright light exposure to improve sleep in community-dwelling persons with Alzheimer's disease: Results of a randomized, controlled trial. *Journal of the American Geriatrics Society, 59*(8), 1393-1402.

McCurry, S. M., Pike, K. C., Vitiello, M. V., Logsdon, R. G., & Teri, L. (2008). Factors associated with concordance and variability of sleep quality in persons with Alzheimer's disease and their caregivers. *Sleep, 31*(5), 741-748.

McCurry, S. M., & Teri, L. (1995). Sleep disturbance in elderly caregivers of dementia patients. *Clinical Gerontologist, 16*(2), 51-65.

McCurry, S. M., Vitiello, M. V., Gibbons, L. E., Logsdon, R. G., & Teri, L. (2006). Factors associated with caregiver reports of sleep disturbances in persons with dementia. *American Journal of Geriatric Psychiatry, 14*(2), 112-120.

McGinty, D., & Szymusiak, R. (2000). The sleep-wake switch: A neuronal alarm clock. *Nature Medicine, 6*(5), 510-511.

McGinty, D., & Szymusiak, R. (2011). Neural control of sleep in mammels. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 76-91). St. Louis, MO: Saunders, Elsiver Inc.

McKee, K. J., Philp, I., Lamura, G., Prouskas, C., Oberg, B., Krevers, B., . . . Szczerbinska, K. (2003). The COPE index - A first stage assessment of negative impact, positive value and quality of support of caregiving in informal carers of older people. *Aging and Mental Health, 7*(1), 39-52.

McKeith, I. (2008). Dementia with Lewy bodies. *Psychiatry, 7*(1), 20-23.

McKeown, J., Clarke, A., Ingleton, C., & Repper, J. (2010). Actively involving people with dementia in qualitative research. *Journal of Clinical Nursing, 19*(13-14), 1935-1943.

McKhann, G., Drachman, D., & Folstein, M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology, 34*(7), 939-944.

McKibbin, C. L., Ancoli-Israel, S., Dimsdale, J., Archuleta, C., Von Kanel, R., Mills, P., . . . Grant, I. (2005). Sleep in spousal caregivers of people with Alzheimer's disease. *Sleep, 28*(10), 1245-1250.

McLennan, S. (Writer). (1999). Easy exercise, *Sit and be fit: Level 1, series 2, for adults with limited mobility* [DVD]. New Zealand: Net Fit.

Meadows, R., Luff, R., Eyers, I., Venn, S., Cope, E., & Arber, S. (2010). An actigraphic study comparing community dwelling poor sleepers with non-demented care home residents. *Chronobiology International, 27*(4), 842-854.

Medical Research Council. (1991). *The ethical conduct of research on the mentally incapacitated* Retrieved from http://ebooks.cambridge.org/ doi:10.1017/CBO9780511550089.062

Meguro, K., Meguro, M., Tanaka, Y., Akanuma, K., Yamaguchi, K., & Itoh, M. (2004). Risperidone is effective for wandering and disturbed sleep/wake patterns in Alzheimer's disease. *Journal of Geriatric Psychiatry and Neurology, 17*(2), 61-67.

Meguro, K., Yamaguchi, S., Itoh, M., Fujiwara, T., & Yamadori, A. (1997). Striatal dopamine metabolism correlated with frontotemporal glucose utilization in Alzheimer's disease: A double-tracer PET study. *Neurology, 49*(4), 941-945.

Melamed, E. (1979). Early-morning dystonia. A late side effect of long-term levodopa therapy in Parkinson's disease. *Archives of Neurology, 36*(5), 308-310.

Middelkoop, H. A. M., Smilde-van Den Doel, D. A., Neven, A. K., Kamphuisen, H. A. C., & Springer, C. P. (1996). Subjective sleep characteristics of 1,485 males and females aged 50-93: Effects of sex and age, and factors related to self-evaluated quality of sleep. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 51*(3), 108-115.

Middleton, L. E., & Yaffe, K. (2009). Promising strategies for the prevention of dementia. *Archives of Neurology, 66*(10), 1210-1215.

Mihaere, K. M., Harris, R., Gander, P. H., Reid, P. M., Purdie, G., Robson, B., & Neill, A. (2009). Obstructive sleep apnea in New Zealand adults: Prevalence and risk factors among Māori and non-Māori. *Sleep, 32*(7), 949-956.

Mimeault, V., & Morin, C. M. (1999). Self-help treatment for insomnia: Bibliotherapy with and without professional guidance. *Journal of Consulting and Clinical Psychology, 67*(4), 511-519.

Ministry of Health. (2010). How much activity is recommended? *Physical activity* Retrieved March 5, 2012, from http://www.health.govt.nz/your-health/healthy-living/food-and-physical-activity/physical-activity/how-much-activity-recommended

Ministry of Health. (2012a). Neighbourhood deprivation (50+ years). *Socioeconomic determinants of health* Retrieved March 5, 2014, from http://www.health.govt.nz/nz-health-statistics/health-statistics-and-data-sets/maori-health-data-and-stats/tatau-kura-tangata-health-older-maori-chart-book/nga-awe-o-te-hauora-socioeconomic-determinants-health-50-years/neighbourhood-deprivation-50-years

Ministry of Health. (2012b). Population projections 50+ years. *Demographics* Retrieved April 6, 2014, from http://www.health.govt.nz/nz-health-statistics/health-statistics-and-data-sets/maori-health-data-and-stats/tatau-kura-tangata-health-older-maori-chart-book/tatauranga-taupori-demographics-50-years/population-projections-50-years

Mioshi, E., Dawson, K., Mitchell, J., Arnold, R., & Hodges, J. R. (2006). The Addenbrooke's Cognitive Examination revised (ACE-R): A brief cognitive test battery for dementia screening. *International Journal of Geriatric Psychiatry, 21*(11), 1078-1085.

Miranda-Castillo, C., Woods, B., Galboda, K., Oomman, S., Olojugba, C., & Orrell, M. (2010). Unmet needs, quality of life and support networks of people with dementia living at home. *Health and Quality of Life Outcomes, 8*, 132-145.

Mishima, K., Hishikawa, Y., & Okawa, M. (1998). Randomized, dim light controlled, crossover test of morning bright light therapy for rest-activity rhythm disorders in patients with vascular dementia and dementia of Alzheimer's type. *Chronobiology International, 15*(6), 647-654.

Mishima, K., Okawa, M., Hishikawa, Y., Hozumi, S., Hori, H., & Takahashi, K. (1994). Morning bright light therapy for sleep and behavior disorders in elderly patients with dementia. *Acta Psychiatrica Scandinavica, 89*(1), 1-7.

Mishima, K., Okawa, M., Shimizu, T., & Hishikawa, Y. (2001). Diminished melatonin secretion in the elderly caused by insufficient environmental illumination. *Journal of Clinical Endocrinology and Metabolism, 86*(1), 129-134.

Miu, D. K. Y., & Szeto, S. S. L. (2012). Sleep disturbances among a group of dementia participants. *Journal of Clinical Gerontology and Geriatrics, 3*(3), 105-109.

Moe, K. E., Vitiello, M. V., Larsen, L. H., & Prinz, P. N. (1995). Sleep/wake patterns in Alzheimer's disease: Relationships with cognition and function. *Journal of Sleep Research, 4*(1), 15-20.

Molano, J., & Vaughn, B. V. (2014). Approach to insomnia in patients with dementia. *Neurology: Clinical Practice, 4*(1), 7-15.

Monane, M., Glynn, R. J., & Avorn, J. (1996). The impact of sedative-hypnotic use on sleep symptoms in elderly nursing home residents. *Clinical Pharmacology and Therapeutics, 59*(1), 83-92.

Monk, T. H. (1995). Circadian temperature rhythms of older people. *Experimental Gerontology, 30*(5), 455-474.

Monk, T. H., Buysse, D., Hall, M., Nofzinger, E., Thompson, W. K., Mazumdar, S., & Reynolds Iii, C. (2006). Age-related differences in the lifestyle regularity of seniors experiencing bereavement, care-giving, insomnia, and advancement into old-old age. *Chronobiology International, 23*(4), 831-841.

Monk, T. M. (2010). Circadian rhythm dysregulation in the elderly: Shift work. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 143-149). Cambridge, England: Cambridge University Press.

Montgomery, P. (2002). Treatments for sleep problems in elderly people: Cognitive behavioural therapy is useful, but its benefits seem to be short lived. *British Medical Journal, 325*(7372), 1049-1049.

Montgomery, P., & Dennis, J. (2002). Bright light therapy for sleep problems in adults aged 60+. *Cochrane Database of Systematic Reviews,* (2). Retrieved from http://www.scopus.com/inward/record.url?eid=2-s2.0-0036051478&partnerID=40&md5=c930df7cf28f8a4f9c2d0c706d2984a0

Montgomery, P., & Dennis, J. (2003). Cognitive behavioural interventions for sleep problems in adults aged 60+. *Cochrane Database of Systematic Reviews,* (1). Retrieved from http://www.scopus.com/inward/record.url?eid=2-s2.0-0037265016&partnerID=40&md5=8189ce83317fd12f2f454e0e22267a60

Montgomery, P., & Dennis, J. (2004). A systematic review of non-pharmacological therapies for sleep problems in later life. *Sleep Medicine Reviews, 8*(1), 47-62.

Montgomery, R., Gonyea, J. G., & Hooyman, N. R. (1985). Caregiving and the experience of subjective and objective burden. *Family Relations, 34*(1), 19-26.

Monti, J. M., & Monti, D. (2010). Management of insomia in the elderly: The efficacy and safety of non-benzodiazepine hypnotics. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 343-361). Cambridge, England: Cambridge University Press.

Moore, R., Harmell, A. L., Chattillion, E., Ancoli-Israel, S., Grant, I., & Mausbach, B. T. (2011). PEAR model and sleep outcomes in dementia caregivers: Influence of activity restriction and pleasant events on sleep disturbances. *International Psychogeriatrics, 23*(9), 1462-1469.

Moore, T., & Hollett, J. (2003). Giving voice to persons living with dementia: The researcher's opportunities and challenges. *Nursing Science Quarterly, 16*(2), 163-167.

Moran, M., Lynch, C. A., Walsh, C., Coen, R., Coakley, D., & Lawlor, B. A. (2005). Sleep disturbance in mild to moderate Alzheimer's disease. *Sleep Medicine, 6*(4), 347-352.

Morgan, D. (1997). *Focus groups as qualitative research* (2nd ed.). Thousand Oaks, CA: Sage.

Morgan, K. (2003). Daytime activity and risk factors for late-life insomnia. *Journal of Sleep Research, 12*(3), 231-238.

Morgenthaler, T. I., Gay, P. C., Gordon, N., & Brown, L. K. (2007). Adaptive servoventilation versus noninvasive positive pressure ventilation for central, mixed, and complex sleep apnea syndromes. *Sleep, 30*(4), 468-475.

Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *Journal of the American Medical Association, 281*(11), 991-999.

Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *American Journal of Psychiatry, 151*(8), 1172-1180.

Morin, C. M., Kowatch, R. A., Barry, T., & Walton, E. (1993). Cognitive-behavior therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology, 61*(1), 137-146.

Morris, R. G. (1994). Working memory in Alzheimer-type dementia. *Neuropsychology, 8*(4), 544-554.

Most, E. I. S., Scheltens, P., & Van Someren, E. J. W. (2010). Prevention of depression and sleep disturbances in elderly with memory-problems by activation of the biological clock with light - a randomised clinical trial. *Trials, 11*. Retrieved from http://www.trialsjournal.com/content/11/1/19

Motohashi, Y., Maeda, A., Wakamatsu, H., Higuchi, S., & Yuasa, T. (2000). Circadian rhythm abnormalities of wrist activity of institutionalized dependent elderly persons with dementia. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 55*(12), 740-743.

Mrosovsky, N. (1996). Locomotor activity and non-photic influences on circadian clocks. *Biological Reviews of the Cambridge Philosophical Society, 71*(3), 343-372.

Mueller, S. G., Schuff, N., Yaffe, K., Madison, C., Miller, B., & Weiner, M. W. (2010). Hippocampal atrophy patterns in mild cognitive impairment and Alzheimer's disease. *Human Brain Mapping, 31*(9), 1339-1347.

Murphy, M. R., Escamilla, M. I., Blackwell, P. H., Lucke, K. T., Miner-Williams, D., Shaw, V., & Lewis, S. L. (2007). Focus on research methods assessment of caregivers' willingness to participate in an intervention research study. *Research in Nursing and Health, 30*(3), 347-355.

Murtagh, D. R. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: A meta-analysis. *Journal of Consulting and Clinical Psychology, 63*(1), 79-89.

Naismith, S. L., Rogers, N. L., Hickie, I. B., MacKenzie, J., Norrie, L. M., & Lewis, S. J. (2010). Sleep well, think well: Sleep-wake disturbance in mild cognitive impairment. *Journal of Geriatric Psychiatry and Neurology, 23*(2), 123-130.

Namazi, K. H., Zadorozny, C. A., & Gwinnup, A. B. (1995). The influences of physical activity on patterns of sleep behavior of patients with Alzheimer's disease. *International Journal of Aging and Human Development, 40*(2), 145-153.

Nasreddine, Z. S., Phillips, N. A., Bedirian, V., Charbonneau, S., Whitehead, V., Collin, I., . . . Chertkow, H. (2005). The montreal cognitive assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*(4), 695-699.

National Ethics Advisory Committee. (2012a). *Ethical guidelines for intervention studies* (Revised ed.). Wellington, New Zealand: Ministry of Health.

National Ethics Advisory Committee. (2012b). *Ethical guidelines for observational studies:Observational research, audits and related activities.* (Revised ed.). Wellington, New Zealand: Ministry of Health.

National Institute of Aging. (2009). Age page: A good night's sleep. *Health and aging* Retrieved March 8, 2013, from http://www.nia.nih.gov/health/publication/good-nights-sleep

National Institute of Health Consensus Group. (1991). National Institutes of Health Consensus Development Conference statement: The treatment of sleep disorders of older people March 26-28, 1990. *Sleep, 14*(2), 169-177.

National Sleep Foundation. (2011). Sleep hygiene. *Ask the expert* Retrieved September 2, 2011, from http://www.sleepfoundation.org/article/ask-the-expert/sleep-hygiene

Nausieda, P. A., Weiner, W. J., Kaplan, L. R., Weber, S., & Klawans, H. L. (1982). Sleep disruption in the course of chronic levodopa therapy: An early feature of the levodopa psychosis. *Clinical Neuropharmacology, 5*(2), 183-194.

Naylor, E., Penev, P. D., Orbeta, L., Janssen, I., Ortiz, R., Colecchia, E. F., . . . Zee, P. C. (2000). Daily social and physical activity increases slow-wave sleep and daytime neuropsychological performance in the elderly. *Sleep, 23*(1), 87-95.

Neutel, C. I. (2005). The epidemiology of long-term benzodiazepine use. *International Review of Psychiatry, 17*(3), 189-197.

Neutel, C. I., & Patton, S. B. (2010). Epidemiology of sleep medication use in the elderly. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 332-343). Cambridge, England: Cambridge University Press.

Nolan, M., Grant, C., & keady, J. (1996). *Understanding family care: A multidimensional model of caring and coping.* Buckingham, England: Open University Press.

Nowak, L., & Davis, J. E. (2007). A qualitative examination of the phenomenon of sundowning. *Journal of Nursing Scholarship, 39*(3), 256-258.

Nygård, L. (2006). How can we get access to the experiences of people with dementia? *Scandinavian Journal of Occupational Therapy, 13*(2), 101-112.

Nygård, M., Hill, R. H., Wikström, M. A., & Kristensson, K. (2005). Age-related changes in electrophysiological properties of the mouse suprachiasmatic nucleus in vitro. *Brain Research Bulletin, 65*(2), 149-154.

O'Keeffe, S. T., Gavin, K., & Lavan, J. N. (1994). Iron status and restless legs syndrome in the elderly. *Age and Ageing, 23*(3), 200-203.

Oeppen, J., & Vaupel, J. W. (2002). Demography: Broken limits to life expectancy. *Science, 296*(5570), 1029-1031.

Ohashi, Y., Okamoto, N., Uchida, K., Iyo, M., Mori, N., & Morita, Y. (1999). Daily rhythm of serum melatonin levels and effect of light exposure in patients with dementia of the Alzheimer's type. *Biological Psychiatry, 45*(12), 1646-1652.

Ohayon, M. M. (2002). Epidemiology of insomnia: What we know and what we still need to learn. *Sleep Medicine Reviews, 6*(2), 97-111.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*(7), 1255-1273.

Ohayon, M. M., & Roth, T. (2002). Prevalence of restless legs syndrome and periodic limb movement disorder in the general population. *Journal of Psychosomatic Research, 53*(1), 547-554.

Ohayon, M. M., & Vecchierini, M. F. (2002). Daytime sleepiness and cognitive impairment in the elderly population. *Archives of Internal Medicine, 162*(2), 201-208.

Ohayon, M. M., Zulley, J., Guilleminault, C., Smirne, S., & Priest, R. G. (2001). How age and daytime activities are related to insomnia in the general population: Consequences for older people. *Journal of the American Geriatrics Society, 49*(4), 360-366.

Okudaira, N., Kripke, D. F., & Webster, J. B. (1983). Naturalistic studies of human light exposure. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 14*(4), 613-615.

Okumoto, Y., Koyama, Emi., Matsubara, Hozumi., Nakano, Toshio., Nakamura, Reizo. (1998). Sleep improvement by light in a demented aged individual. *Japanese Society of Sleep Research*

Olde Rikkert, M. G. M., & Rigaud, A. S. P. (2001). Melatonin in elderly patients with insomnia: A systematic review. *Zeitschrift fur Gerontologie und Geriatrie, 34*(6), 491-497.

Orrell, M., Butler, R., & Bebbington, P. (2000). Social factors and the outcome of dementia. *International Journal of Geriatric Psychiatry, 15*(6), 515-520.

Ory, M. G., Hoffman Iii, R. R., Yee, J. L., Tennstedt, S., & Schulz, R. (1999). Prevalence and impact of caregiving: A detailed comparison between dementia and nondementia caregivers. *Gerontologist, 39*(2), 177-185.

Oster, H., Baeriswyl, S., van der Horst, G. T. J., & Albrecht, U. (2003). Loss of circadian rhythmicity in aging mPer1-/- mCry2-/- mutant mice. *Genes and Development, 17*(11), 1366-1379.

Ownby, R. L., Saeed, M., Wohlgemuth, W., Capasso, R., Acevedo, A., Peruyera, G., & Sevush, S. (2010). Caregiver reports of sleep problems in non-Hispanic white, Hispanic, and African American patients with Alzheimer dementia. *Journal of Clinical Sleep Medicine, 6*(3), 281-289.

Paine, S. J., & Gander, P. H. (2013). Sleep, sleepiness, and sleep disorders: Principles for examining differences by ethnicity. In C. Kushida (Ed.), *The Encyclopedia of Sleep,* (Vol. 2, pp. 691-698). Ealtham, MA: Academic Press.

Paine, S. J., Gander, P. H., Harris, R., & Reid, P. (2004). Who reports insomnia? Relationships with age, sex, ethnicity, and socioeconomic deprivation. *Sleep, 27*(6), 1163-1169.

Paine, S. J., Gander, P. H., Harris, R. B., & Reid, P. (2005). Prevalence and consequences of insomnia in New Zealand: Disparities between Maori and non-Maori. *Australian and New Zealand Journal of Public Health, 29*(1), 22-28.

Paine, S. J., Gander, P. H., & Travier, N. (2006). The epidemiology of morningness/eveningness: influence of age, gender, ethnicity, and socioeconomic factors in adults (30-49 years). *Jounral of Sleep and Biological Rhythms, 21*(1), 68-76.

Paine, S. J., Harris, R., & Mihaere, K. M. (2007). Sleep problems. In B. Robson & R. Harris (Eds.), *Hauora: Maori Standards of Health* (Vol. 4, pp. 199-207). Wellington, New Zealand: Te Ropu Rangahau Hauora a Eru Pomare.

Pandi-Perumal, S. R., Spence, D. W., & Sharma, V. K. (2010). Aging and circadian rhythms: General trends. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 3-21). Cambridge, England: Cambridge University Press.

Pat-Horenczyk, R., Klauber, M. R., Shochat, T., & Ancoli-Israel, S. (1998). Hourly profiles of sleep and wakefulness in severely versus mild- moderately demented nursing home patients. *Aging Clinical and Experimental Research, 10*(4), 308-315.

Patel, J., Renvoize, E., Higham, S., Crawford, T., & Suriya, A. (2005). The human face of dementia research. *Clinical Psychology Forum*(152), 33-36.

Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior, 19*(1), 2-21.

Peigneux, P., & Smith, C. (2011). Memory processing in relation to sleep. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 335-347). St. Louis, MO: Saunders, Elsiver Inc.

Peng, H. L., & Chang, Y. P. (2013). Sleep disturbance in family caregivers of individuals with dementia: A review of the literature. *Perspectives in Psychiatric Care, 49*(2), 135-146.

Penrose, F. K. (2005). Can exercise affect cognitive functioning in Alzheimer's disease? A review of the literature. *Activities, Adaptation and Aging, 29*(4), 15-40.

Perri, M., 3rd. , Menon, A. M., Deshpande, A. D., Shinde, S. B., Jiang, R., Cooper, J. W., . . . Lorys, R. A. (2005). Adverse outcomes associated with inappropriate drug use in nursing homes. *Annals of Pharmacotherapy, 39*(3), 405-411.

Perry, E., Walker, M., Grace, J., & Perry, R. (1999). Acetylcholine in mind: A neurotransmitter correlate of consciousness? *Trends in Neurosciences, 22*(6), 273-280.

Perry, R., & Hodges, J. R. (1999). Attention and executive deficits in Alzheimer's disease. A critical review. *Brain, 122*(3), 383-404.

Petersen, R. C., Doody, R., Kurz, A., Mohs, R. C., Morris, J. C., Rabins, P. V., . . . Winblad, B. (2001). Current concepts in mild cognitive impairment. *Archives of Neurology, 58*(12), 1985-1992.

Petersen, R. C., Parisi, J. E., Dickson, D. W., Johnson, K. A., Knopman, D. S., Boeve, B. F., . . . Kokmen, E. (2006). Neuropathologic features of amnestic mild cognitive impairment. *Archives of Neurology, 63*(5), 665-672.

Petersen, R. C., Roberts, R. O., Knopman, D. S., Boeve, B. F., Geda, Y. E., Ivnik, R. J., . . . Jack, C. R., Jr. (2009). Mild cognitive impairment: Ten years later. *Archives of Neurology, 66*(12), 1447-1455.

Petersen, R. C., Stevens, J. C., Ganguli, M., Tangalos, E. G., Cummings, J. L., & DeKosky, S. T. (2001). Practice parameter: Early detection of dementia: Mild cognitive impairment (an evidence-based review). *Neurology, 56*(9), 1133-1142.

Petersen, R. C., Thomas, R. G., Grundman, M., Bennett, D., Doody, R., Ferris, S., . . . Thal, L. J. (2005). Vitamin E and donepezil for the treatment of mild cognitive impairment. *New England Journal of Medicine, 352*(23), 2379-2388.

Petit, D., Montplaisir, J., & Boeve, B. F. (2011). Alzheimer's disease and other dementias. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 1038-1047). St. Louis, MO: Saunders, Elsiver Inc.

Phillips, B., & Ancoli-Israel, S. (2001). Sleep disorders in the elderly. *Sleep Medicine, 2*(2), 99-114.

Phillips, B., Hening, W., Britz, P., & Mannino, D. (2006). Prevalence and correlates of restless legs syndrome: Results from the 2005 National Sleep Foundation Poll. *Chest, 129*(1), 76-80.

Phillips, B., Young, T., Finn, L., Asher, K., Hening, W. A., & Purvis, C. (2000). Epidemiology of restless legs symptoms in adults. *Archives of Internal Medicine, 160*(14), 2137-2141.

Plihal, W., & Born, J. (1997). Effects of early and late nocturnal sleep on declarative and procedural memory. *Journal of Cognitive Neuroscience, 9*(4), 534-547.

Pollak, C. P., & Perlick, D. (1991). Sleep problems and institutionalization of the elderly. *Journal of Geriatric Psychiatry and Neurology, 4*(4), 204-210.

Pollak, C. P., & Stokes, P. E. (1997). Circadian rest-activity rhythms in demented and nondemented older community residents and their caregivers. *Journal of the American Geriatrics Society, 45*(4), 446-452.

Pollak, C. P., Tryon, W. W., Nagaraja, H., & Dzwonczyk, R. (2001). How accurately does wrist actigraphy identify the states of sleep and wakefulness? *Sleep, 24*(8), 957-965.

Post, S. G. (2003). Full-spectrum proxy consent for research participation when persons with Alzheimer disease lose decisional capacities: Research ethics and the common good. *Alzheimer Disease and Associated Disorders, 17*(1), 3-11.

Pratt, R. (2002). 'Nobody's ever asked how I felt'. In H. Wilkinson (Ed.), *The perspectives of people with dementia: Research methods and motivations* (pp. 165-182). London, England: Jessica Kingsley Publishers.

Prinz, P. N., Peskind, E. R., & Vitaliano, P. P. (1982). Changes in the sleep and waking EEGs of nondemented and demented elderly subjects. *Journal of the American Geriatrics Society, 30*(2), 86-93.

Prinz, P. N., Vitaliano, P. P., Vitiello, M. V., Bokan, J., Raskind, M., Peskind, E., & Gerber, C. (1982). Sleep, EEG and mental function changes in senile dementia of the Alzheimer's type. *Neurobiology of Aging, 3*(4), 361-370.

Qazi, A., Spector, A., & Orrell, M. (2010). User, carer and staff perspectives on anxiety in dementia: A qualitative study. *Journal of Affective Disorders, 125*(1-3), 295-300.

Quality Metric. (2013). SF health surveys. *What we do* Retrieved August 2, 2013, from http://www.qualitymetric.com/WhatWeDo/SFHealthSurveys/tabid/184/Default.aspx

Qureshi, A., & Johri, A. (2008). Issues involving informed consent for research participants with Alzheimer's disease. *Journal of Academic Ethics, 6*(3), 197-203.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*(1), 385-401.

Ragin, C. C. (1992). Introduction Cases of "what is a case". In C. C. Ragin & H. S. Becker (Eds.), *What is a case?* (pp. 1-18). Cambridge, England: Cambridge University Press.

Rajaratnam, S. M. W., & Arendt, J. (2001). Health in a 24-h society. *Lancet, 358*(9286), 999-1005.

Ralph, M. R., Foster, R. G., Davis, F. C., & Menaker, M. (1990). Transplanted suprachiasmatic nucleus determines circadian period. *Science, 247*(4945), 975-978.

Rapp, S. R., & Chao, D. (2000). Appraisals of strain and of gain: Effects on psychological wellbeing of caregivers of dementia patients. *Aging and Mental Health, 4*(2), 142-147.

Raskind, M. A. (1998). Psychopharmacology of noncognitive abnormal behaviors in Alzheimer's disease. *Journal of Clinical Psychiatry, 59*(9), 28-32.

Ray, W. A., Griffin, M. R., Schaffner, W., Baugh, D. K., & Melton, L. J., 3rd. (1987). Psychotropic drug use and the risk of hip fracture. *New England Journal of Medicine, 316*(7), 363-369.

Rebok, G. W., Rovner, B. W., & Folstein, M. F. (1991). Sleep disturbance and Alzheimer's disease: Relationship to behavioral problems. *Aging - Clinical and Experimental Research, 3*(2), 193-196.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standadised terminology, techniques and scoring systemsfor sleep stages of human subjects*. Los Angeles, CA: University of California.

Reid, D., Ryan, T., & Enderby, P. (2001). What does it mean to listen to people with dementia? *Disability and Society, 16*(3), 377-392.

Reid, K., Martinovich, Z., Finkel, S., Harter, K., & Zee, P. C. (2006). Sleep: A marker of physical and mental health in the elderly. *The American Journal of Geriatric Psychiatry 14*(10), 860-866.

Reinkes, J., Sparrow, M., & Campbell, A. J. (1982). The giving and taking of psychotropic drugs in New Zealand. *New Zealand Medical Journal, 95*(712), 489-492.

Reisberg, B., Doody, R., Stöffler, A., Schmitt, F., Ferris, S., & Möbius, H. J. (2003). Memantine in moderate-to-severe Alzheimer's disease. *New England Journal of Medicine, 348*(14), 1333-1341.

Reynolds, C. F., Kupfer, D. J., Hoch, C. C., & Sewitch, D. E. (1985). Sleeping pills for the elderly: Are they ever justified? *Journal of Clinical Psychiatry, 46*(2 II), 9-12.

Riemersma-van Der Lek, R. F., Swaab, D. F., Twisk, J., Hol, E. M., Hoogendijk, W. J. G., & Van Someren, E. J. W. (2008). Effect of bright light and melatonin on cognitive and noncognitive function in elderly residents of group care facilities: A randomized controlled trial. *Journal of the American Medical Association, 299*(22), 2642-2655.

Rogers, S. L., Farlow, M. R., Doody, R. S., Mohs, R., Friedhoff, L. T., Ieni, J., & Warner, J. P. (1998). Donepezil improved cognitive and global function in mild-to-moderate Alzheimer disease. *Evidence-Based Medicine, 3*(5), 155.

Rolland, Y., Pillard, F., Klapouszczak, A., Reynish, E., Thomas, D., Andrieu, S., . . . Vellas, B. (2007). Exercise program for nursing home residents with Alzheimer's disease: A 1-year randomized, controlled trial. *Journal of the American Geriatrics Society, 55*(2), 158-165.

Rose, K. M., Beck, C., Tsai, P. F., Liem, P. H., Davila, D. G., Kleban, M., . . . Richards, K. C. (2011). Sleep disturbances and nocturnal agitation behaviors in older adults with dementia. *Sleep, 34*(6), 779-786.

Rosenwasser, A. M., & Turek, F. W. (2011). Physiology of the mammillian circadian system. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 390-401). St. Louis, MO: Saunders, Elsiver Inc.

Ross, J. S., & Shua-Haim, J. R. (1998). Aricept-induced nightmares in Alzheimer's disease: 2 case reports. *Journal of the American Geriatrics Society, 46*(1), 119-120.

Roud, H., Keeling, S., & Sainsbury, R. (2006). Using the COPE assessment tool with informal carers of people with dementia in New Zealand. *New Zealand Medical Journal, 119*(1237)

Rowe, M. A., & Fehrenbach, N. (2004). Injuries sustained by community-dwelling individuals with dementia. *Clinical Nursing Research, 13*(2), 98-110.

Rowe, M. A., McCrae, C. S., Campbell, J. M., Benito, A. P., & Cheng, J. (2008). Sleep pattern differences between older adult dementia caregivers and older adult noncaregivers using objective and subjective measures. *Journal of Clinical Sleep Medicine, 4*(4), 362-369.

Ruehland, W. R., Rochford, P. D., O'Donoghue, F. J., Pierce, R. J., Singh, P., & Thornton, A. T. (2009). The new AASM criteria for scoring hypopneas: Impact on the apnea hypopnea index. *Sleep, 32*(2), 150-157.

Ruiter, M. E., DeCoster, J., Jacobs, L., & Lichstein, K. L. (2010). Sleep disorders in African Americans and Caucasian Americans: A meta-analysis. *Behavioral Sleep Medicine, 8*(4), 246-259.

Ruiter, M. E., Vander Wal, G. S., & Lichstein, K. L. (2010). Insomnia in the elderly. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 271-279). Cambridge, England: Cambridge University Press.

Sabat, S. R. (2005). Capacity for decision-making in Alzheimer's disease: Selfhood, positioning and semiotic people. *Australian and New Zealand Journal of Psychiatry, 39*(11-12), 1030-1035.

Sabat, S. R., & Harfe, R. (1992). The construction and deconstruction of the self in Alzheimer's disease. *Ageing and Society, 12*, 443-461.

Sack, R. L., Auckley, D., Auger, R. R., Carskadon, M. A., Wright, K. P., Jr., Vitiello, M. V., & Zhdanova, I. V. (2007). Circadian rhythm sleep disorders: Part I, basic principles, shift work and jet lag disorders: An American Academy of Sleep Medicine review. *Sleep, 30*(11), 1460-1483.

Sack, R. L., Brandes, R. W., Kendall, A. R., & Lewy, A. J. (2000). Entrainment of free-running circadian rhythms by melatonin in blind people. *New England Journal of Medicine, 343*(15), 1070-1077.

Sack, R. L., Lewy, A. J., & Hughes, R. J. (1998). Use of melatonin for sleep and circadian rhythm disorders. *Annals of Medicine, 30*(1), 115-121.

Sacre, S. (2010). Improving the sleep of carers of people with dementia through the use of a simple information package. *Proceedings of the Sleep Down Under meeting of the Australasian Sleep Association: Sleep and Biological Rhythms, 8 (Suppl1)*

Sadeh, A., & Anders, T. F. (1993). Infant sleep problems - origins, assessment, interventions. *Infant Ment Health J, 14*(1), 17-34.

Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep, 18*(4), 288-302.

Sadeh, A., Sharkey, K. M., & Carskadon, M. A. (1994). Activity-based sleep-wake identification - an empirical-test of methodological issues. *Sleep, 17*(3), 201-207.

Salami, O., Lyketsos, C., & Rao, V. (2011). Treatment of sleep disturbance in Alzheimer's dementia. *International Journal of Geriatric Psychiatry, 26*(8), 771-782.

Salmond, C., Crampton, P., & Atkinson, J. (2007). NZDep2006 index of deprivation: Users manual Retrieved from http://www.otago.ac.nz/wellington/otago020337.pdf

Salzman, C., Shader, R. I., Greenblatt, D. J., & Harmatz, J. S. (1983). Long v. short half-life benzodiazepines in the elderly. Kinetics and clinical effects of diazepam and oxazepam. *Archives of General Psychiatry, 40*(3), 293-297.

Sangha, O., Stucki, G., Liang, M. H., Fossel, A. H., & Katz, J. N. (2003). The Self-Administered Comorbidity Questionnaire: A new method to assess comorbidity for clinical and health services research. *Arthritis Care and Research, 49*(2), 156-163.

Saper, C. B., Chou, T. C., & Scammell, T. E. (2001). The sleep switch: Hypothalamic control of sleep and wakefulness. *Trends in Neurosciences, 24*(12), 726-731.

Saper, C. B., Lu, J., Chou, T. C., & Gooley, J. (2005). The hypothalamic integrator for circadian rhythms. *Trends in Neurosciences, 28*(3), 152-157.

Saper, C. B., Scammell, T. E., & Lu, J. (2005). Hypothalamic regulation of sleep and circadian rhythms. *Nature, 437*(7063), 1257-1263.

Sarter, M., & Bruno, J. P. (2004). Developmental origins of the age-related decline in cortical cholinergic function and associated cognitive abilities. *Neurobiology of Aging, 25*(9), 1127-1139.

Satlin, A., Volicer, L., Ross, V., Herz, L., & Campbell, S. (1992). Bright light treatment of behavioral and sleep disturbances in patients with Alzheimers-disease. *American Journal of Psychiatry, 149*(8), 1028-1032.

Satlin, A., Volicer, L., Stopa, E. G., & Harper, D. (1995). Circadian locomotor activity and core-body temperature rhythms in Alzheimer's disease. *Neurobiology of Aging, 16*(5), 765-771.

Scarmeas, N., Luchsinger, J. A., Schupf, N., Brickman, A. M., Cosentino, S., Tang, M. X., & Stern, Y. (2009). Physical activity, diet, and risk of Alzheimer disease. *Journal of the American Medical Association, 302*(6), 627-637.

Scheer, F. A., & Shea, S. A. (2007). Fundamentals of the circadian system. In C. J. Amlaner & O. M. Buxton (Eds.), Slides to accompany the Sleep Research Society Basics of Sleep Guide (1.1 ed.). Darien, IL: Sleep Research Society.

Schneider, L. S., Dagerman, K., & Insel, P. S. (2006). Efficacy and adverse effects of atypical antipsychotics for dementia: Meta-analysis of randomized, placebo-controlled trials. *American Journal of Geriatric Psychiatry, 14*(3), 191-210.

Schnelle, J. F., Cruise, P. A., Alessi, C. A., Ludlow, K., Al-Samarrai, N. R., & Ouslander, J. G. (1998). Sleep hygiene in physically dependent nursing home residents: Behavioral and environmental intervention implications. *Sleep, 21*(5), 515-523.

Schredl, M., Weber, B., Braus, D., & Heuser, I. (2000). The effect of rivastigmine on sleep in elderly healthy subjects. *Experimental Gerontology, 35*(2), 243-249.

Schredl, M., Weber, B., Leins, M. L., & Heuser, I. (2001). Donepezil-induced REM sleep augmentation enhances memory performance in elderly, healthy persons. *Experimental Gerontology, 36*(2), 353-361.

Schulz, R., O'Brien, A., Czaja, S., Ory, M., Norris, R., Martire, L. M., . . . Stevens, A. (2002). Dementia caregiver intervention research: In search of clinical significance. *Gerontologist, 42*(5), 589-602.

Serfaty, M., Kennell-Webb, S., Warner, J., Blizard, R., & Raven, P. (2002). Double blind randodmised placebo controlled trial of low dose melatonin for sleep disorders in dementia. *International Journal of Geriatric Psychiatry, 17*(12), 1120-1127.

Shearman, L. P., Sriram, S., Weaver, D. R., Maywood, E. S., Chaves, I., Zheng, B., . . . Reppert, S. M. (2000). Interacting molecular loops in the mammalian circadian clock. *Science, 288*(5468), 1013-1019.

Sheikh, J. I., & Yesavage, J. A. (1986). Geriatric Depression Scale (GDS): Recent evidence and development of a shorter version. *Clinical Gerontologist, 5*(1-2), 165-173.

Shelton, P. S., Hocking, L. B., Guzman, W. M., & Demers, D. (1997). Zolpidem for dementia-related insomnia and nighttime wandering. *Annals of Pharmacotherapy, 31*(3), 319-322.

Sherratt, C., Soteriou, T., & Simon, E. (2007). Ethical issues in social research involving people with dementia. *Dementia, 6*(4), 463-479.

Sherrill, D. L., Kotchou, K., & Quan, S. F. (1998). Association of physical activity and human sleep disorders. *Archives of Internal Medicine, 158*(17), 1894-1898.

Shirani, A., Paradiso, S., & Dyken, M. E. (2011). The impact of atypical antipsychotic use on obstructive sleep apnea: A pilot study and literature review. *Sleep Medicine, 12*(6), 591-597.

Shochat, T., Martin, J., Marler, M., & Ancoli-Israel, S. (2000). Illumination levels in nursing home patients: Effects on sleep and activity rhythms. *Journal of Sleep Research, 9*(4), 373-379.

Signal, T. L., Gale, J., & Gander, P. H. (2005). Sleep measurement in flight crew: Comparing actigraphic and subjective estimates to polysomnography. *Aviation Space and Environmental Medicine, 76*(11), 1058-1063.

Silva, E. J., Wang, W., Ronda, J. M., Wyatt, J. K., & Duffy, J. F. (2010). Circadian and wake-dependent influences on subjective sleepiness, cognitive throughput, and reaction time performance in older and young adults. *Sleep, 33*(4), 481-490.

Simpson, C., & Carter, P. (2013a). Dementia caregivers' lived experience of sleep. *Clinical Nurse Specialist, 27*(6), 298-306.

Simpson, C., & Carter, P. (2013b). Short-term changes in sleep, mastery & stress: Impacts on depression and health in dementia caregivers. *Geriatric Nursing, 34*(6), 509-516.

Singer, C., Tractenberg, R. E., Kaye, J., Schafer, K., Gamst, A., Grundman, M., . . . Thal, L. J. (2003). A multicenter, placebo-controlled trial of melatonin for sleep disturbance in Alzheimer's disease. *Sleep, 26*(7), 893-901.

Singer, M., Romero, B., Koenig, E., Förstl, H., & Brunner, H. (2005). Nightmares in patients with Alzheimer's disease caused by donepezil. Therapeutic effect depends on the time of intake. *Nervenartz, 76*(9), 1127-1129.

Sirven, J. I., & Mancall, E. L. (2008). Neurological examination of the older adult. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology of the older adult* (Vol. 2, pp. 5-7). Philadelphia, PA: Wolters Kluwer: Lippincott Williams & Wilkins.

Sivertsen, B., Omvik, S., Havik, O. E., Pallesen, S., Bjorvatn, B., Nielsen, G. H., . . . Nordhus, I. H. (2006). A comparison of actigraphy and polysomnography in older adults treated for chronic primary insomnia. *Sleep, 29*(10), 1353-1358.

Skevington, S. M., Lotfy, M., & O'Connell, K. A. (2004). The World Health Organization's WHOQOL-BREF quality of life assessment: Psychometric properties and results of the international field trial a Report from the WHOQOL Group. *Quality of Life Research, 13*(2), 299-310.

Slaughter, S., Cole, D., Jennings, E., & Reimer, M. A. (2007). Consent and assent to participate in research from people with dementia. *Nursing Ethics, 14*(1), 27-40.

Sloane, P. D., Williams, C. S., Mitchell, M., Preisser, J. S., Wood, W., Barrick, A. L., . . . Zimmerman, S. (2007). High intensity environmental light in dementia: Effect on sleep and activity. *The American Geriatrics Society, 55*(10), 152401533.

Small, G. W., Rabins, P. V., Barry, P. P., Buckholtz, N. S., DeKosky, S. T., Ferris, S. H., . . . Tune, L. E. (1997). Diagnosis and treatment of Alzheimer disease and related disorders: Consensus statement of the American Association for Geriatric Psychiatry, the Alzheimer's Association, and the American Geriatrics Society. *Journal of the American Medical Association, 278*(16), 1363-1371.

Smith, C., Aubrey, J. B., & Peters, K. R. (2004). Different roles for REM and Stage 2 sleep in motor learning: A proposed model. *Psychologica Belgica, 44*(1-2), 81-104.

Smith, S., Murray, J., Banerjee, S., Foley, B., Cook, J. C., Lamping, D. L., . . . Mann, A. (2005). What constitutes health-related quality of life in dementia? Development of a conceptual framework for people with dementia and their carers. *International Journal of Geriatric Psychiatry, 20*(9), 889-895.

Solms, M. (1997). *The neuropsychology of dreams: A clinico-anatomical study*. Mahwah, NJ: Lawrence Erlbaum.

Song, Y., Dowling, G. A., Wallhagen, M. I., Lee, K. A., & Strawbridge, W. J. (2010). Sleep in older adults with Alzheimer's disease. *Journal of Neuroscience Nursing, 42*(4), 190-198.

Spector, A., Thorgrimsen, L., Woods, B., Royan, L., Davies, S., Butterworth, M., & Orrell, M. (2003). Efficacy of an evidence-based cognitive stimulation therapy programme for people with dementia: Randomised controlled trial. *British Journal of Psychiatry, 183*, 248-254.

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perpective on insomnia treatment. *Psychiatric Clinics of North America, 10*(4), 541-553.

Sport and Recreation New Zealand. (2003). Push Play: Stretching. *SPARC (Sport and Recreation New Zealand Ihi Aotearoa)*. Retrieved from www.pushplay.org.nz

Sproule, B. A., Busto, U. E., Buckle, C., Herrmann, N., & Bowles, S. (1999). The use of non-prescription sleep products in the elderly. *International Journal of Geriatric Psychiatry, 14*(10), 851-857.

Squire, L. R. (2004). Memory systems of the brain: A brief history and current perspective. *Neurobiology of Learning and Memory, 82*(3), 171-177.

Squire, L. R., & Zola, S. M. (1996). Structure and function of declarative and nondeclarative memory systems. *Proceedings of the National Academy of Sciences of the United States of America, 93*(24), 13515-13522.

Staedt, J., & Stoppe, G. (2005). Treatment of rest-activity disorders in dementia and special focus on sundowning. *International Journal of Geriatric Psychiatry 20*, 507-511.

Stahl, S. M., Markowitz, J. S., Gutterman, E. M., & Papadopoulos, G. (2003). Co-use of donepezil and hypnotics among Alzheimer's disease patients living in the community. *Journal of Clinical Psychiatry, 64*(4), 466-472.

Staner, L., Demazieres, A., & Luthringer, R. (2010). Use of psychotropic drugs in the elderly: effects of sleep architecture. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 371-383). Cambridge, England: Cambridge University Press.

Staples, V. S. L., Archer, S. N., Arber, S., & Skene, D. J. (2009). Daily light exposure profiles in older non-resident extreme morning and evening types. *Journal of Sleep Research, 18*(4), 466-471.

Statistics New Zealand. (2006a). 2006 Census. *Census* Retrieved June 24, 2014, from http://www.stats.govt.nz/Census/2006CensusHomePage.aspx

Statistics New Zealand. (2006b). Demographic aspects of New Zealand's ageing population. *People and communities* Retrieved April 27, 2013, from http://www.stats.govt.nz/browse_for_stats/people_and_communities/older_people/demographic-aspects-nz-ageing-population.aspx

Statistics New Zealand. (2012). Statistics New Zealand: Life expectancy. *Health* Retrieved April 27, 2013, from http://www.stats.govt.nz/browse_for_stats/health/life_expectancy.aspx

Stephens, M. A. P., Franks, M. M., & Atienza, A. A. (1997). Where two roles intersect: Spillover between parent care and employment. *Psychology and Aging, 12*(1), 30-37.

Stern, Y., Gurland, B., Tatemichi, T. K., Tang, M. X., Wilder, D., & Mayeux, R. (1994). Influence of education and occupation on the incidence of Alzheimer's disease. *Journal of the American Medical Association, 271*(13), 1004-1010.

Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature, 437*(7063), 1272-1278.

Stocking, C. B., Hougham, G. W., Danner, D. D., Patterson, M. B., Whitehouse, P. J., & Sachs, G. A. (2006). Speaking of research advance directives: Planning for future research participation. *Neurology, 66*(9), 1361-1366.

Stone, K., & Ancoli-Israel, S. (2011). Actigraphy. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 1668-1675). St. Louis, MO: Saunders, Elsiver Inc.

Stone, K., Ensrud, K. E., & Ancoli-Israel, S. (2008). Sleep, insomnia and falls in elderly patients. *Sleep Medicine, 9*(1), S18-S22.

Stopa, E. G., Volicer, L., Kuo-Leblanc, V., Harper, D., Lathi, D., Tate, B., & Satlin, A. (1999). Pathologic evaluation of the human suprachiasmatic nucleus in severe dementia. *Journal of Neuropathology and Experimental Neurology, 58*(1), 29-39.

Stopford, C. L., Thompson, J. C., Neary, D., Richardson, A. M. T., & Snowden, J. S. (2012). Working memory, attention, and executive function in Alzheimer's disease and frontotemporal dementia. *Cortex, 48*(4), 429-446.

Strain, L. A., Blandford, A. A., Mitchell, L. A., & Hawranik, P. G. (2003). Cognitively Impaired Older Adults: Risk Profiles for Istitutionalization. *International Psychogeriatrics, 15*(4), 351-366.

Stuart-Hamilton, I. (2006). *The Psychology of Ageing* (4th ed.). London, England: Jessica Kingsley Publishers,.

Sullivan, C., Issa, F. G., Berthon-Jones, M., & Eves, L. (1981). Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *Lancet, 1*(8225), 862-865.

Sullivan, S., & Richards, K. C. (2004). Predictors or circadian sleep-wake rhythm maintenance in elders with dementia. *Aging and Mental Health, 8*(2), 143-152.

Sumaya, I. C., Rienzi, B. M., Deegan Ii, J. F., & Moss, D. E. (2001). Bright light treatment decreases depression in institutionalized older adults: A placebo-controlled crossover study. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 56*(6), 356-360.

Swaab, D. F., Fliers, E., & Partiman, T. S. (1985). The suprachiasmatic nucleus of the human brain in relation to sex, age and senile dementia. *Brain Research, 342*(1), 37-44.

Swaab, D. F., Van Someren, E. J. W., Zhou, J. N., & Hofman, M. A. (1996). Biological rhythms in the human life cycle and their relationship to functional changes in the suprachiasmatic nucleus. *Progress in Brain Research, 111*, 349-368.

Swain, J., Heyman, B., & Gillman, M. (1998). Public research, private concerns: Ethical issues in the use of open-ended interviews with people who have learning difficulties. *Disability and Society, 13*(1), 21-36.

Sweeting, H., & Gilhooly, M. (1997). Dementia and the phenomenon of social death. *Sociology of Health and Illness, 19*(1), 93-117.

Tanaka, H., Taira, K., Arakawa, M., Toguti, H., Urasaki, C., Yamamoto, Y., . . . Shirakawa, S. (2001). Effects of short nap and exercise on elderly people having difficulty in sleeping. *Psychiatry and Clinical Neurosciences, 55*(3), 173-174.

Taylor, D. J., Jenni, O. G., Acebo, C., & Carskadon, M. A. (2005). Sleep tendency during extended wakefulness: Insights into adolescent sleep regulation and behavior. *Journal of Sleep Research, 14*(3), 239-244.

Telford, R., Beverley, C. A., Cooper, C. L., & Boote, J. D. (2002). Consumer involvement in health research: Fact or fiction? *British Journal of Clinical Governance, 7*(2), 92-103.

Teri, L., Gibbons, L. E., McCurry, S. M., Logsdon, R. G., Buchner, D. M., Barlow, W. E., . . . Larson, E. B. (2003). Exercise plus behavioral management in patients with Alzheimer disease: A randomized controlled trial. *Journal of the American Medical Association, 290*(15), 2015-2022.

Teri, L., McCurry, S. M., Buchner, D. M., Logsdon, R. G., Lacroix, A. Z., Kukull, W. A., . . . Larson, E. B. (1998). Exercise and activity level in Alzheimer's disease: A potential treatment focus. *Journal of Rehabilitation Research and Development, 35*(4), 411-419.

Teri, L., Truax, P., Logsdon, R., Uomoto, J., Zarit, S., & Vitaliano, P. P. (1992). Assessment of behavioral-problems in dementia - The revised memory and behavior problems checklist. *Psychology and Aging, 7*(4), 622-631.

Terman, M., Remé, C. E., Rafferty, B., Gallin, P. F., & Terman, J. S. (1990). Bright light therapy for winter depression: Potential ocular effects and theoretical implications. *Photochemistry and photobiology, 51*(6), 781-792.

Terman, M., & Terman, J. S. (2005). Light therapy for seasonal and nonseasonal depression: Efficacy, protocol, safety, and side effects. *Cambridge Neurological Society: Spectrums, 10*(8), 647-663.

The National Archives. (2005). *Mental capacity act*. Buckingham, England: The Stationary Office Limited.

Thomas, G. (2011). A typology for the case study in social science following a review of definition, discourse, and structure. *Qualitative Inquiry, 17*(6), 511-521.

Thommessen, B., Aarsland, D., Braekhus, A., Oksengaard, A. R., Engedal, K., & Laake, K. (2002). The psychosocial burden on spouses of the elderly with stroke, dementia and Parkinson's disease. *International Journal of Geriatric Psychiatry, 17*(1), 78-84.

Thompson, R. L., Lewis, S. L., Murphy, M. R., Hale, J. M., Blackwell, P. H., Acton, G. J., . . . Bonner, P. N. (2004). Are there sex differences in emotional and biological responses in spousal caregivers of patients with Alzheimer's disease? *Biological Research for Nursing, 5*(4), 319-330.

Thorgrimsen, L., Selwood, A., Spector, A., Royan, L., De Madariaga Lopez, M., Woods, R. T., & Orrell, M. (2003). Whose quality of life is it anyway? The validity and reliability of the Quality of Life-Alzheimer's Disease (QoL-AD) scale. *Alzheimer Disease and Associated Disorders, 17*(4), 201-208.

Tobias, M., Yeh, L. C., & Johnson, E. (2008). Burden of Alzheimer's disease: Population-based estimates and projections for New Zealand, 2006-2031. *Australian and New Zealand Journal of Psychiatry, 42*(9), 828-836.

Touitou, Y., & Haus, E. (2000). Alterations with aging of the endocrine and neuroendocrine circadian system in humans. *Chronobiology International, 17*(3), 369-390.

Tractenberg, R. E., Singer, C. M., Cummings, J. L., & Thal, L. J. (2003). The Sleep Disorders Inventory: An instrument for studies of sleep disturbance in persons with Alzheimer's disease. *Journal of Sleep Research, 12*(4), 331-337.

Tractenberg, R. E., Singer, C. M., & Kaye, J. A. (2005). Symptoms of sleep disturbance in persons with Alzheimer's disease and normal elderly. *Journal of Sleep Research, 14*(2), 177-185.

Tractenberg, R. E., Singer, C. M., & Kaye, J. A. (2006). Characterizing sleep problems in persons with Alzheimer's disease and normal elderly. *Journal of Sleep Research, 15*(1), 97-103.

Trost, S. G., McIver, K. L., & Pate, R. R. (2005). Conducting accelerometer-based activity assessments in field-based research. *Medicine and Science in Sports and Exercise, 37*(11), S531-S543.

Tsai, Y. F., Wong, T. K. S., & Ku, Y. C. (2008). Self-care management of sleep disturbances and risk factors for poor sleep among older residents of Taiwanese nursing homes. *Journal of Clinical Nursing, 17*(9), 1219-1226.

Turjanski, N., Lees, A. J., & Brooks, D. J. (1999). Striatal dopaminergic function in restless legs syndrome: 18F-dopa and 11C-raclopride PET studies. *Neurology, 52*(5), 932-937.

Uchida, K., Okamoto, N., Ohara, K., & Morita, Y. (1996). Daily rhythm of serum melatonin in patients with dementia of the degenerate type. *Brain Research, 717*(1-2), 154-159.

Uplift Technologies. (2007). Light therapy. *Products* Retrieved January 30, 2012, from http://www.up-lift.com/Products/Products/bright-light-therapy.php

Uplift Technologies Inc. (2009). Day-Light user guide. Retrieved from http://www.day-lights.com/

Valenti, D. (2004). The anterior visual system and circadian function with reference to Alzheimer's disease. In A. Cronin-Golomb & P. R. Hof (Eds.), *Vision in Alzheimer's disease* (pp. 1-29). Basel, Switzerland: Karger.

Van't Leven, N., Prick, A. E. J. C., Groenewoud, J. G., Roelofs, P. D. D. M., De Lange, J., & Pot, A. M. (2013). Dyadic interventions for community-dwelling people with dementia and their family caregivers: A systematic review. *International Psychogeriatrics, 25*(10), 1581-1603.

van der Ploeg, E. S., & O'Connor, D. W. (2014). Methodological challenges in studies of bright light therapy to treat sleep disorders in nursing home residents with dementia. *Psychiatry and Clinical Neurosciences*. Retrieved from http://www.scopus.com/inward/record.url?eid=2-s2.0-84902234384&partnerID=40&md5=3bea5dc55e101bfdb5b34349c87e8ed3 doi:10.1111/pcn.12192

Van Nispen, R. M. A., Hoeijmakers, J. G. J., De Boer, M. R., Ringens, P. J., & Van Rens, G. H. M. B. (2008). Agreement between self-reported co-morbidity of visually impaired older patients and reports from their general practitioners. *Visual Impairment Research, 10*(2-3), 49-56.

Van Someren, E. J. W. (2000). More than a marker: Interaction between the circadian regulation of temperature and sleep, age-related changes, and treatment possibilities. *Chronobiology International, 17*(3), 313-354.

Van Someren, E. J. W. (2007). Improving actigraphic sleep estimates in insomnia and dementia: How many nights? *Journal of Sleep Research, 16*(3), 269-275.

Van Someren, E. J. W., Hagebeuk, E. E. O., Lijzenga, C., Scheltens, P., De Rooij, S. E. J. A., Jonker, C., . . . Swaab, D. F. (1996). Circadian rest-activity rhythm disturbances in Alzheimer's disease. *Biological Psychiatry, 40*(4), 259-270.

Van Someren, E. J. W., Kessler, A., Mirmiran, M., & Swaab, D. F. (1997). Indirect bright light improves circadian rest-activity rhythm disturbances in demented patients. *Biological Psychiatry, 41*(9), 955-963.

Van Someren, E. J. W., Lijzenga, C., Mirmiran, M., & Swaab, D. F. (1997). Long-term fitness training improves the circadian rest-activity rhythm in healthy elderly males. *Journal of Biological Rhythms, 12*(2), 146-156.

Van Someren, E. J. W., & Riemersma-Van Der Lek, R. F. (2007). Live to the rhythm, slave to the rhythm. *Sleep Medicine Reviews, 11*(6), 465-484.

Van Someren, E. J. W., Riemersma, R. F., Raymann, R. J. E. M., & Swaab, D. F. (2005). The effect of illumination and temperature on sleep-wake rhythm disturbances in the elderly. *Elsevier Ergonomics Book Series, 3*(C), 31-34.

Van Someren, E. J. W., Swaab, D. F., Colenda, C. C., Cohen, W., McCall, W. V., & Rosemnquist, P. B. (1999). Bright light therapy: Improved sensitivity to its effects on rest-activity thythms by application of nonparametric methods. *Chronobiology International, 16*(4), 505-518.

Venn, S., & Arber, S. (2009). Poor sleep among community dwelling older people. *Workpackage 2, briefing paper 2, SomnIA sleep in ageing* Retrieved December 10, 2011, from http://www.somnia.surrey.ac.uk/bp.html

Venn, S., & Arber, S. (2011). Day-time sleep and active ageing in later life. *Ageing and Society, 31*(2), 197-216.

Venn, S., & Arber, S. (2012). Understanding older peoples' decisions about the use of sleeping medication: Issues of control and autonomy. *Sociology of Health and Illness, 34*(8), 1215-1229.

Venn, S., Meadows, R., & Arber, S. (2013). Gender differences in approaches to self-management of poor sleep in later life. *Social Science and Medicine, 79*(1), 117-123.

Vetter, P. H., Krauss, S., Steiner, O., Kropp, P., Moller, W. D., Moises, H. W., & Köller , O. (1999). Vascular dementia versus dementia of Alzheimer's type: Do they have differential effects on caregivers' burden? *Journals of Gerontology - Series B Psychological Sciences and Social Sciences, 54*(2), 93-98.

Vitaliano, P. P., Russo, J., Young, H. M., Becker, J., & Maiuro, R. D. (1991). The Screen for Caregiver Burden. *Gerontologist, 31*(1), 76-83.

Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin, 129*(6), 946-972.

Vitiello, M. V., & Borson, S. (2001). Sleep disturbances in patients with Alzheimer's disease: Epidemiology, pathophysiology and treatment. *Cambridge Neurological Society: Drugs, 15*(10), 777-796.

Vitiello, M. V., Larsen, L. H., & Moe, K. E. (2004). Age-related sleep change: Gender and estrogen effects on the subjective-objective sleep quality relationships of healthy, noncomplaining older men and women. *Journal of Psychosomatic Research, 56*(5), 503-510.

Volicer, L., Harper, D. G., Manning, B. C., Goldstein, R., & Satlin, A. (2001). Sundowning and circadian rhythms in Alzheimer's disease. *American Journal of Psychiatry, 158*(5), 704-711.

Von Känel, R., Ancoli-Israel, S., Dimsdale, J. E., Mills, P. J., Mausbach, B. T., Ziegler, M. G., . . . Grant, I. (2010). Sleep and biomarkers of atherosclerosis in elderly Alzheimer caregivers and controls. *Gerontology, 56*(1), 41-50.

Von Känel, R., Dimsdale, J. E., Ancoli-Israel, S., Mills, P. J., Patterson, T. L., McKibbin, C. L., . . . Grant, I. (2006). Poor sleep is associated with higher plasma proinflammatory cytokine interleukin-6 and procoagulant marker fibrin D-dimer in older caregivers of people with Alzheimer's disease. *Journal of the American Geriatrics Society, 54*(3), 431-437.

Von Känel, R., Mausbach, B. T., Ancoli-Israel, S., Dimsdale, J. E., Mills, P. J., Patterson, T. L., . . . Grant, I. (2012). Sleep in spousal Alzheimer caregivers: A longitudinal study with a focus on the effects of major patient transitions on sleep. *Sleep, 35*(2), 247-255.

Von Känel, R., Mausbach, B. T., Ancoli-Israel, S., Mills, P. J., Dimsdale, J. E., Patterson, T. L., & Grant, I. (2014). Positive affect and sleep in spousal Alzheimer caregivers: A longitudinal study. *Behavioral Sleep Medicine*

Walker, M. P., Ayre, G. A., Ashton, C. H., Marsh, V. R., Wesnes, K., Perry, E. K., . . . Ballard, C. G. (1999). A psychophysiological investigation of fluctuating consciousness in neurodegenerative dementias. *Human Psychopharmacology, 14*(7), 483-489.

Ware, J. E., Kosinski, M., & Dewey, J. E. (Eds.). (2000). *How to score version two of the SF 36 Health Survey*. Lincoln, RI: Quality Metric.

Washburn, R. A., Smith, K. W., Jette, A. M., & Janney, C. A. (1993). The Physical Activity Scale for the Elderly (PASE): Development and evaluation. *Journal of Clinical Epidemiology, 46*(2), 153-162.

Weaver, T. E., & George, C. F. P. (2011). Cognition and performance in patients with obstructive sleep apnea. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5 ed., pp. 1194-1205). St. Louis, MO: Saunders, Elsiver Inc.

Webb, I. C., Antle, M. C., & Mistlberger, R. E. (2014). Regulation of circadian rhythms in mammals by behavioral arousal. *Behavioral Neuroscience, 128*(3), 304-325.

Weinert, D. (2000). Age-dependent changes of the circadian system. *Chronobiology International, 17*(3), 261-283.

Welsh, D. K., & Ptácek. (2010). Circadian rythm dysregulation in the elderly: Advanced sleep phase syndrome. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 131-142). Cambridge, England: Cambridge University Press.

Werth, E., Savaskan, E., Knoblauch, V., Gasio, P. F., Van Someren, E. J. W., Hock, C., & Wirz-Justice, A. (2002). Decline in long-term circadian rest-activity cycle organization in a patient with dementia. *Journal of Geriatric Psychiatry and Neurology, 15*(1), 55-59.

Whitebird, R. R., Kreitzer, M. J., Lewis, B. A., Hanson, L. R., Crain, A. L., Enstad, C. J., & Mehta, A. (2011). Recruiting and retaining family caregivers to a randomized controlled trial on mindfulness-based stress reduction. *Contemporary Clinical Trials, 32*(5), 654-661.

Wibom, R. (1993). Light-definitions and measurements. In L. Wetterberg (Ed.), *Light and biological rhythms in man* (pp. 23-28). Oxford, England: Pergamon Press.

Wickland, C., & Turek, F. W. (1994). Lesions of the thalamic intergeniculate leaflet block activity-induced phase shifts in the circadian activity rhythm of the golden hamster. *Brain Research, 660*(2), 293-300.

Wickstrom, G., & Bendix, T. (2000). The "Hawthorne effect" - what did the original Hawthorne studies actually show? *Scandinavian Journal of Work Environment and Health, 26*(4), 363-367.

Wider, B., & Pittler, M. H. (2010). Complementary and alternative medicine for sleep disturbances in the elderly. In S. R. Pandi-Perumal, J. M. Monti & A. A. Monjan (Eds.), *Principles and practice of geriatric sleep medicine* (pp. 403-412). Cambridge, England: Cambridge University Press.

Wilcox, S., & King, A. C. (1999). Sleep complaints in older women who are family caregivers. *Journals of Gerontology - Series B Psychological Sciences and Social Sciences, 54*(3), 189-198.

Wilkinson, H. (2002). Including people with dementia in research. In H. Wilkinson (Ed.), *The perspectives of people with dementia: Research methods and motivations* (pp. 9-24). London, England: Jessica Kingsley Publishers.

Williams, D., Vitiello, M. V., Ries, R. K., Bokan, J., & Prinz, P. N. (1988). Successful recruitment of elderly community-dwelling subjects for Alzheimer's Disease research. *Journals of Gerontology, 43*(3), 69-74.

Williams, S. (2005). *Sleep and society: Sociological ventures into the un(known)*. London, England: Routledge.

Williamson, A., & Friswell, R. (2011). Investigating the relative effects of sleep deprivation and time of day on fatigue and performance. *Accident Analysis and Prevention, 43*(3), 690-697.

Wirz-Justice, A. (1998). Beginning to see the light. *Archives of General Psychiatry, 55*(10), 861-862.

Wirz-Justice, A., Benedetti, F., & Terman, M. (2008). *Chronotherapies for affective disorders: A clincian's manual for light and wake therapy*. Basel, Switzerland: Karger.

Witting, W., Kwa, I. H., Eikelenboom, P., Mirmiran, M., & Swaab, D. F. (1990). Alterations in the circadian rest-activity rhythm in aging and Alzheimer's disease. *Biological Psychiatry, 27*(6), 563-572.

Wolkove, N., Elkholy, O., Baltzan, M., & Palayew, M. (2007). Sleep and aging: 1. Sleep disorders commonly found in older people. *Canadian Medical Association Journal, 176*(9), 1299-1304.

Woodruff, B. J. (2008). Dementia: Diagnostic evaluation and treatment. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology of the older adult* (Vol. 2, pp. 303-317). Philadelphia,PA: Wolters Kluwer: Lippincott Williams & Wilkins.

Wright, K. P., & Frey, D. (2008). Age related changes in sleep and circadian physiology: From brain mechanisms to sleep behaviour. In A. Y. Avidan & C. Alessi (Eds.), *Geriatric sleep medicine* (pp. 1-18). New York, NY: Informa Healthcare USA.

Wu, Y. H., Feenstra, M. G. P., Zhou, J. N., Liu, R. Y., Toranõ, J. S., Van Kan, H. J. M., . . . Swaab, D. F. (2003). Molecular Changes Underlying Reduced Pineal Melatonin Levels in Alzheimer Disease: Alterations in Preclinical and Clinical Stages. *Journal of Clinical Endocrinology and Metabolism, 88*(12), 5898-5906.

Wu, Y. H., Fischer, D. F., Kalsbeek, A., Garidou-Boof, M. L., van der Vliet, J., van Heijningen, C., . . . Swaab, D. F. (2006). Pineal clock gene oscillation is disturbed in Alzheimer's disease, due to functional disconnection from the "master clock". *Jounral of the Federation of American Societies for Experimental Biology, 20*(11), 1874-1876.

Wu, Y. H., & Swaab, D. F. (2005). The human pineal gland and melatonin in aging and Alzheimer's disease. *Journal of Pineal Research, 38*(3), 145-152.

Wu, Y. H., & Swaab, D. F. (2007). Disturbance and strategies for reactivation of the circadian rhythm system in aging and Alzheimer's disease. *Sleep Medicine, 8*(6), 623-636.

Wu, Y. H., Zhou, J. N., Van Heerikhuize, J., Jockers, R., & Swaab, D. F. (2007). Decreased MT1 melatonin receptor expression in the suprachiasmatic nucleus in aging and Alzheimer's disease. *Neurobiology of Aging, 28*(8), 1239-1247.

Wulff, K., Gatti, S., Wettstein, J. G., & Foster, R. G. (2010). Sleep and circadian rhythm disruption in psychiatric and neurodegenerative disease. *Nature Reviews Neuroscience: Perspectives, 11*(8), 589-599.

Yamadera, H., Ito, T., Suzuki, H., Asayama, K., Ito, R., & Endo, S. (2000). Effects of bright light on cognitive and sleep-wake (circadian) rhythm disturbances in Alzheimer-type dementia. *Psychiatry and Clinical Neurosciences, 54*(3), 352-353.

Yesavage, J. A. (1988). Geriatric Depression Scale. *Psychopharmacology Bulletin, 24*(4), 709-710.

Yesavage, J. A., Friedman, L., Ancoli-Israel, S., Bliwise, D., Singer, C., Vitiello, M. V., . . . Lebowitz, B. (2003). Development of diagnostic criteria for defining sleep disturbance in Alzheimer's disease. *Journal of Geriatric Psychiatry and Neurology, 16*(3), 131-139.

Yesavage, J. A., Taylor, J. L., Kraemer, H., Noda, A., Friedman, L., & Tinklenberg, J. R. (2002). Sleep/wake cycle disturbance in Alzheimer's disease: How much is due to an inherent trait? *International Psychogeriatrics, 14*(1), 73-81.

Yoon, I. Y., Kripke, D. F., Youngstedt, S. D., & Elliott, J. A. (2003). Actigraphy suggests age-related differences in napping and nocturnal sleep. *Journal of Sleep Research, 12*(2), 87-93.

Yoshitake, T., Kiyohara, Y., Kato, I., Ohmura, T., Iwamoto, H., Nakayama, K., . . . Fujishima, M. (1995). Incidence and risk factors of vascular dementia and Alzheimer's disease in a defined elderly Japanese population: The Hisayama study. *Neurology, 45*(6), 1161-1168.

Young, T., Shahar, E., Nieto, F. J., Redline, S., Newman, A. B., Gottlieb, D. J., . . . Samet, J. M. (2002). Predictors of sleep-disordered breathing in community-dwelling adults: The Sleep Heart Health Study. *Archives of Internal Medicine, 162*(8), 893-900.

Youngstedt, S. D. (2005). Effects of exercise on sleep. *Clinics in Sports Medicine, 24*(2), 355-365.

Youngstedt, S. D., & Freelove-Charton, J. D. (2005). Exercise and sleep. In G. E. J. Faulkner & A. H. Taylor (Eds.), *Exercise, health and mental health: Emerging relationships* (pp. 159-189). Oxon, England: Routledge.

Youngstedt, S. D., Kripke, D. F., Klauber, M. R., Sepulveda, R. S., & Mason, W. J. (1998). Periodic leg movements during sleep and sleep disturbances in elders. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 53*(5), 391-394.

Youngstedt, S. D., O'Connor, P. J., & Dishman, R. K. (1997). The effects of acute exercise on sleep: A quantitative synthesis. *Sleep, 20*(3), 203-214.

Zarit, S. H., Reever, K. E., & Bach-Peterson, J. (1980). Relatives of the impaired elderly: Correlates of feelings of burden. *Gerontologist, 20*(6), 649-655.

Zeitzer, J. M., Dijk, D. J., Kronauer, R. E., Brown, E. N., & Czeisler, C. A. (2000). Sensitivity of the human circadian pacemaker to nocturnal light: Melatonin phase resetting and suppression. *Journal of Physiology, 526*(3), 695-702.

Zhou, J., Hofman, M. A., & Swaab, D. F. (1995). VIP neurons in the human SCN in relation to sex, age, and Alzheimer's disease. *Neurobiology of Aging, 16*(4), 571-576.

Zhou, X., Ferguson, S. A., Matthews, R. W., Sargent, C., Darwent, D., Kennaway, D. J., & Roach, G. D. (2011). Sleep, wake and phase dependent changes in neurobehavioral function under forced desynchrony. *Sleep, 34*(7), 931-941.

Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica, 67*(6), 361-370.

8 APPENDICIES

1 Statement of Contributions to Doctoral Thesis Containing Publications

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Rosemary Gibson

Name/Title of Principal Supervisor: Professor Philippa Gander

Name of Published Research Output and full reference:

Sleep of Maori and non-Maori of Advanced Age
Gibson, R., Gander, P., Paine, S-J., Kepa,M., Dyall, L., Moyes, S., & Kerse, N
submitted for consideration for publication for the Australasian Journal of Ageing

In which Chapter is the Published Work: 1

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:

  and / or

- Describe the contribution that the candidate has made to the Published Work:

  planned analyses, co-wrote proposal for collaboration, analysed the sleep-related data, written the paper

r.gibson@massey.ac.nz — Digitally signed by r.gibson@massey.ac.nz DN: cn=r.gibson@massey.ac.nz Date: 2014.08.25 13:30:32 +12'00'
Candidate's Signature

23.8.2014
Date

Philippa Gander — Digitally signed by Philippa Gander DN: cn=Philippa Gander, o=Massey University, ou=Sleep/Wake Research Centre, email=p.h.gander@massey.ac.nz, c=NZ Date: 2014.08.25 16:19:22 +12'00'
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25 August 2014
Date

GRS Version 3– 16 September 2011

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION TO DOCTORAL THESIS CONTAINING PUBLICATIONS

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We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Rosemary Gibson

Name/Title of Principal Supervisor: Professor Philippa Gander

Name of Published Research Output and full reference:

The Effect of Caregiving Status on the Sleep of Older New Zelanders
Gibson, R., Gander, P., Alpass, F., & Stephens, C
Accepted for publication for the Australasian Journal of Ageing (in press)

In which Chapter is the Published Work: 1

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:

  and / or

- Describe the contribution that the candidate has made to the Published Work:

  planned analyses, analysed the sleep-related data, written the paper

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25 August 2014
Date

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MASSEY UNIVERSITY
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STATEMENT OF CONTRIBUTION TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Rosemary Gibson

Name/Title of Principal Supervisor: Professor Philippa Gander

Name of Published Research Output and full reference:

Understanding the Sleep Problems of People with Dementia and their Family Caregivers
The final version of this paper has been has been published in Dementia: The International Journal of Social Research and Practice (volume 13, issue 3, pages 348 – 363, May 2014) by SAGE Publications Ltd., (http://dem.sagepub.com/content/13/3/350.abstract). All rights reserved © (Gibson, R. H., Gander, P. H., & Jones, L. M.).

In which Chapter is the Published Work: 2

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:

  and / or

- Describe the contribution that the candidate has made to the Published Work:

  designed the study, applied for ethics, conducted the focus groups, analysed the data, wrote up paper

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25 August 2014
Date

GRS Version 3– 16 September 2011

2 Study 3: Supporting Documents

Health and Disability Ethics Committees

Central Regional Ethics Committee
Ministry of Health
Level 2, 1–3 The Terrace
PO Box 5013
Wellington
Phone: (04) 496 2405
Fax: (04) 496 2191
Email: central_ethicscommittee@moh.govt.nz

9 August 2010

Ms Rosemary Gibson
Sleep/Wake Research Centre
Massey University
Wellington
Private Bag 756

Dear Rosemary

Re: Ethics ref: **CEN/10/EXP/33** (please quote in all correspondence)
Study title: Sleep of Older People with Dementia and Those who Live with Them
Investigators: Ms Rosemary Gibson

This study was given ethical approval by the Central Regional Ethics Committee on the 9th of August 2010. A list of members of the Committee is attached.

Approved Documents

- Protocol Version 1 dated the 22nd of June 2010
- Information sheet version 2 dated the 9th of August 2010
- Consent form version 2 dated the 9th of August 2010
- Letter of Invite version 2 dated the 9th of August 2010
- Schedule for Participants versions 1 dated the 22nd of June 2010

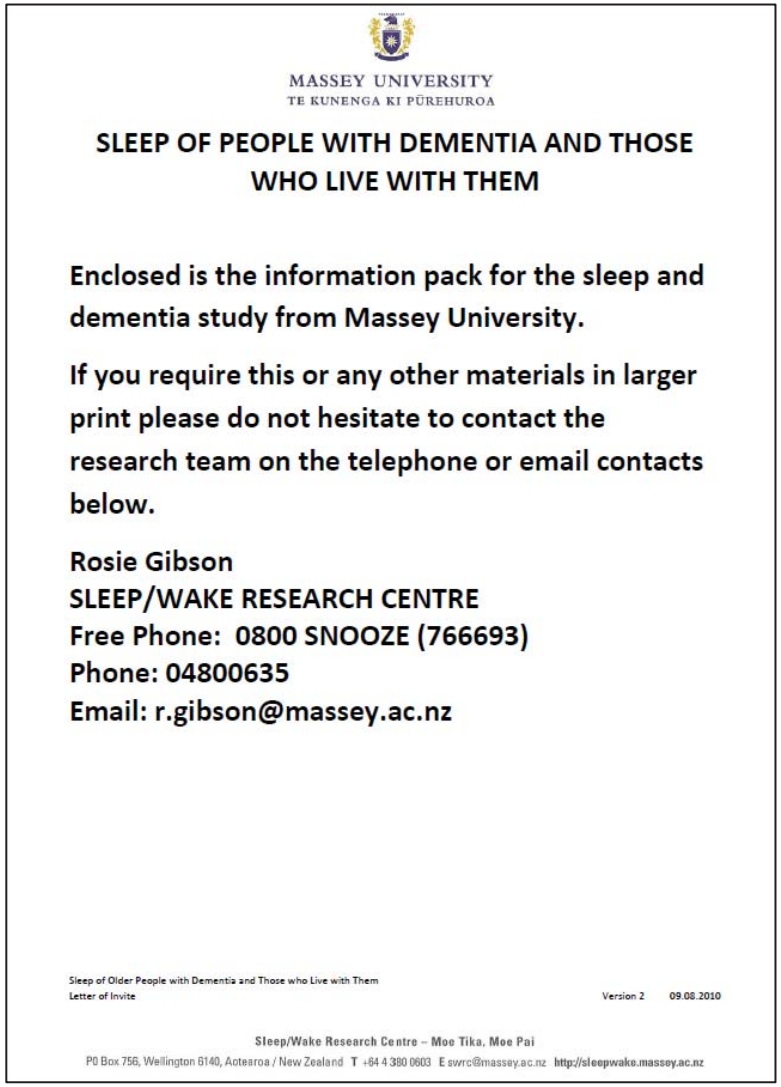
This approval is valid until the 30th of November 2010. A Final Report is required at the conclusion of the study. The Final Report Form is available at www.thicscommittees.health.govt.nz

Amendments and Protocol Deviations

All significant amendments to this proposal must receive prior approval from the Committee. Significant amendments include (but are not limited to) changes to:

- the researcher responsible for the conduct of the study at a study site
- the addition of an extra study site
- the design or duration of the study
- the method of recruitment
- information sheets and informed consent procedures.

Significant deviations from the approved protocol must be reported to the Committee as soon as possible.



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SLEEP OF PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM

Enclosed is the information pack for the sleep and dementia study from Massey University.

If you require this or any other materials in larger print please do not hesitate to contact the research team on the telephone or email contacts below.

Rosie Gibson
SLEEP/WAKE RESEARCH CENTRE
Free Phone: 0800 SNOOZE (766693)
Phone: 04800635
Email: r.gibson@massey.ac.nz

Sleep of Older People with Dementia and Those who Live with Them
Letter of Invite Version 2 09.08.2010

Sleep/Wake Research Centre – Moe Tika, Moe Pai
PO Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

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SLEEP/WAKE RESEARCH CENTRE
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http://sleepwake.massey.ac.nz

November 2010

SLEEP OF OLDER PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM
FOCUS GROUPS

Dear ....
Thank you for your interest in participating in a focus group to evaluate the sleep of people with dementia and those living with them.
Details are
Date: 22nd November
Venue: Alzheimers Wellington, 55 Hutt Road, Petone
Time: 10:15 am for welcome and preparation for a 10:30 start

An information sheet and a consent form are enclosed. Please bring the signed consent form with you on the day.
It is anticipated that the group will run for about 1 ½ hours. We then invite you to join us for lunch from approximately 12:00 pm. We will provide you with a petrol or taxi voucher as compensation for travel costs.

The session will be run by myself - Rosie Gibson, a member of Alzheimers Wellington will also be present. It will be semi-structured but informal and more details are given on the attached information sheet. If you are unable to make it on the day please contact me on 0800 SNOOZE, or by email (r.gibson@massey.ac.nz).
Thank you for your interest in this study. I look forward to meeting you.

Yours Sincerely,

Rosie Gibson
Research Officer

Sleep of Older People with Dementia and Those who Live with Them
Letter of Invite Version 2 09.08.2010

Sleep/Wake Research Centre – Moe Tika, Moe Pai
PO Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

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November 2010

INFORMATION SHEET

SLEEP OF OLDER PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM FOCUS GROUPS

You have been invited to take part in a focus group to discuss your sleep. Your participation is entirely your choice. You do not have to take part. Please inform the researcher if you wish to join a focus group and bring the signed consent form with you.

Inclusion and exclusion criteria

We are looking for people with mild stages of dementia who are:

- Of or over 65 years of age
- Live with someone who supports them
- Have disrupted sleep 3 or more times a week

We also invite supporters to the focus groups who are:

- Of or over 65 years of age
- Living with the participant with dementia
- Identifies sleep as a problem for the person they support

About the Study

The aim of running the focus groups is to gain insight of issues or concerns surrounding the sleep of those with dementia and those supporting them within the home. We would also like to understand the feasibility of some non pharmacological interventions to improve sleep. The findings will help to design of a larger intervention study in 2011.

Please Turn

We are looking for approximately 15 dementia/supporter pairs. Each focus group will include up to 4 pairs. Focus groups will be conducted by a facilitator from the research team, a member of Alzheimers Wellington will also be present. The session will take the form of a semi-structured discussion. Focus groups will be audio-taped and reviewed for information to help understand the issues surrounding sleep and to design future studies.

What is involved if you decide to participate?

If you decided to be in a focus group:

- You will be asked to attend one session at a community centre in the location most convenient to you (Petone or Kapiti coast).
- The session will last approximately 1½ hours
- Discussion at the group will be audio-taped. You will be provided with a written copy of the key findings from the discussion. You will have the opportunity to amend any of your contributions, have them withdrawn, or consent for their use for review.
- You will receive a petrol voucher to cover the coast of your travel (or taxi vouchers for those who do not drive)
- Lunch will be provided afterwards

Ethics

This study has received ethical approval from The Health and Disability Ethics Committee (Ref CEN/10/EXP/33)

Important points

- Information from the session will not be fully transcribed but used as a guide for future research design. Information will only be used once you have seen a copy and consented to its use.
- Direct quotes may be used, but no material that could personally identify you will be used in any reports on this study.
- Electronic data will be stored on password protected computer systems and DVD's that will be kept in a locked filing cabinet at the Sleep/Wake Research Centre, which is protected by an electronic alarm system. Paper based data will be kept in a locked filing cabinet at the Sleep/Wake Research Centre, which is protected by an electronic alarm system.

Please Turn

- There are no personal risks involved with taking part in the focus group
- Results from this study together will be sent directly to participants and be made available from Alzheimers Wellington.
- If you have any queries or concerns regarding your rights as a participant in this study, you may wish to contact an independent health and disability advocate:
  Free phone: 0800 555 050
  Free fax: 0800 2 SUPPORT (0800 2787 7678)
  Email: advocacy@hdc.org.nz

Participation
You are under no obligation to accept this invitation. If you decide to participate you have the right to:

- Decline to answer any particular question;
- Make no comments on the discussion items raised;
- Ask for the audio tape to be turned off at any time during the process;
- Withdraw from the focus group-either during the session or upon receipt of the information and quotes produced;
- Ask any questions about the study or focus group at any time during participation;
- Provide information on the understanding that your name will not be used unless you give permission to the researcher;
- Be given access to a summary of the project findings when it is concluded.

What do I do now?

If you both choose to participate in a focus group after reading this information sheet, please complete the attached consent form. Please bring this with you to the focus group. Only the names and contact details of those who agree to participate will be retained.

If you do choose to participate please take the time to look over the focus group schedule included in this pack. This will allow you to talk about the points together and prepare your thoughts for the discussion.

Please Turn

3

- There are no personal risks involved with taking part in the focus group
- Results from this study together will be sent directly to participants and be made available from Alzheimers Wellington.
- If you have any queries or concerns regarding your rights as a participant in this study, you may wish to contact an independent health and disability advocate:
  Free phone: 0800 555 050
  Free fax: 0800 2 SUPPORT (0800 2787 7678)
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Participation
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If you do choose to participate please take the time to look over the focus group schedule included in this pack. This will allow you to talk about the points together and prepare your thoughts for the discussion.

Please Turn

Sleep of Older People with Dementia and Those who Live with Them
Information Sheet Version 2 09.08.2010

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

CONSENT FORM
Sleep of Older People with Dementia and Those who Live with Them

- I have read and I understand the information sheet date 9/8/2010 for volunteers taking part in a focus group concerning the sleep people with dementia and those who live with them. I have had the opportunity to ask any questions about this focus group and I am satisfied with the answers I have been given.
- I have had the opportunity to use whānau support or a friend to help me ask questions and understand the study
- I understand that participation in this focus group is voluntary (my choice), and that I may withdraw from the group at any time and this will in no way affect my continued support.
- I understand that participation in this focus group is confidential. Direct quotes may be used but no material which could identify me or my family members will be used in any reports on this study.
- I consent to my being audiotaped during this study
- I have had time to consider whether to take part in the study.
- I know whom to contact if I have any questions about the study.
- A petrol voucher will be given to me at the end of the focus group to cover travel expenses (taxi vouchers can be provided for non-drivers). No other compensation will be made.
- This consent form will be held for a period of five (5) years.

Sleep/Wake Research Centre – Moe Tika, Moe Pai
P0 Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

Those who have dementia:

I *(print full name)* _____
Hereby agree to the above statements and consent to take part in this study

Date_____ Signature _____

Supporters:

I *(print full name)* _____
Hereby agree to the above statements and consent to take part in this study

Date_____ Signature _____

Researchers
Rosemary Gibson, MSc
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Project explained by: Rosie Gibson
Project role: Principle investigator
Signature:
Date:

Sleep of Older People with Dementia and Those who Live with Them
Consent Form
Version 2 09.08.2010

Sleep/Wake Research Centre – Moe Tika, Moe Pai
P0 Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

Sleep of older people with dementia and those who live with them
Focus groups schedule for participants

Below is an outline of the areas to be discussed at the focus group. There is also an outline of three sleep aids that may be useful for people with dementia. These aids will also be discussed in terms of how practical and useful you feel they may be.

Please feel free to discuss these questions together and use these pages for notes prior to or during the focus group. This document will not be collected by the research team.

When thinking about each area consider what causes certain issues with your sleep and what the consequences are.

For each point some examples are listed to help promote discussion between you and your partner as well as with the rest of the focus group. You may have other answers to these questions which we invite you to note down and discuss on the day.

Areas to discuss

We will discuss the following questions firstly for the person with dementia and then again for the person in the supporting role.

1. Do you find it hard to get to sleep?

Why can't you get to sleep?

Examples of things that might make it hard to get to sleep: noise, light, too hot or cold, thinking or worried about something, too busy, feeling unwell…

What happens when you can't sleep?

Examples of consequences: worry about not getting to sleep, feel lonely, feel sleepy or hard to concentrate the next day, feel frustrated…

2. Do you sleep during the day?

Why do you sleep in the daytime?

Examples of reasons: feel very tired, bored, cold, fall asleep unintentionally…

What happens when you sleep during the day?

Examples of consequences: can't get to sleep at night, feel groggy, lose track of time, miss social events….

3. Is your sleep disrupted in the night?

What disrupts it?

Examples of things that might disrupt sleep: noise, light, too hot or cold, need to use the bathroom, someone else disturbs your sleep, hungry, feeling unwell…

What happens when your sleep is disturbed?

Examples of consequences: can't get back to sleep, nap the next day, feel very tired or find it hard to concentrate the next day, feel worried or stressed….

The research team will Introduce sleep aids for people with dementia (see attached pages)

Areas to discuss **Notes**

4. Sleep Aids for those with Dementia

a) Use of light therapy

How useful do you think this would be?

Would you use this within your home?

b) Use of physical exercise regimes

How useful do you think this would be?

Would you incorporate this into your normal routine?

c) Use of sleep hygiene education

How useful do you think this would be?

Would you incorporate this into your normal routine?

5. Finally

Is there anything related to this topic that has not been discussed and you would like to bring up now?

Overview of sleep aids for people with dementia which will be presented and discussed

1. Light Therapy

The human body uses light cues to regulate sleep, mood and energy. These daily cycles often fall out of sync for people with dementia resulting in deregulated sleep and mood.

Light therapy helps to shift the rhythms of sleep and mood back into cycle by stimulating the production of key hormones in the brain. Light therapy naturally affects the body by mimicking the power of the sun cuing the body clock to synchronise to the time of day.

An example of a light box will be shown and discussed at the focus group.

(Day-Lights light therapy device in use, http://day-lights.com/canadaindex.html)

To use the light box you need to sit approximately 12 inches away from the device which should be positioned just above eye level. Keep your eyes open but do not look directly into the light. You can perform sedentary activities such as reading, eating or crafts whilst using the light box.

One sleep aid would be to use light therapy for 30 minutes during the morning (between 9-11am).

Frequently asked questions about using light therapy

Do I have to take off my glasses to use?

No, it will work with or without glasses, but should not be used with sunglasses.

Are there any major side effects?

Light therapy is safe when used according to the recommendations. Some report temporary headaches or nauseas which are usually resolved by sitting further away from the device. You cannot get sunburn as UV light is eliminated.

Can it damage my eyes?

The highest risk (for damage to the skin, and cornea and lens of the eyes) is from invisible, short-wavelength ultraviolet (UV) light, which is filtered out of the recommended light therapy device.

Who is Light therapy <u>not</u> suitable for?

Light therapy may not be suitable for Individuals who have had cataract surgery, or pre-existing eye or skin conditions (such as retinal dystrophies, macular degeneration, porphyria, lupus erythematodes, chronic actinic dermatitis and solar urticaria). In such cases, bright light therapy should be administered only under guidance of an ophthalmologist or dermatologist. It is also unsuitable for those with bipolar disorder. Light therapy should not be used in conjunction with photosensitizing drugs such as Psychiatric neuroleptic drugs (e.g., phenothiazine), antiarrhythmic drugs (e.g., amiodarone), antimalarial and antirheumatic drugs.

Should I use it every day?

Light therapy can be used every day. It is especially important in the winter or at times when you are unable to go outside and receive natural sun light for one or two hours.

Can I use light therapy in the evening?

If you use light therapy too close to your normal bedtime then it may be difficult to fall asleep.

For more information prior to the focus group please visit <u>http://www.chronotherapeutics.org</u> or contact the research team.

2. Physical Exercise Regimes

Exercise also helps to cue the body's internal clock and synchronises sleep and wake. Regular low-impact aerobic exercise has been shown to improve sleep quality and duration as well as make people feel more alert during the day. It does this by raising the body's core temperature as well as promoting relaxation.

As with light therapy, the best time of day for physical exercise is during the morning, as exercising too close to bedtime may make it harder to get to sleep.

Potential exercise regimes could be:

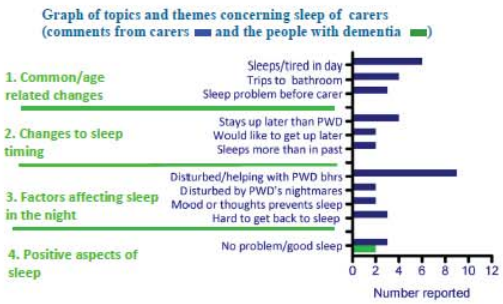
- **30 -40 minutes of walking during the morning (between 11am – 1pm) 3-4 times per week.**
- **Or 30-40 minutes of yoga style stretches and movement (between 11am-1pm) 3-4 times per week.**

3. Sleep Hygiene Education

This technique involves a collection of guidelines that, when followed can promote healthy sleep. The key theme is that activities during the day have an influence on sleep at night. Information delivered may include:

	Examples
• Management of daytime sleepiness	Reduce afternoon naps
• Sleep scheduling	A regular bedtime
• The sleeping environment	No television in the bedroom
• Diet and sleep	No caffeine after lunch
• Exercise and sleep	Brisk walks in the morning
• Light and sleep	Keeping a rooms dimly lit at night
• Relaxation techniques	A hot bath before bed

Appreciating and enforcing sleep hygiene guidelines will involve reading and following the guide but once you find which areas are useful to you they ought to become a part of your daily routine (for example, no caffeine after lunchtime and the removal of the television from the bedroom.



Carers typically went to bed (or wanted to go to bed) later. Some used this time for relaxing, but others rushed around finishing chores around the house.

Most carers reported that their sleep was most disturbed by their partner's activities in the night.

Disturbances included:

- Restlessness and being *"knocked around"*
- Frustration while waiting for their partner to get back to bed, making it difficult for them to resettle
- Providing physical or mental support to enable their partner to sleep

Unlike those with dementia, carers didn't find it so easy to get back to sleep after disturbances.

Coping with sleep troubles

Many had positive attitudes about coping with sleep, working as a team, and of sense of humour helped. Others felt it a little harder and had *"learnt to just live with it"*, and in some cases people expected that sleep might get worse.

Although many had some good strategies to help sleep, research is needed to find sleep strategies to help people living with dementia at home.

Sleep tips

Examples of things that people found helpful included:

- **Environment:** Separated beds or bedrooms to reduce disturbance, and making the bedroom relaxing .
- **Safety:** Dim lighting to reduce the risk of falls, keeping doors and windows secure, and using a bell to alarm the carer if needed
- **Relaxing:** keeping routines, hot baths or drinks before bed, allowing time to wind down at night.
- **Staying awake:** Puzzles or books to stop falling asleep, walking or gardening to get worn out for bed.

Special thanks to the people who gave their time to take part in these extremely interesting discussions, and also the staff of Alzheimers Wellington for supporting this research.

I am looking for participants to take part in research trialling a simple intervention for improving sleep of people with dementia. Please contact me on the number below for more information.

Rosie Gibson
Sleep/Wake Research Centre
0800 SNOOZE (766693)
04 3800 635
R.gibson@massey.ac.nz

Sleep of people with dementia and their family carers

Findings from community focus groups

Sleep/Wake Research Centre
& Alzheimers Wellington

MASSEY UNIVERSITY

Background

Sleep disturbances associated with dementia can be challenging. Some dementia-related sleep problems have been linked to reduced activity in areas of the brain involved in sleep timing.

Sleep disturbances are also common for family caregivers, and when both the person with dementia and caregiver are sleeping poorly, the overall care giving situation becomes more difficult.

This study aimed to:

- Understand the types of sleep problems experienced by those with dementia and their family carers
- Identify what strategies people have in place to manage sleep

What we did

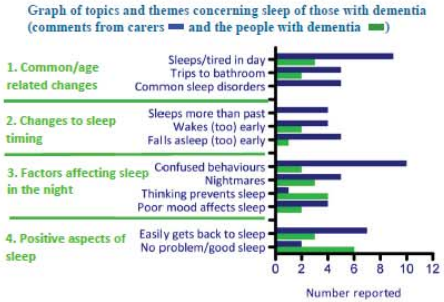
- Members of Alzheimers Wellington were invited to take part in one of three focus groups across the Wellington region
- Participants were 12 pairs including a person with dementia and their family carer (aged 65 or over) who considered sleep to be problematic
- Group discussions lasted 60-90 minutes. They were recorded, transcribed, and analysed for common themes

This leaflet provides a brief overview of what we found.

Funded by the Massey University Research Fund & Maurice and Phyllis Paykel Trust. Ethical approval was granted by the Central Regional Ethics Committee (10/EXP/33).

Sleep of the people with dementia

Some of the sleep issues were typical, age-related changes to sleep such as being more tired, getting up to go to the bathroom, and signs of common sleep disorders such as twitchy legs or snoring.



Some carers thought that earlier sleep timing and sleeping more in the day was related to chronic sleepiness, or maybe boredom.

Confused awakenings and bizarre behaviours occurring at night were a key problem. These included restlessness, hallucinations, and automatic behaviours such as wandering, looking for things, and checking doors and windows.

Some of these behaviours were thought to be triggered by the carer's movements at night or to be related to activities of the previous day or planned for the next day.

Those with dementia generally seemed to be able to get back to sleep easily after a disturbance, and had little awareness of their behaviours, which made these situations difficult for some cares to cope with.

Those with dementia spoke more often about nightmares and worrying at night preventing them from sleep. Some remembered their nightmares vividly whereas others simply woke feeling afraid.

Depression and paranoia were also factors which made going to bed alone or in the dark difficult for some. In the daytime, some carers noted that reduced motivation might be related to dozing off or poor sleep routines.

Half of the people with dementia said they had no problem or even good quality sleep, despite the disturbances listed above. This could be due to memory difficulties or a simple lack of awareness (we often don't remember what we do when we're half asleep!) This normalisation or denial of problems made it difficult for some carers to manage dementia-related sleep problems:

"She doesn't realise what I pick up at night. She has beautiful sleeps she says, which no doubt she does, but she doesn't realise during the time she is up and down..."

Sleep of family carers

Some carers reported restlessness or trouble getting to sleep prior to being a care giver. Many were sleepy in the day but not all felt they could nap because of their daily responsibilities:

"Just tired really yes, just tired all day. But you still have to do things because it is your responsibility really, you are the carer. I find sometimes it is a bit hard."

3 Study 4: Ethics Approval

Health and Disability Ethics Committees

Central Regional Ethics Committee
c/- Ministry of Health
PO Box 5013
1 the Terrace
Wellington
Phone: (04) 816 2405
Email: central_ethicscommittee@moh.govt.nz

28 April 2011

Ms Rosemary Gibson
Sleep/Wake Research Centre
Massey University
Private Bag 756
Wellington

Dear Ms Gibson

Ethics ref: **CEN/11/02/01** (please quote in all correspondence)
Study title: Sleep of Older People with Dementia and Those Who Live with Them :Pilot of an Intervention

Thank you for your letter dated the 14th of February 2011 enclosing documentation relating to the above named study. This documentation has been reviewed and approved by the Chairperson of the Central Regional Ethics Committee under delegated authority.

Approved Documents

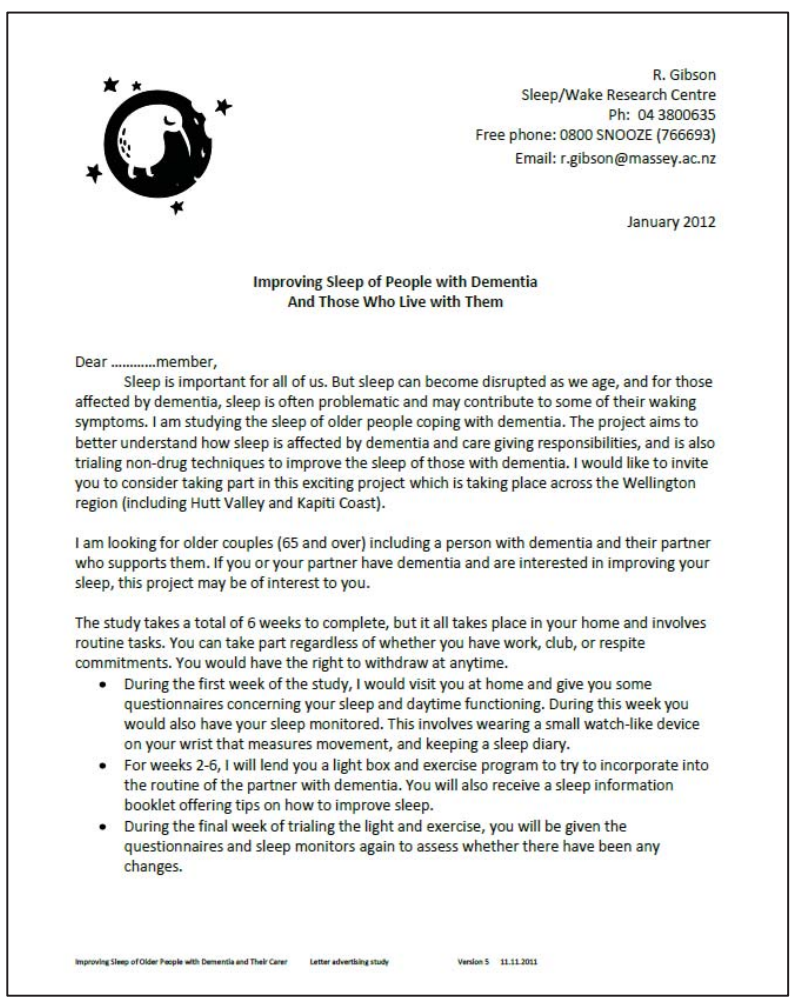
- Page 12 and 13 of the Application Form
- Information Sheet Version 2
- Consent Form Version 2
- Statement by Partner Version 2
- Questionnaire concerning participants with Dementia Version 2 (relevant section)
- Protocol Version 2
- Locality Assessment form from Alzheimers Wellington

Please do not hesitate to contact me should you have any queries.

Yours sincerely

Awhina Rangiwai
Administrator
Central Regional Ethics Committee
Email: central_ethicscommittee@MOH.govt.nz

4 Study 4: Examples of Recruitment-Related Documents[1]



R. Gibson
Sleep/Wake Research Centre
Ph: 04 3800635
Free phone: 0800 SNOOZE (766693)
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January 2012

Improving Sleep of People with Dementia
And Those Who Live with Them

Dear .....member,

Sleep is important for all of us. But sleep can become disrupted as we age, and for those affected by dementia, sleep is often problematic and may contribute to some of their waking symptoms. I am studying the sleep of older people coping with dementia. The project aims to better understand how sleep is affected by dementia and care giving responsibilities, and is also trialing non-drug techniques to improve the sleep of those with dementia. I would like to invite you to consider taking part in this exciting project which is taking place across the Wellington region (including Hutt Valley and Kapiti Coast).

I am looking for older couples (65 and over) including a person with dementia and their partner who supports them. If you or your partner have dementia and are interested in improving your sleep, this project may be of interest to you.

The study takes a total of 6 weeks to complete, but it all takes place in your home and involves routine tasks. You can take part regardless of whether you have work, club, or respite commitments. You would have the right to withdraw at anytime.

- During the first week of the study, I would visit you at home and give you some questionnaires concerning your sleep and daytime functioning. During this week you would also have your sleep monitored. This involves wearing a small watch-like device on your wrist that measures movement, and keeping a sleep diary.
- For weeks 2-6, I will lend you a light box and exercise program to try to incorporate into the routine of the partner with dementia. You will also receive a sleep information booklet offering tips on how to improve sleep.
- During the final week of trialing the light and exercise, you will be given the questionnaires and sleep monitors again to assess whether there have been any changes.

I would visit you at home to work through the questionnaires as well as to deliver the sleep monitors and equipment. I will also be available to visit if you require assistance throughout, and a free phone number is also available for you to contact me at any point during the study

If you and your partner are interested in taking part or have any questions please contact me and I will send you some more detailed information for you to consider before enrolling:

Free phone: 0800 SNOOZE (766693), phone: 04 3800 635, email: r.gibson@massey.ac.nz.

If this study does not apply to you personally, you may know someone who may be interested. If so, please feel free to pass on this information. If you are interested in the study but unable to take part, please don't hesitate to call if you would like further information about sleep or a copy of the study results when it is complete.

Yours sincerely,

Rosie Gibson
Principle Investigator

[1] Note documents are all the most recent versions

Do you or your partner have DEMENTIA & TROUBLE SLEEPING?

You are invited to take part in a sleep study. This involves simple sleep monitoring, questionnaires, and trying some activities to help improve sleep.

I am looking for people with dementia and their supporting partners who are aged 65 or over, and living together in the Greater Wellington Region.

Why take part?

- Enjoy learning more about your sleep
- Receive copies of your sleep studies
- Explore new ways to improve sleep
- Contribute to a better understanding of sleep problems related to dementia

- ✓ You can join the study anytime throughout 2012
- ✓ The study all takes place at home
- ✓ You would have the right to withdraw at anytime

Contact...

If you are interested in taking part in this study please contact me for more information:

Rosie Gibson
Sleep/Wake Research Centre,
Massey University
Private Bag 756, Wellington
Free Phone: ***0800 SNOOZE*** (766693)
Phone: 04 3800635
email: r.gibson@massey.ac.nz

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Version 3. 11/11/2011

SLEEP~WAKE *research centre*
MOETIKA~MOE PAI

Sleep and Ageing

Rosie Gibson
Sleep/Wake Research Centre, Massey University Wellington, New Zealand

0800 SNOOZE (766693)
r.gibson@massey.ac.nz

How did you sleep last night?

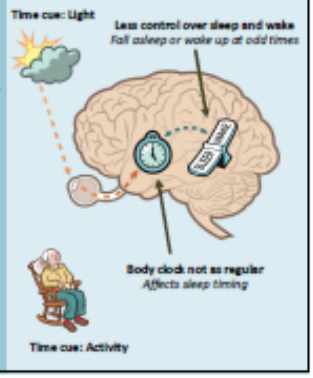
1. Very well?
2. Fairly well?
3. Fairly badly?
4. Very badly?

Sleep & Ageing

With ageing we tend to see more:

- Shallow & fragmented sleep
- Early morning awakenings
- More daytime sleepiness & napping

Sleep regulation as we get older



Some sleep disorders are more common as we grow older...

Disorder	Symptoms
Insomnia	Unable to get to sleep or stay asleep
Sleep apnoea	Snoring and Pauses in breathing during sleep
Restless legs	Urge to move and discomfort in the legs
Nocturia	Needing to urinate during the night
Parasomnias (common in childhood)	Seemingly automatic behaviours during sleep (e.g. Walking and talking)

How do you feel after a bad night's sleep?

Poor sleep can influence:

- Mood
- Attention and thinking
- Risk of accidents
- Health
- Increased appetite

Medicines and sleep

Sleeping pills:

- Help fall asleep but..
  also may be groggy in the day
- Disrupt sleep quality
- Use with caution

Many medications have sleep related side effects (check with GP)

Drinks and sleep

Alcohol:

Makes us fall asleep but...

- Blocks dreaming sleep
- Disrupts sleep later in night

Caffeine:

Keeps us awake but....

- Has effects for 3-5 hours
- Avoid before bed

Naps

Improve your functioning

Short naps:

- Less than 40 minutes
- Avoids feeling groggy

Longer naps:

- Sleep cycle is 90 mins
- Allow time for 1 or 2 cycles

Good sleep habits

What do you do?...

- Regular routine
- Keep the bedroom a "safe sleep" zone
- Avoid eating or drinking too much before bed
- Avoid alcohol and caffeine before bed
- Relaxation techniques to help fall asleep
- If you don't fall asleep in about 30 minutes, get out of bed for a little while

Source: National sleep Foundation

Sleep of those affected by dementia

Sleep and dementia:

- More fragmented sleep/wake cycle
- Variable sleep timing
- Confused awakenings & behaviours in night

Sleep of carers:

- Woken by those in their care
- Providing support in night
- Unable to get back to sleep after a disturbance

My Research

- Aims to better understand & manage dementia-related sleep problems for those living in the community
- Assess whether interventions to improve the sleep of people with dementia also influences:
  - Daytime functioning
  - Mood
  - Quality of life
  - Carers sleep

My Research

1. Measure sleep of people with dementia and their carers
2. Trial simple techniques to improve sleep

Sleep monitoring:

Activity watch & diaries

Questionnaires:

Sleep, memory, health, mood and coping

Things to try over a few weeks

1. Light
2. Activity
3. Information

This important research needs volunteers!

I am looking for:

- Couples consisting of a person with dementia and their carer
- 65 years old or over
- Living at home in Wellington region
- Are interested in improving their sleep

This important research needs volunteers!

- The study is simple and rewarding
- All takes place at home
- You can withdraw at any time
- Participating will help us to understand and manage dementia-related sleep problems

Any Questions?

My contact:
Rosie Gibson
Sleep/Wake Research Centre
0800 SNOOZE (766693)
04 3800635
r.gibson@massey.ac.nz

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Sleep and Ageing

Rosie Gibson
Sleep/Wake Research Centre, Massey University, Wellington
Free phone: 0800 SNOOZE (766693)
Email: r.gibson@massey.ac.nz

With age sleep becomes more disrupted including:

- Shallow and fragmented sleep
- Early morning awakenings
- Daytime sleepiness and napping
- Increased sleep disorders including snoring, insomnia or restlessness

This can lead to:

- Poor mood (feeling low or upset)
- Less able to pay attention or think clearly
- Risk of accidents (e.g. driving or falls)
- Poorer general health

Sleep and Dementia

For those with Alzheimer's disease or other types of dementia, sleep can become more fragmented, and sleep timing may be more unusual. Some also experience confused awakenings in the night. Sleep disruptions may contribute to waking symptoms of dementia.

Caregivers may also have disrupted sleep due to providing support in the night, or increased trouble getting back to sleep after an awakening.

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Some Sleep Hints and Tips

- Try to maintain a regular routine
- Avoid eating or drinking too much before bed (but also avoid going to bed hungry)
- Avoid alcohol, cigarettes or caffeine before bed
- Have a relaxing pre-sleep routine to help mind and body relax and fall asleep
- Keep the bedroom a "safe sleep" zone for example:
  - appropriate lighting for sleep and safety
  - block out disturbing noise
  - avoid watching TV, or listening to radio in bed
  - make the bed nice and comfortable
  - check your bedroom is a comfortable temperature
- If you don't fall asleep in about 20 minutes, get out of bed and spend a little time doing a relaxing activity before going back to trying to sleep

Source: National Sleep Foundation

Do you or your partner have Alzheimer's or dementia & trouble sleeping?

Sleep is important for all of us. But for those affected by dementia it is often problematic. It's important to better understand sleep so we can help manage dementia-related sleep problems.

People with dementia and their supporting partners across Wellington (inc. Hutt and kapiti) are needed to take part in a sleep study.
This takes place in the home and involves simple sleep monitoring, questionnaires, and trying some routine activities to help with sleep.

If you would like to find out more about this exciting study, please contact Rosie Gibson:

Free phone: 0800 SNOOZE (766693) **Phone:** 04 3800635
Email: r.gibson@massey.ac.nz

Research

Sleep Solutions

Rosie Gibson is a Massey University Doctoral Candidate. She is researching ways to improve sleep for people with dementia living in the community, through working with them and their caregivers. Below she outlines the problem of sleep as well as the exciting research project which is taking place in Wellington.

Sleep is important for all of us, but as we get older it often becomes problematic. As we age, it is normal for sleep to become shallower and fragmented, with early morning awakenings, and often daytime sleepiness. Sleep disorders including snoring, insomnia or restless legs also become more common with age.

For those affected by dementia (such as Alzheimer's disease), sleep often becomes even more fragmented, and sleep timing more unusual than in healthy ageing. Some people also experience confused awakenings and wandering type behaviours in the night.

Family caregivers often have disrupted sleep as well. This may be due to having different sleep timing to those in their care, being woken or providing support in the night, or increased trouble getting to sleep.

Sleep disruptions have been found to contribute to problems with memory, mood and daytime functioning. Therefore managing sleep of people affected by dementia may have a significant impact on the waking symptoms of dementia, quality of life, and coping.

My research aims to:

1. Better understand dementia-related sleep problems of couples living in the community.
2. Assess whether strategies aimed at improving the sleep of those with dementia are feasible to use in the home.

Background of the project

We each have a circadian body clock in the brain, which is the pacemaker for daily rhythms of many bodily functions including the sleep/wake cycle, helping keep us alert in the daytime and asleep at night. With dementia, the body clock typically reduces in size and activity, and also becomes less sensitive to the cues that keep it in step with the day/night cycle.

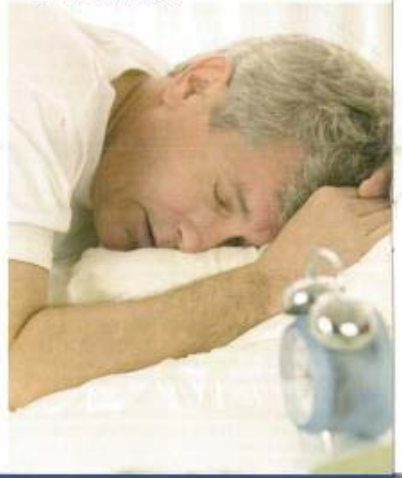
The body clock is usually kept in step with the 24 hour day through light input via a special pathway from the eyes. Routine physical activity and socialising can also help. However for many people with dementia, time spent in bright light or engaged in activities is compromised.

This research is trialling techniques to boost the activity within the body clock of people with dementia. The study includes both people with dementia and their supporting partner, and involves home-based light therapy and physical activities to try and incorporate into the routine of those with dementia. Participating pairs will also be given a sleep information booklet.

What the project entails...

The study involves couples consisting of a person with dementia and their supporting partner. It takes a total of 6 weeks to complete, takes place in the home and involves routine tasks. Couples can enroll regardless of any work, club, or respite commitments.

- During the first week your sleep will be monitored. This involves wearing a small watch-like device on your wrist that measures movement, and keeping a sleep diary. We would also bring some questionnaires concerning sleep, mood and daytime functioning.



- For weeks 2-6 you will be provided with a light box and exercise program to try to incorporate into the routine of the partner with dementia when possible. You will also receive the sleep information booklet.
- During the final week of trialling the light and exercise you will be given the questionnaires and sleep monitors again to assess whether there have been any changes.
- At the conclusion of the sleep study you will be sent you your sleep monitor reports. The final study results will be mailed to you when they become available.

In order to understand and manage dementia-related sleep problems Rosie needs more volunteers!

If you or your partner have Alzheimer's or other type of dementia and have troubled sleep, this project may be of interest to you!

She is looking for couples consisting of a person with dementia and their supporting partner who:

- Live in the Wellington region (including Hutt Valley and Kapiti Coast)
- Are aged 65 and over
- Are interested in improving their sleep

Rosie believes participants so far have found the study simple and rewarding. It all takes place in the home and you would have the right to withdraw at any time.

If you would like to ask any questions or have a study information pack sent to you please contact Rosie at the Sleep/Wake Research Centre:

Rosie Gibson
Free phone: 0800 SNOOZE (766693)
Phone: 04 380 0635
Email: r.gibson@massey.ac.nz

Tai Chi

Matthew Croucher, Susan Gee, Margaret Francis, Rachael Beever and Gillian Bastion (grant recipients in 2009) have completed their project "A settling effect at a difficult time of day. Does Tai Chi have beneficial effects on challenging behaviour in an inpatient psychogeriatric ward?". Matthew Croucher reports:

"The CDHB-hosted Psychiatry of Old Age Academic Unit recently evaluated the short-term effect of Tai Chi on behavioural disturbance in a dementia specialty inpatient ward. The Alzheimer's NZ Charitable Trust and the Canterbury Health Care of the Elderly Education Trust kindly gave grants to enable two summer studentships in 2009 / 2010 as well as equipment to assist with this research. A randomised controlled study was conducted comparing three conditions - standard care, music, and Tai Chi in the mid afternoon. This is a time of "sundowning" and increased frequency of challenging behaviours from some people with dementia. Although no difference between the conditions was found for direct observational measures of agitation or changes in emotion in the late afternoon, a combined measure of the use of non-scheduled psychotropic medication and use of physical restraint was slightly but significantly lower on Tai Chi days than on standard care days. The study has also provided valuable experience in the use of formal measuring tools for research into challenging behaviour so that advice can be given to future researchers. The findings have been presented at the 2010 "Change Champions" conference in Sydney and a formal journal article is being submitted to a health care scientific journal early in 2011."

Funding dementia research

The Alzheimers New Zealand Charitable Trust is solely dedicated to dementia research. The Trust's primary objective is to support individuals and organisations who are providing medical and social research for the benefit of people (directly or indirectly) affected by dementia.

The closing date for the next funding round has been extended. Up to $15,000 is available for research focused on the medical and social aspects of dementia. Any relevant topic will be considered. The closing date for applications is now Friday 27 January 2012. Applicants should follow the information provided in the document "Small Project Grants" available at http://www.alzheimersresearch.org.nz.

ALZHEIMERS™
NEW ZEALAND CHARITABLE TRUST
Promoting and supporting dementia research

In 2010 an Alzheimers New Zealand Charitable Trust research grant of $14,715 was awarded for the study *Sleep of older people with dementia and those who live them: Pilot of an intervention* (Miss Rosemary Gibson, Professor Philippa Gander, Dr Linda Jones and Professor Tony Dowell).

5 Questionnaire: PWD

'SLEEP~WAKE' research centre
'MOE TIKA~MOE PAI'

SLEEP STUDY

If you have any questions, please call 0800 SNOOZE (766693) or 04 3800635

- Where possible, this questionnaire should be answered by the participant .
- Please try to answer all the questions and work through the questions together where needed

Most questions require you to TICK your answer in a box or circle a response. If you make a mistake, cross out the incorrect box and tick the correct answer. Only tick one option unless otherwise instructed.

Examples of how to mark the questionnaire

Age [68] Years

	Yes	No
To answer "no"	☐	☑
To answer "yes"	☑	☐

Office Use Only [ ] [ ] [ ]

TODAY'S DATE: _____ / _____ / _____
Day Month Year

A LITTLE ABOUT YOURSELF

1. What is your current age? ☐ Years
2. What is your gender? ☐ Male ☐ Female

YOUR SLEEP

The following questions relate to your usual sleep habits during the <u>past month</u> only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

During the past month....

3. What time have you usually gone to bed at night? ☐
4. How long (in minutes) has it usually taken you to fall asleep each night? ☐
5. What time have you usually got up in the morning? ☐
6. How many hours of actual sleep did you get at night? ☐
   (This may be different than the number of hours you spent in bed.)

	Yes	No	Sometimes
7. Do you nap during the day?	☐	☐	☐

	Hours	Mins
If yes, for approximately how long in total per day? (considering the past month)?	☐	☐

	Yes	No
8. Have you been diagnosed with a sleep disorder?	☐	☐

If yes, which sleep disorder (e.g. insomnia, obstructive sleep apnoea) and when were you diagnosed?

Type of sleep disorder	Year diagnosed
☐	☐

9. During the past month, how often have you had trouble sleeping because you...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week	Don't know
a) Cannot get to sleep within 30 minutes					
b) Wake up in the middle of the night or early morning					
c) Have to get up to use the bathroom					
d) Cannot breathe comfortably					
e) Cough or snore loudly					
f) Feel too cold					
g) Feel too hot					
h) Have bad dreams					
i) Have pain					
j) Other reason(s), please describe:					
10. **During the past month, how often have you taken medicine to help you sleep (prescribed or 'over the counter')?**					
11. **During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activities?**					

12. **During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?**	No problem at all	Only a slight problem	Somewhat of a problem	A very big problem
13. **During the past month, how would you rate your sleep quality overall?**	Very good	Fairly good	Fairly bad	Very bad

If they are able, please ask your partner or family member to help answer these questions (you may not know because they are about things that happen when you are asleep).

14. How often in the past month you have had...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week	Don't Know
a) Loud snoring					
b) Long pauses between breaths while asleep					
c) Legs twitching or jerking while you sleep					
d) Episodes of disorientation or confusion during sleep					
e) Other restlessness while you sleep, describe:					

Improving Sleep of People with Dementia and Those who Live with Them Questionnaire for PWD Version 3 27.03.2012

HEALTH

The following questions focus on health problems you may have. Please tick the boxes marked 'yes' to indicate if a doctor, nurse or other health care worker has told you that you have any of the following health problems. If you have a particular problem, please also indicate whether you receive treatment for it and if it limits your activities.

15.	Problem	Do you have the problem?		Do you receive treatment for it?		Does it limit your activities?	
		No	Yes →	No	Yes	No	Yes
a)	Heart disease	N	Y	N	Y	N	Y
b)	High blood pressure	N	Y	N	Y	N	Y
c)	Lung disease	N	Y	N	Y	N	Y
d)	Diabetes	N	Y	N	Y	N	Y
e)	Ulcer or stomach disease	N	Y	N	Y	N	Y
f)	Kidney disease	N	Y	N	Y	N	Y
g)	Liver disease	N	Y	N	Y	N	Y
h)	Anemia or blood disease	N	Y	N	Y	N	Y
i)	Cancer	N	Y	N	Y	N	Y
j)	Depression	N	Y	N	Y	N	Y
k)	Osteoarthritis, degenerative arthritis	N	Y	N	Y	N	Y
l)	Back pain	N	Y	N	Y	N	Y
m)	Rheumatoid arthritis	N	Y	N	Y	N	Y
n)	Other medical problems (please write in)						
	Other…	N	Y	N	Y	N	Y
	Other…	N	Y	N	Y	N	Y

16. Have you been diagnosed as having dementia? Yes ☐ No ☐

If yes, what type of dementia were you diagnosed with? Type:

What year were you diagnosed? Year:

MEDICATIONS

17. **Please use this space to list the medications you currently take (if different from those given on consent form)**

Medication Name	Dose

Thank You For Taking The Time To Complete This Questionnaire

Please Use This Space For Any Other Comments You'd Like To Make

Improving Sleep of People with Dementia and Those who Live with Them Questionnaire for PWD Version 3 27.03.2012

Researcher Signature _____
Date _____

QUALITY OF LIFE

The following questions are about your quality of life. The researcher will ask you about each area listed below and ask you to point to your answer.

a)	Physical Health	*Poor*	*Fair*	*Good*	*Excellent*
b)	Energy	*Poor*	*Fair*	*Good*	*Excellent*
c)	Mood	*Poor*	*Fair*	*Good*	*Excellent*
d)	Living situation	*Poor*	*Fair*	*Good*	*Excellent*
e)	Memory	*Poor*	*Fair*	*Good*	*Excellent*
f)	Family	*Poor*	*Fair*	*Good*	*Excellent*
g)	Marriage/closest relationship status	*Poor*	*Fair*	*Good*	*Excellent*
h)	Friends	*Poor*	*Fair*	*Good*	*Excellent*
i)	Self as whole	*Poor*	*Fair*	*Good*	*Excellent*
j)	Ability to do chores around the house	*Poor*	*Fair*	*Good*	*Excellent*
k)	Ability to do things for fun	*Poor*	*Fair*	*Good*	*Excellent*
l)	Money	*Poor*	*Fair*	*Good*	*Excellent*
m)	Life as a whole	*Poor*	*Fair*	*Good*	*Excellent*

Mini-Mental Status Exam (1)

Years of schooling .....highest level attained.....

Activity **Score**

Orientation – 1 point for each answer

Ask: "What is the (year)(season)(date)(day)(month)?" _____/5
Ask: "Where are we? (country)(province)(city)(street)(office) _____/5

Registration – 1 point for each object properly repeated

Name three objects: e.g. SHOE CAR APPLE (The examiner should pronounce the words at a rate of one per second with up to 5 repeat attempts)
Ask the patient to: repeat all three after you have said them.
Repeat them until the patient learns all three. _____/3

Attention & Calculation – 1 point for each correct subtraction
e.g. Serial 7's. For 5 subtractions (93, 86, 79, 72, 65)
OR - Ask the patient to: spell WORLD forward, then backwards
(D-L-R-O-W: 1 point for each correct letter backwards) _____/5

Recall - 1 point for each correct answer

Ask the patient to: name the three objects from above. _____/3

Language

Ask the patient to:

- identify and name a pencil and a watch (1 pt each) _____/2
- repeat the phrase, "No ifs, ands or buts." _____/1
- "Take a paper in your right hand, fold it in half, and put it on the floor" (1 point for each task completed properly) _____/3
- read & obey the following:"Close your eyes" _____/1
- write a sentence. _____/1
- copy a complex diagram of two interlocking pentagons _____/1

TOTAL: _____/30

6 Questionnaire: Carer

'SLEEP~WAKE' research centre
'MOE TIKA~MOE PAI'

SLEEP STUDY

Questionnaire For Family Carer

If you have any questions, please call 0800 SNOOZE (766693) or 04 3800635

- This is a questionnaire concerning the participant who is the caregiver. You can complete this in your own time, or with the researcher present if you prefer.
- Please try to answer all the questions and work through the questions together if needed.

Most questions require you to TICK your answer in a box or circle a response. If you make a mistake, cross out the incorrect box and tick the correct answer. Only tick one option unless otherwise instructed.

Examples of how to mark the questionnaire

Age [68] Years

	Yes	No
To answer "no"	☐	☑
To answer "yes"	☑	☐

Office Use Only ☐☐☐

TODAY'S DATE: _____ / _____ / _____
Day Month Year

A LITTLE ABOUT YOURSELF

1. What is your current age? ☐ Years
2. What is your gender? ☐ Male ☐ Female

3. Do you currently work in paid employment outside your home? Yes ☐ No ☐

   If yes, on average how many hours per week do you work in paid employment outside your home? ☐ Hours

YOUR SLEEP

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

During the past month....

4. What time have you usually gone to bed at night? ☐
5. How long (in minutes) has it usually taken you to fall asleep each night? ☐
6. What time have you usually got up in the morning? ☐
7. How many hours of actual sleep did you get at night? ☐
   (This may be different than the number of hours you spent in bed.)
8. Do you nap during the day? Yes ☐ No ☐ Sometimes ☐

   If yes, for approximately how long in total per day? (considering the past month) Hours ☐ Mins ☐

9. Have you been diagnosed with a sleep disorder? Yes ☐ No ☐

   If yes, which sleep disorder (e.g. insomnia, obstructive sleep apnoea) and when were you diagnosed?

Type of sleep disorder	Year diagnosed

10. During the past month, how often have you had trouble sleeping because you...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week	Don't know
a) Cannot get to sleep within 30 minutes					
b) Wake up in the middle of the night or early morning					
c) Have to get up to use the bathroom					
d) Cannot breathe comfortably					
e) Cough or snore loudly					
f) Feel too cold					
g) Feel too hot					
h) Have bad dreams					
i) Have pain					
j) Other reason(s), please describe:					
11. During the past month, how often have you taken medicine to help you sleep (prescribed or 'over the counter')?					
12. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activities?					

13. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?	No problem at all	Only a slight problem	Somewhat of a problem	A very big problem
14. Where do you and your spouse/partner usually sleep?	Separate rooms	Same room but not bed	Same bed	
15. During the past month, how would you rate your sleep quality overall?	Very good	Fairly good	Fairly bad	Very bad

If they are able, please ask your partner/family member to help answer these questions (you may not know because they are about things that happen when you are asleep).

16. How often in the past month you have had...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week	Don't know
a) Loud snoring					
b) Long pauses between breaths while asleep					
c) Legs twitching or jerking while you sleep					
d) Episodes of disorientation or confusion during sleep					
e) Other restlessness while you sleep, describe:					

YOUR FEELINGS

The following questions focus on your feelings. Please circle the response that most applies to you for each feeling listed below.

17. **Answer how you currently would describe your feelings and give your most immediate response.**

a)	**I feel tense or 'wound up'**	*Not at all*	*Occasionally*	*A lot of the time*	*Most of the time*
b)	**I still enjoy the things I used to enjoy**	*Hardly at all*	*Only a little*	*Not quite as much*	*Definitely as much*
c)	**I get a sort of frightened feeling as if something awful is about to happen**	*Not at all*	*A little, doesn't worry me*	*Yes, but not badly*	*Very definitely and quite badly*
d)	**I can laugh and see the funny side of things**	*Not at all*	*Definitely not so much now*	*Not quite as much now*	*As much as I always could*
e)	**Worrying thoughts go through my mind**	*Only occasionally*	*From time to time, not too often*	*A lot of the time*	*A great deal of the time*
f)	**I feel cheerful**	*Not at all*	*Not often*	*Sometimes*	*Most of the time*
g)	**I can sit at ease and feel relaxed**	*Not at all*	*Not often*	*Usually*	*Definitely*
h)	**I feel as if I am slowed down**	*Not at all*	*Sometimes*	*Very often*	*Nearly all the time*
i)	**I get a sort of frightened feeling like ' butterflies' in my stomach**	*Not at all*	*Occasionally*	*Quite often*	*Very often*
j)	**I have lost interest in my appearance**	*I take as much care as ever*	*I may not take quite as much care*	*I don't take as much care as I should*	*Definitely*
k)	**I feel restless as I have to be on the move**	*Not at all*	*Not very much*	*Quite a lot*	*Very much indeed*
l)	**I look forward with enjoyment to things**	*Hardly at all*	*Definitely less than I used to*	*Rather less than I used to*	*As much as I ever did*
m)	**I get sudden feelings of panic**	*Not at all*	*Not very often*	*Quite often*	*Very often indeed*
n)	**I can enjoy a good book or radio or TV program**	*Very seldom*	*Not often*	*Sometimes*	*Often*

HEALTH

The following questions focus on health problems you may have. Please tick the boxes marked 'yes' to indicate if a doctor, nurse or other health care worker has told you that you have any of the following health problems. If you have a particular problem, please also indicate whether you receive treatment for it and if it limits your activities.

18.	Problem	Do you have the problem?		Do you receive treatment for it?		Does it limit your activities?	
		No	Yes →	No	Yes	No	Yes
a)	Heart disease	N	Y	N	Y	N	Y
b)	High blood pressure	N	Y	N	Y	N	Y
c)	Lung disease	N	Y	N	Y	N	Y
d)	Diabetes	N	Y	N	Y	N	Y
e)	Ulcer or stomach disease	N	Y	N	Y	N	Y
f)	Kidney disease	N	Y	N	Y	N	Y
g)	Liver disease	N	Y	N	Y	N	Y
h)	Anemia or blood disease	N	Y	N	Y	N	Y
i)	Cancer	N	Y	N	Y	N	Y
j)	Depression	N	Y	N	Y	N	Y
k)	Osteoarthritis, degenerative arthritis	N	Y	N	Y	N	Y
l)	Back pain	N	Y	N	Y	N	Y
m)	Rheumatoid arthritis	N	Y	N	Y	N	Y
n)	Other medical problems (please write in)						
	Other…	N	Y	N	Y	N	Y
	Other…	N	Y	N	Y	N	Y

IMPACT OF CAREGIVING

19. **How long have you been providing dementia related support for your family member?**

Years ☐ Months ☐

20. **On average how much time do you spend providing care each day? (please tick one box)**

All day and night ☐ All day ☐ All night ☐ Several hours ☐ About an hour ☐

21. **Do you also provide care/support for others?** Yes ☐ No ☐

22. **Do you have any support in terms of relief from care-giving, house work, daily chores?** Yes ☐ No ☐

23. **Answer the questions below with regards to how you currently would describe your feelings and give your most immediate response**

		Always	Often	Sometimes	Never	Not applicable
a)	Overall do you feel well supported in your role of caregiver?	☐	☐	☐	☐	☐
b)	Do you feel you cope well as a caregiver?	☐	☐	☐	☐	☐
c)	Do you find caregiving too demanding?	☐	☐	☐	☐	☐
d)	Does caregiving cause difficulties in your relationships with friends?	☐	☐	☐	☐	☐
e)	Does caregiving have a negative effect on your physical health?	☐	☐	☐	☐	☐
f)	Does caregiving cause difficulties in your relationship with your family?	☐	☐	☐	☐	☐
g)	Does caregiving cause you financial difficulties?	☐	☐	☐	☐	☐
h)	Do you feel trapped in your role as a caregiver?	☐	☐	☐	☐	☐
i)	Do you feel well supported by your friends and/or neighbours?	☐	☐	☐	☐	☐
j)	Do you find caregiving worthwhile?	☐	☐	☐	☐	☐
k)	Do you feel well supported by your family?	☐	☐	☐	☐	☐
l)	Do you have a good relationship with the person you care for?	☐	☐	☐	☐	☐
m)	Do you feel well supported by health and social services? (for example, public, private, voluntary)	☐	☐	☐	☐	☐
n)	Do you feel that anyone appreciates you as a caregiver?	☐	☐	☐	☐	☐
o)	Does caregiving have a negative effect on your emotional well-being?	☐	☐	☐	☐	☐

MEDICATIONS

24. **Please use this space to list the medications you currently take (if different from those given on consent form)**

Medication Name	Dose

PLEASE USE THIS SPACE FOR ANY OTHER COMMENTS YOU'D LIKE TO MAKE ABOUT YOURSELF

Please turn over for final questions

Improving Sleep of People with Dementia and Those who Live with Them Questionnaire for carer Version 3 27.03.2012

QUESTIONS CONCERNING THE FAMILY MEMBER IN YOUR CARE

The following sets of questions concern the sleep, quality of life, and memory and behaviour of participants with dementia. These are to be answered from the perspective of the supporter.

SLEEP PROBLEMS RELATED TO DEMENTIA

Please indicate whether any of the sleep behaviours listed below occurred during the previous 2 weeks. Use the following scales to rate the frequency, severity and amount of distress each behaviour causes you (how emotionally distressing do you find this behaviour?):

Scale	FREQUENCY	Scale	SEVERITY	Scale	CARER DISTRESS
0	Not present in the last 2 weeks	0	Not present	0	Not at all
1	Less than once per week	1	Mild: night-time behaviours occur but are not particularly disruptive	1	Minimally
2	One to two times per week	2	Moderate: night-time behaviours occur and disturb both person with dementia and the sleep of the caregiver; more than one type of night-time behaviours may be present	2	Mildly
3	Several times per week but less than every day	3	Marked: night-time behaviours occur; several types of night-time behaviours may be present; the person with dementia is very distressed during the night and the caregiver's sleep is markedly disturbed	3	Moderately
4	Once or more per day (every night)			4	Severely
				5	Very severely/extremely

25.	During the past 2 weeks did any of these sleep behaviours occur?	Frequency	Severity	Carer distress	Not sure/NA
a)	Difficulty falling asleep	☐	☐	☐	☐
b)	Getting up during the night (do not count if the subject gets up once or twice per night to go to the bathroom and quickly falls back to sleep)	☐	☐	☐	☐
c)	Wandering, pacing or getting involved in inappropriate activities at night	☐	☐	☐	☐
d)	Awakening others during the night	☐	☐	☐	☐
e)	Awakening at night, dressing, and planning to go out, thinking that it is morning and time to start the day	☐	☐	☐	☐
f)	Awakening too early in the morning (earlier than is habit)	☐	☐	☐	☐
g)	Sleeping excessively during the day	☐	☐	☐	☐
h)	Other night-time behaviours that are bothersome	☐	☐	☐	☐

MEMORY AND BEHAVIOUR OF YOUR FAMILY MEMBER

The following is a list of problems those with dementia sometimes have. Please indicate if any of these problems have occurred in the past week.
If so, how much has this bothered you or upset you when it happened?

Use the following scales for frequency of the problem and your reaction to it. Please circle your answers.

Frequency ratings		Reaction Ratings	
0	Never occurred	0	Not at all
1	Not in the past week	1	A little
2	1-2 times in the past week	2	Moderately
3	3-6 times in the past week	3	Very much
4	Daily or more often	4	Extremely
9	Don't know or not applicable	9	Don't know or not applicable

26.		Frequency	Reaction
a)	Asking the same question over and over	0 1 2 3 4 9	0 1 2 3 4 9
b)	Trouble remembering recent events (e.g. items in the newspaper or on TV)	0 1 2 3 4 9	0 1 2 3 4 9
c)	Trouble remembering significant past events	0 1 2 3 4 9	0 1 2 3 4 9
d)	Losing or misplacing things	0 1 2 3 4 9	0 1 2 3 4 9
e)	Forgetting what day it is	0 1 2 3 4 9	0 1 2 3 4 9
f)	Starting, but not finishing things	0 1 2 3 4 9	0 1 2 3 4 9
g)	Difficulty concentrating on a task	0 1 2 3 4 9	0 1 2 3 4 9
h)	Destroying property	0 1 2 3 4 9	0 1 2 3 4 9
i)	Does things that embarrass you	0 1 2 3 4 9	0 1 2 3 4 9
j)	Waking you or other family members up at night	0 1 2 3 4 9	0 1 2 3 4 9
k)	Talking loudly and rapidly	0 1 2 3 4 9	0 1 2 3 4 9
l)	Appears anxious or worried	0 1 2 3 4 9	0 1 2 3 4 9
m)	Engaging in behaviour that is potentially dangerous to self or others	0 1 2 3 4 9	0 1 2 3 4 9
n)	Threats to hurt oneself	0 1 2 3 4 9	0 1 2 3 4 9
o)	Threats to hurt others	0 1 2 3 4 9	0 1 2 3 4 9
p)	Aggressive to others verbally	0 1 2 3 4 9	0 1 2 3 4 9
q)	Appears sad or depressed	0 1 2 3 4 9	0 1 2 3 4 9
r)	Expressing feelings of hopelessness or sadness about the future (e.g. " nothing worthwhile ever happens", " I never do anything right")	0 1 2 3 4 9	0 1 2 3 4 9
s)	Crying and tearfulness	0 1 2 3 4 9	0 1 2 3 4 9

Continued over page

MEMORY AND BEHAVIOUR CONTINUED...
Please indicate if any of these problems have occurred in the past week and If so, how much has this bothered you or upset you when it happened?

		Frequency						Reaction					
t)	Commenting about death of self or others (e.g., "life isn't worth living", "I'd be better off dead")	0	1	2	3	4	9	0	1	2	3	4	9
u)	Talking about feeling lonely	0	1	2	3	4	9	0	1	2	3	4	9
v)	Comments about feeling worthless or being a burden to others	0	1	2	3	4	9	0	1	2	3	4	9
w)	Comments about feeling like a failure or about not having any worthwhile accomplishments in life	0	1	2	3	4	9	0	1	2	3	4	9
x)	Arguing, irritability, and/or complaining	0	1	2	3	4	9	0	1	2	3	4	9

QUALITY OF LIFE OF YOUR FAMILY MEMBER
Please rate your family member's current situation, as you see it. Please circle the corresponding answer for each aspect contributing to quality of life (your family member will also complete this scale as a one-on-one interview with the researcher).

27.

a)	Physical health	*poor*	*fair*	*Good*	*Excellent*
b)	Energy level	*poor*	*fair*	*Good*	*Excellent*
c)	Mood	*poor*	*fair*	*Good*	*Excellent*
d)	Living situation	*poor*	*fair*	*Good*	*Excellent*
e)	Memory	*poor*	*fair*	*Good*	*Excellent*
f)	Family and family relationship	*poor*	*fair*	*Good*	*Excellent*
g)	Marriage/closest relationship status	*poor*	*fair*	*Good*	*Excellent*
h)	Relationship with friends	*poor*	*fair*	*Good*	*Excellent*
i)	Self as a whole	*poor*	*fair*	*Good*	*Excellent*
j)	Ability to do chores around the house	*poor*	*fair*	*Good*	*Excellent*
k)	Ability to do things for fun	*poor*	*fair*	*Good*	*Excellent*
l)	Money/financial situation	*poor*	*fair*	*Good*	*Excellent*
m)	Life as a whole	*poor*	*fair*	*Good*	*Excellent*

THANK YOU FOR TAKING THE TIME TO COMPLETE THIS QUESTIONNAIRE

PLEASE USE THIS SPACE FOR ANY OTHER COMMENTS YOU'D LIKE TO MAKE

Improving Sleep of People with Dementia and Those who Live with Them Questionnaire for carer Version 3 27.03.2012

Mini-Mental Status Exam (2)

Years of schooling .....highest level attained.....

Activity **Score**

Orientation – 1 point for each answer

Ask: "What is the (year)(season)(date)(day)(month)?" _____/5
Ask: "Where are we? (country)(province)(city)(street)(office) _____/5

Registration – 1 point for each object properly repeated

Name three objects: e.g. SHOE CAR APPLE (The examiner should pronounce the words at a rate of one per second with up to 5 repeat attempts)
Ask the patient to: repeat all three after you have said them.
Repeat them until the patient learns all three. _____/3

Attention & Calculation – 1 point for each correct subtraction
e.g. Serial 7's (93, 86, 79, 72, 65)
Ask the patient to: spell WORLD forward, then backwards
(D-L-R-O-W: 1 point for each correct letter backwards) _____/5

Recall - 1 point for each correct answer

Ask the patient to: name the three objects from above. _____/3

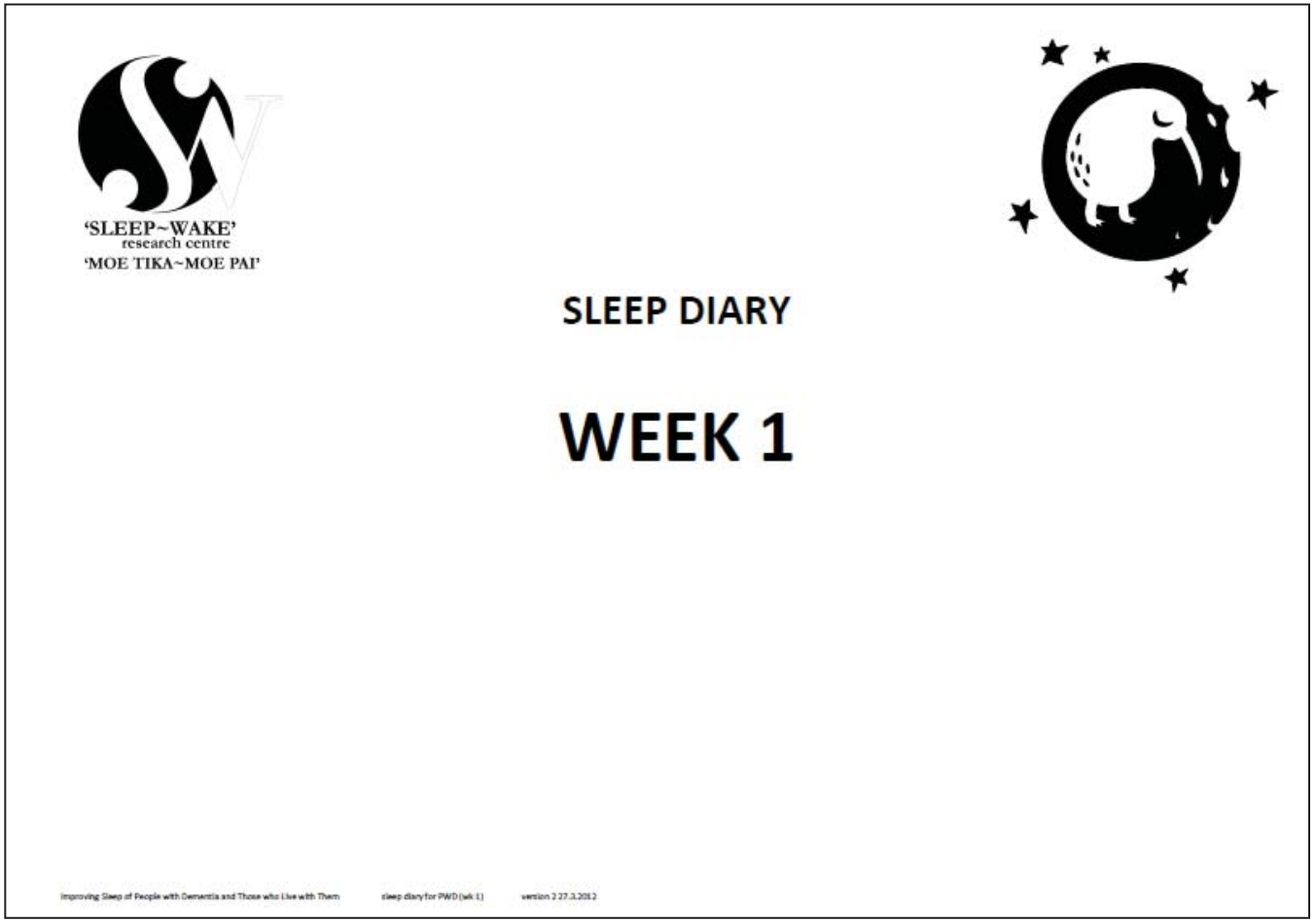
Language

Ask the patient to:

- identify and name a pencil and a watch (1 pt each) _____/2
- repeat the phrase, "No ifs, ands or buts." _____/1
- "Take a paper in your right hand, fold it in half, and put it on the floor" (1 point for each task completed properly) _____/3
- read & obey the following:"Close your eyes" _____/1
- write a sentence. _____/1
- copy a complex diagram of two interlocking pentagons _____/1

TOTAL: _____/30

7 Sleep Diary Examples



SLEEP DIARY

WEEK 1

This diary is the second part of the evaluation of your sleep. It is needed to help analyse the sleep monitor data and record how you felt you slept.

- *Please complete the diary as accurately as you can <u>every day for the week you are wearing the monitor</u>*
- *It should only take about 5-10 minutes to complete each day*
- *It does not matter what day of the week you start*
- *Please make sure that you include all sleeps (including any daytime naps)*
- *Please note any times when the monitor is off (for example when bathing)*

Also

- *Please rate the previous night's sleep quality from 1 (extremely good) to 7 (extremely poor)*

Remember, everything you tell us is completely confidential to the team at the Sleep/Wake Research Centre. We will only report on findings from the combined data from all participants

If you have any questions about this diary or the study please feel free to call the Sleep/Wake Research Centre on 0800 SNOOZE (766693)/04 3800635. All calls will be treated as confidential and you do not need to provide your name or the survey number.

Thank you for your participation.

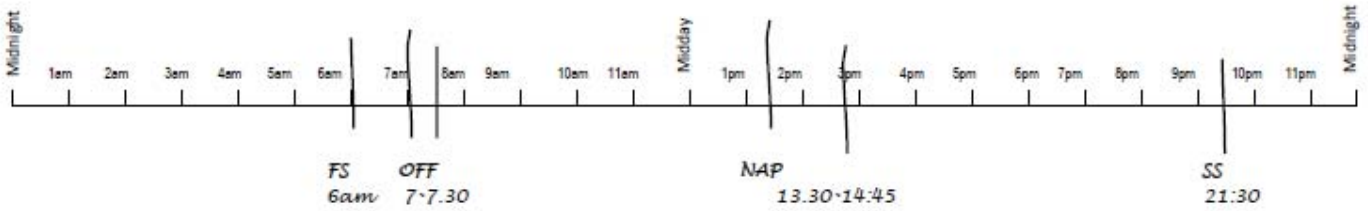
Sleep Diary Example

Each line represents one day (24 hours from midnight to midnight)
For each day, please complete one line. Mark the following times, writing below the clock time (to the nearest 15 minutes):

1. When you started trying to sleep (turned the light out) SS
2. When you finished trying to sleep (turned the light on) FS
3. Times when the actiwatch was removed (OFF)
4. Don't forget to include daytime naps (even if not in bed, estimate the times of sleep and wake up and mark as NAP)

EXAMPLE: Someone woke for the day at 6:00 am (FS). The actiwatch was removed from 7:00 am – 7:30 (OFF). They had a nap after lunch 13:30 – 14.45 and went to bed at 21:30 (SS).

DAY 1 (date 30/06/2011)



Improving Sleep of People with Dementia and Those who Live with Them sleep diary for PWD (wk 1) version 2 27.3.2012

Please mark the following times on the lines below:
Started trying to sleep (SS)
Finish trying to sleep (FS)
Daytime naps (NAP)
When the actiwatch was removed (OFF)

DAY 1 (date_____)

Please circle to rate last night's sleep quality:

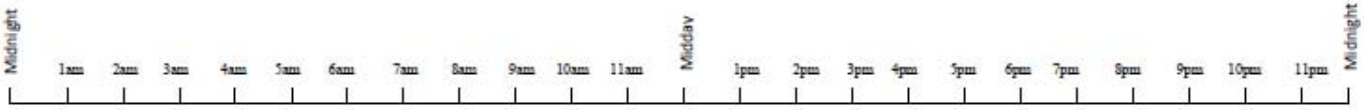
(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

DAY 2 (date_____)

Please circle to rate last night's sleep quality:

(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

DAY 3 (date_____)



Please circle to rate last night's sleep quality:

(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

Notes from week 1

For example any unusual activities, feeling unwell, sleep comments?

You have completed the first week of the sleep study!
Thank you very much for your help, we appreciate your participation in this study. Please have your completed diary with the other questionnaires and Actiwatch ready for collection.

SLEEP AND ACTIVITY DIARY

WEEKS 2-5

These diaries are to be completed during the first 4 weeks of using the light and physical activities

- *Please record the times that you incorporated the light or exercise activities into your day*
- *Also note any of the other sleep tips that you are using from the booklet*
- *Please also note your sleep times (Sleep start (SS) and Sleep Finish (FS)) as well as naps (NAP)*

Also

- *Please rate the previous night's sleep quality from 1 (extremely good) to 7 (extremely poor)*

- ➢ *Please complete the diary as accurately as you can for each day*
- ➢ *It should only take about 5-10 minutes to complete each day*
- ➢ *It does not matter what day of the week you start*

Remember, everything you tell us is completely confidential to the team at the Sleep/Wake Research Centre. We will only report on findings from the combined data from all participants

If you have any questions about this diary or the study please feel free to call the Sleep/Wake Research Centre on 0800 SNOOZE (766693)/04 3800635. All calls will be treated as confidential and you do not need to provide your name or the survey number.

Thank you for your participation.

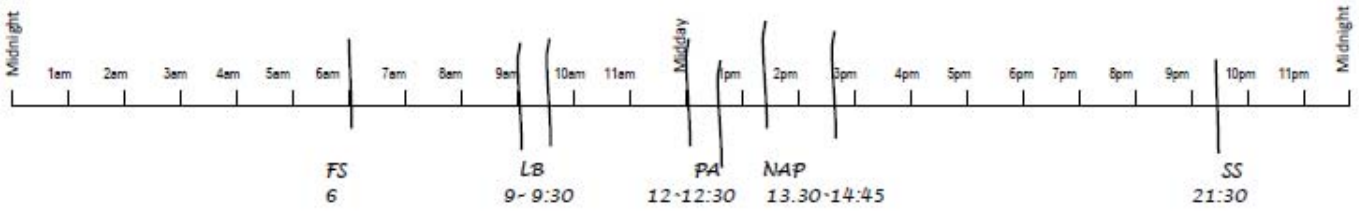
Sleep Diary Example (weeks 2-5)

Each line represents one day (24 hours from midnight to midnight)
For each day, please complete one line. Mark the following times, writing below the clock time (to the nearest 15 minutes):

1. When you used the light box (LB) or went outside into natural light (NL)
2. When you took part in physical activity (PA)
3. When you started trying to sleep (turned the light out, SS)
2. When you finished trying to sleep (turned the light on, FS)
4. Daytime naps (even if not in bed, estimate the times of sleep and wake up and mark as NAP)

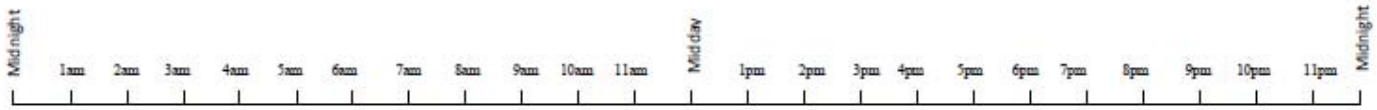
EXAMPLE: Someone woke for the day at 6:00 am (FS). They used the light box from 9- 9.30. They did some physical activity from 12- 12.30 and had a nap after lunch 13:30 – 14.45. they went to bed in the evening at 21:30

DAY 1 (date 30/06/2011)



Please mark the following times on the lines below:
Used the light box (LB) or natural light (NL)
Took part in physical activity (PA)
Started trying to sleep (SS)
Finish trying to sleep (FS)
Daytime naps (NAP)

DAY 1 (date_____)



Please circle to rate last night's sleep quality:

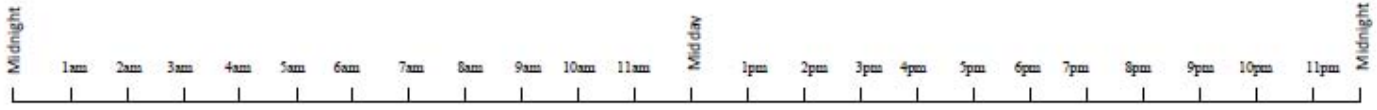
(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

DAY 2 (date_____)

Please circle to rate last night's sleep quality:

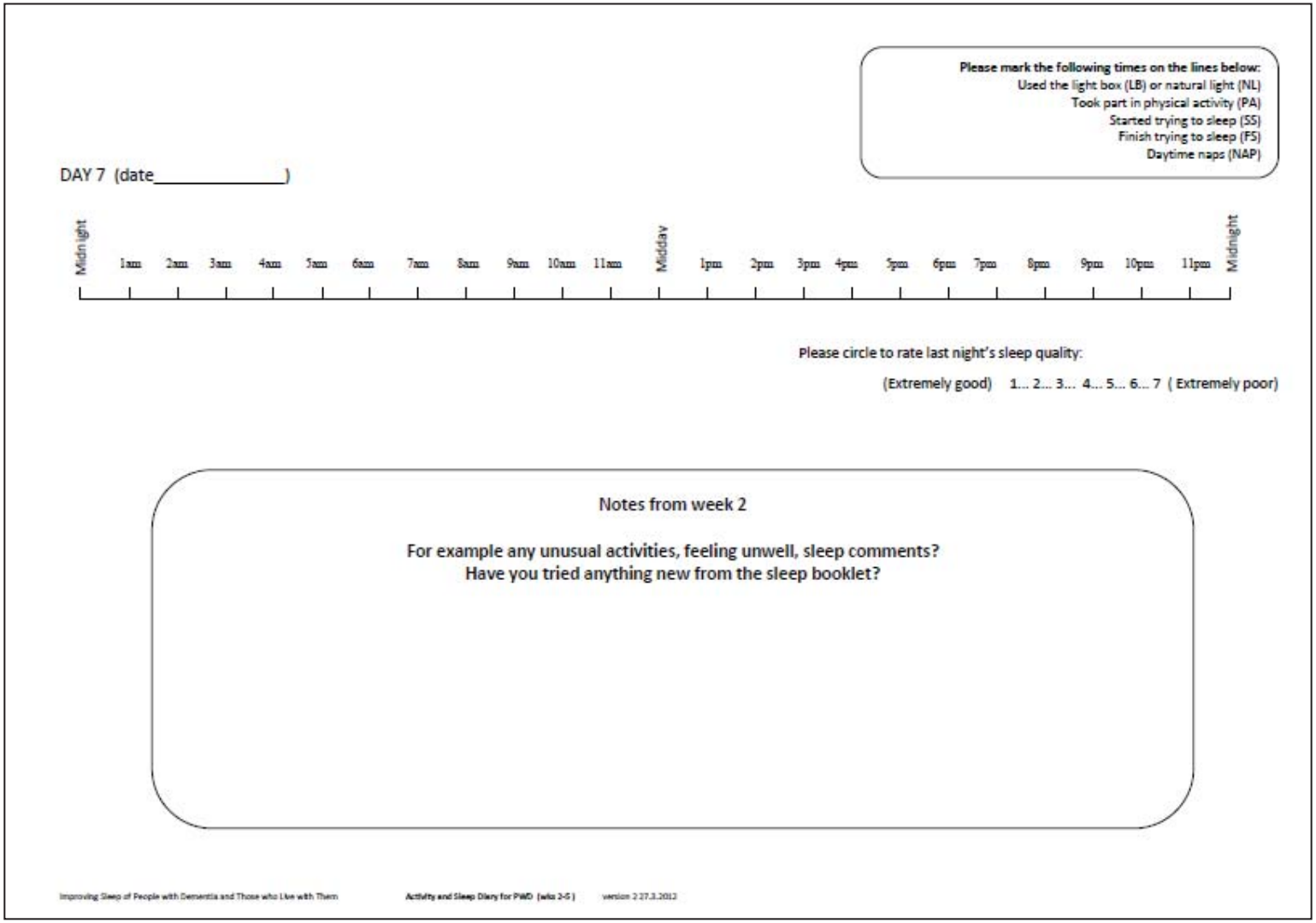
(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

Day3 (date_____)



Please circle to rate last night's sleep quality:

(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)



Please mark the following times on the lines below:
Used the light box (LB) or natural light (NL)
Took part in physical activity (PA)
Started trying to sleep (SS)
Finish trying to sleep (FS)
Daytime naps (NAP)

DAY 7 (date_____)

Midnight 1am 2am 3am 4am 5am 6am 7am 8am 9am 10am 11am Midday 1pm 2pm 3pm 4pm 5pm 6pm 7pm 8pm 9pm 10pm 11pm Midnight

Please circle to rate last night's sleep quality:

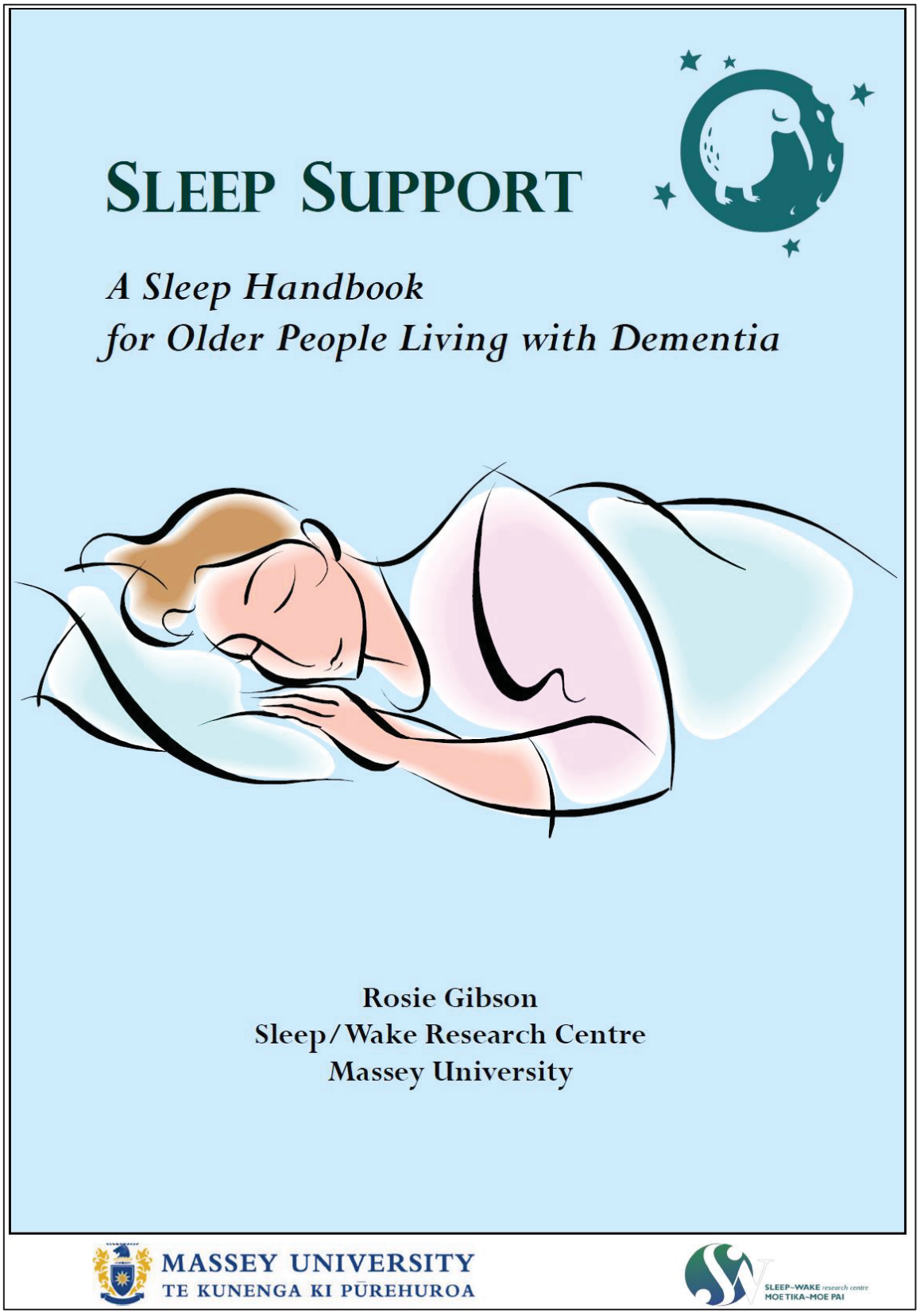
(Extremely good) 1... 2... 3... 4... 5... 6... 7 (Extremely poor)

Notes from week 2

For example any unusual activities, feeling unwell, sleep comments?
Have you tried anything new from the sleep booklet?

Improving Sleep of People with Dementia and Those who Live with Them Activity and Sleep Diary for PWD (wks 2-5) version 2 27.3.2012

8 Sleep Support Handbook



SLEEP SUPPORT

A SLEEP HANDBOOK FOR OLDER PEOPLE LIVING WITH DEMENTIA

CONTENTS

	PAGE NUMBER
WELCOME	1
THINKING ABOUT SLEEP	
• Sleep for everyone	3-4
• Sleep and ageing	5
• Problem sleep	6-7
• Sleep and dementia	8-9
• Sleep of supporters	10
FACTORS AFFECTING SLEEP	
• Napping	11
• Light and sleep	12
• Exercise and sleep	13
• Diet and sleep	14
• Mood and sleep	15
• Evening and bedtime routines	16
• Sleep environment	17-18
• Sleep and medications	19
CONCLUDING THOUGHTS	21
FURTHER READING	22
CONTACTS	23

IMPORTANT INFORMATION - PLEASE READ

The information and suggestions contained in this booklet are not intended to be used for diagnosis or treatment of any medical condition or to otherwise replace medical advice by qualified health professionals. Because each person and situation is unique, you should consult your doctor, support worker, or health professional to evaluate and guide you about your specific concerns or situation.

In the event that any loss is suffered by you as a result of you relying on any information contained in this booklet or due to errors of omissions to this booklet, you agree that Rosemary Gibson/and/or Massey University shall not be held liable for any loss suffered by you.

WELCOME

We spend a third of our lives asleep. It is important to acknowledge that, for all ages, sleep isn't just time out from wakefulness, it is an important time for renewing our physical and mental state. Like food and water, we need sleep to survive.

Research has shown that sleep becomes more disrupted with ageing. Sleep problems have been identified as a key issue for people with dementia and those who support them, because disrupted sleep can have an impact on mood, behaviour and the ability to manage during the day.

The first section of this handbook provides an overview of sleep and how it changes during ageing, for people with dementia and their supporters. There is also specific information on common sleep disorders.

The second half of the handbook focuses on the factors which can affect sleep and offers advice and tips including activities, routines and changes you may wish to make to help improve the quality of your sleep and your partner's sleep.

It is important to identify what may be causing a sleep problem. For instance it could be the environment, the physiological changes from dementia, or maybe medications being taken. It is vital to identify the causes of the problem in order to decide which strategies may be best to try.

When incorporating the ideas and suggestions from this booklet, always consider safety within your home, for example making the bedroom dark enough to promote sleep but light enough to find the way to the bathroom.

How to use this booklet

This handbook provides detailed information concerning sleep, sleep disorders, as well as specific sleep disturbances for people with dementia and supporters, and strategies to improve sleep.

As you may not necessarily have time or be inclined to read all of the material at once, this booklet is designed for you to be able to dip in and out of it. Tips and summaries are highlighted throughout.

Boxes summarising key points and tips are highlighted throughout the text as a quick reference

Look out for the pink circles of caution or advice!

SECTION 1:
THINKING ABOUT SLEEP

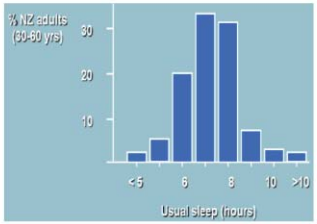
This section gives an overview of sleep and the effects of sleep loss. The aim of this section is to give some background on how sleep works and what happens when it goes wrong. Some of the more common sleep disorders are outlined as well as the changes associated with ageing, dementia and being in a supporting role.

SLEEP FOR EVERYONE

Sleepiness (like hunger or thirst) is a signal from the brain that you are not meeting a vital biological need. However sleepiness is different, because if you keep ignoring this message, eventually you will fall asleep, whether you want to or not.

How much sleep is enough?

Despite the importance of sleep, sleep loss is a very common problem in society. Work and lifestyle choices, as well as responsibilities within the family, and poor health can all contribute to sleep problems. There is no magic number of hours that we 'should' be sleeping. The graph below shows the sleep durations of New Zealand (NZ) adults (30-59 yrs old). Some report usually getting 5 hours sleep a night whereas others report over 10 hours per night. However, over a third of these people report that they are not getting enough sleep.



Data courtesy of Dr Ricci Harris (sample n=7,051)

The amount of sleep you need is best judged by how well you feel and function during the day. Do you feel better if you get an extra hour of sleep? How much do you need to feel well rested?

When you've have enough sleep, you can go through the next day without feeling tired. If sleepiness is affecting your ability to get on with your normal daily activities, you may not be getting enough or good quality sleep.

The internal structure of sleep

Sleep is made up of 2 different states:

- Rapid Eye Movement (REM) sleep is the stage of sleep in which the brain is quite active but the body is very still (except for eye movements). It is also the stage in which vivid dreams usually occur
- Non-REM is subdivided into four stages termed 1,2,3 and 4 which represent the depth of sleep (1 being the lightest, 4 being the deepest). As opposed to REM sleep, during Non-REM the brain is relatively inactive but the body can move.

Sleep cycles through these stages several times in the night, the first third of the night typically having more deep non-REM sleep, and the final third more REM sleep. The different stages and depths of sleep have different functions with regards to learning, memory and daytime functioning.

Sleep loss

Sleepiness can have severe consequences (Box 1). It reduces our capacity to perform physical or mental work. This is because sleepiness slows down our physical reaction time and mental processing, and also affects our memory. Like the effects of alcohol on performance and memory, sleep loss and sleepiness can lead to an increased risk of safety incidents and accidents at work.

BOX 1. Not enough sleep can lead to:

- Feeling tired, low energy
- Harder to concentrate
- Slower reaction times and poorer coordination
- Slower and more muddled thinking
- Feelings of irritability and/or depression
- An increased appetite (junk food)
- Weakens immune system (more susceptible to catching infections)

If you don't get enough sleep for several nights in a row, then the effects build up day-by-day. So if you don't recover your sleep 'debt' (for example on the weekends or through naps), the ability to function safely during the day becomes more difficult. When trying to recover from sleep debt, you do not literally need to replace each hour lost. Allowing yourself one or two prolonged, undisturbed sleeps will provide deeper , higher quality sleep, giving sufficient restoration for the following day.

Sleep and Ageing

Sleep is regulated by two processes. One process is the circadian body clock, a pacemaker in the brain that coordinates daily rhythms in many body functions. The circadian body clock helps keep us alert in the daytime and asleep at night. It is sensitive to light (through being connected to a special network of cells in the retina of the eyes). This light sensitivity, along with sensitivity to patterns of physical activity, keeps the circadian body clock in step with the day/night cycle. The second process which regulates sleep is our natural 'drive' for sleep. This occurs as pressure for sleep that increases with time awake and is discharged across sleep. With normal ageing, sleep changes (Figure 1).

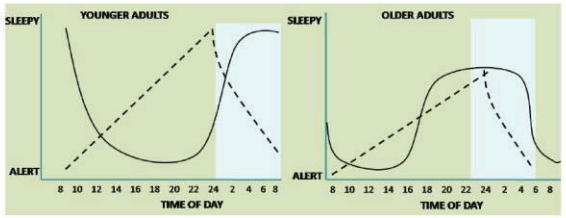


Figure 1. The two-process model of sleep and alertness (Redrawn from Wright & Frey, 2008). The circadian body clock's rhythm (solid lines) and the drive for sleep (dashed lines) become less defined as we age. The body clock may lose some of its sensitivity to environmental and the drive for sleep becomes less sensitive to prior sleep and wake patterns. These changes make us more susceptible to falling asleep in the daytime and waking up at night.

As we age, the quality of sleep also changes. We have less of the deep stages of Non-REM sleep which means that we become more susceptible to waking up in the night, and that sleep may be less restorative than when we were younger. Symptoms of menopause can often contribute to women's sleep problems, as can prostate problems for men.

In addition to aging of sleep itself, there are other things that can affect sleep (Box 2). Common medical conditions such as diabetes, respiratory disorders, obesity, pain, and depression can all contribute to a bad night's sleep (and can in turn be exacerbated by poor sleep). Medications used to treat sleep problems and other medical disorders often have side effects including daytime sleepiness and problems going to sleep. Factors affecting sleep will be addressed in more detail in part 2.

BOX 2. Other things that can affect sleep:

- Medical conditions and medications
- Activity during the day
- Light exposure during the day
- What and when we eat and drink
- How we feel
- The bedtime routine
- Sleeping environment

Problem Sleep

The prevalence of sleep problems and disorders increases as we age. Some of the more common problems and their prevalence from middle to older adulthood are shown in Box 3.

BOX 3. Prevalence of common sleep problems

Disorder	*Symptoms*	*Middle-aged adults (40-59)*	*Older adults (60+)*
Insomnia	Unable to get to sleep or stay asleep	4-12%	12-40%
Obstructive sleep apnoea	Snoring, pauses of breathing during sleep	2-5%	13-40%
Restless legs syndrome	Urge to move and discomfort in the legs when attempting to sleep	5-15%	9-20%
Visits to the bathroom	2 or more trips to urinate in the night	6-7%	27-32%

Insomnia

Insomnia is characterised by difficulty falling asleep or staying asleep, accompanied by daytime sleepiness. The prevalence of insomnia increases with age and is more common in women than men. Insomnia has been related to anxiety and depression, as well as poor physical health. Financial worries, social or family changes, bereavement and other medical conditions can all lead to symptoms of insomnia. Insomnia contributes to daytime sleepiness, poorer daytime functioning, and can have an impact on mood, concentration, and quality of life.

Obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is defined as frequent pauses in breathing during sleep. Pauses in breathing are caused by obstructions in the airway, with airway collapse due to the weaker muscles and/or less effort to breathe. Loud snoring is often present at night, which stops during the breathing pauses. The prevalence of OSA increases with the presence of other medical conditions such as heart problems, stroke, diabetes, and obesity. OSA can lead to more time awake at night, more trips to the bathroom, morning headaches, daytime sleepiness, as well as poorer daytime functioning associated with poor quality sleep and breathing.

Restless legs syndrome

Restless legs syndrome (RLS) presents as the urge to move the legs accompanied by unpleasant (some say crawling, burning, or painful) sensations in the legs. The symptoms increase in the early evening or at sleep onset. Symptoms are relieved by movement of the legs and worsen with rest. Therefore RLS prolongs the time taken to get to sleep. It is very common among older people, probably more so than indicated in box 2 as many people don't raise these symptoms with their doctor. It has also been associated with arthritis, anxiety and the side effects of medications.

Visits to the bathroom

The need to get up and urinate in the night increases with age. Reasons for this include more urine being produced in the night, and normal age-related growth of the prostate gland in men, along with other medical conditions such as diabetes, heart trouble or obesity, and the side effects of medications. Needing to get up in the night not only causes disruption to sleep, but may also lead to fall-related injuries.

REM sleep behaviour disorder

REM behaviour disorder (RBD) is the presence of uncontrollable muscular movements in REM (dreaming) sleep. It is associated with vigorous and often violent dream enacting behaviour. This includes shouting, grasping, hitting out and vivid dreams. This can cause problems with injury of the sleeper as well as the bed partner. It is more common in males over the age of 50 years.

Advanced Sleep Phase Type

Advanced sleep phase (ASP) means that the timing of sleep is abnormally early compared to a conventional or socially desired schedule. People with ASP typically complain of early sleep time (between 6-9 pm) and early morning awakening (between 2-5 am). Being more of a 'morning type' increases with age and many do not complain of ASP as it does not necessarily interfere with day-to-day life.

If you would like further information concerning a particular sleep disorder, please contact the team at the Sleep/Wake Research Centre for a pamphlet or journal article

Sleep and Dementia

People with dementia often have greater sleep disturbances than other people of their age. The cycle of the circadian body clock and the sleep drive can become less organised which contributes to disturbed sleep at night and daytime sleepiness. Sleep often becomes more fragmented as it is more difficult to go to sleep and stay asleep. Changes to sleep timing have been related to more rapid ageing of the circadian body clock with dementia compared to healthy ageing. These physiological changes begin during the early stages of dementia and progress with the disease. In the later stages a completely disrupted sleep/wake rhythm is often observed, with 1-2 hour naps occurring throughout the 24-hour day.

The likelihood of having a sleep disorder increases with dementia. For instance, sleep disordered breathing (including snoring and pauses in breathing) has been identified as two times more likely to be present in patients with Alzheimer's disease compared to a healthy population of the same age.

Sleep structure changes

The internal structure of sleep also shows changes, including less deep sleep and disrupted dreaming sleep compared to older people without dementia. These changes are thought to have effects on memory and daytime functioning. Furthermore, waking brain activity sometimes resembles that of sleep when people with dementia are behaviourally awake. This 'blurring of boundaries' may be lead to periods of agitation, confusion and hallucinations in the early evening which are typical symptoms of 'sundowning syndrome'.

Nightmares

Some people with dementia report having vivid dreams or nightmares. Such nightmares can disrupt sleep and contribute to feelings of anxiety during the day. The dreams may be related to attempting to emotionally adapt to life changes, and recurrent dreams in particular have been associated with dealing with anxiety, depression and general life stressors.

Some of the medications used to treat the symptoms of dementia have the potential side effect of nightmares. This possibility is an unfortunate outcome of treating the cognitive and behavioural aspects of dementia. If nightmares are a particular problem, it may be worth discussing the dose or timing of medications with your doctor.

Sleep can be further disrupted by aspects of dementia such as confusion in the night, medication side effects, and incontinence. Environmental cues to the circadian body clock are also reduced, with less time spent in situations with bright light, physical exercise or interacting in social activities. These more adaptable environmental influences will be covered in the second half of this handbook. Sleep disruptions for people with dementia are summarised in Box 4.

BOX 4. Sleep disruption of those with dementia:

- Disorientation and automatic behaviours at night (e.g. looking for things or dressing)
- More daytime napping (e.g. unplanned dozes after lunch or while reading)
- Some people report more vivid nightmares or hallucinations
- Sleep loss can contribute to poorer mood and functioning during the day
- Blurred boundaries between sleep and wake causes confusion and agitation in the day
- Weakening influence of the circadian body clock on sleep timing may mean that those with dementia feel like sleeping at less usual times of the day

Sleep of Supporters

Those supporting loved ones with dementia often sleep with one eye and ear open. For some it can be a bit like being a shift worker – needing to be alert both by day and night with inconsistent schedules. This on top of the age-related changes mentioned on page 5, as well as the stress and emotions related to providing care, increase the likelihood of sleep problems. Factors contributing to sleep problems for supporters can be found in Box 5 below.

Implications of sleep disturbances for supporters

Supporters of people with dementia get less sleep than non-supporters and they also have more complaints about the quality of sleep and daytime sleepiness. This has substantial implications for waking function. As sleep deprivation builds up, our ability to concentrate, make sensible decisions, act quickly and mentally cope reduce. This can influence the health of both the supporter and their partner in terms of increased risk of falls, missed medications, coping with dementia-related behaviours, as well as everyday activities such as driving and maintaining a household.

As sleep becomes more disturbed, the ability to maintain quality support reduces. In fact sleep deprivation of supporters has been identified as one of the leading factors in deciding to move family members into institutionalised care. So sleep needs to be taken seriously, both for those with dementia and for their supporters in order to deal with the day-to-day symptoms of dementia.

BOX 5. Factors contributing to poor sleep of supporters:

- Sleep disturbances and behaviour of those with dementia
- Care giving responsibilities, changes to routines, or daytime sleepiness can lead to restrictions in social and physical activities which help cue the internal body clock
- Providing care contributes to being physically tired and having to perform tasks potentially around the clock
- Night time disruptions, especially if emotionally charged, can contribute to difficulties falling back to sleep
- Changes in mood (for example feelings of loneliness, depression, anxiety) can make it difficult to get to sleep or return to sleep
- Poor coping strategies for sleep disturbances, for example taking long naps in the daytime or drinking caffeinated drinks, can exacerbate sleep problems
- Poor management of own health (such as not taking sufficient respite or maintaining own medications) can lead to increased daytime sleepiness or increase the risk of developing a sleep disorder

SECTION 2:
FACTORS AFFECTING SLEEP

This section covers some of the things which can influence sleep as well as advice on how you can make adaptations to help improve your sleep and the sleep of your partner. Bear in mind that some of the strategies covered in this package may take days or weeks to have an effect on your sleep. This is because it takes time for your body to adjust to the changes of routine. So it is important to stick with the changes you choose to make in order to see improvements.

Strategies to improve sleep can take days or weeks to have an effect so it is important to persevere

Napping

Some people like to take daytime naps, a preference which increases with age. Naps can be a good way to 'top-up' the sleep debt if you have had a poor night's sleep. However if you sleep too much in the daytime, or too late in the day, it can make getting to sleep at night more difficult. It is important to know that although you may not feel great after a short nap, you will be more alert and functional for several hours.

Dozing off or napping?

There is a big difference between unintentionally dozing off and taking an intended nap. The active "taking a nap" is often associated with preparing the environment for sleep, whereas when passively dozing off, the environment is often less conducive to sleep.

When and how much to nap...

Humans have a biological tendency to feel sleepier in the afternoon. From infancy through to old age we are more likely to feel tired between 1-4 pm, and this is the optimal window of time for taking a nap. Naps will improve your waking function, however they may leave you feeling a little groggy. Shorter naps (less than 40 minutes) are less likely to lead to the groggy feeling. If you need a longer sleep it is best to allow about 2 hours in order to cycle thorough all of the sleep stages.

BOX 6. Nap tips:

- Learn to recognise the signs of sleepiness and schedule a timely nap rather than dozing off
- A schedule encouraging activity up to an intended afternoon nap can enhance post-nap functioning and mood.
- If you or your partner are having trouble getting to sleep at night, you may want to consider reducing your daytime naps

LIGHT AND SLEEP

The circadian body clock is sensitive to light, which enables it to track the day/night cycle and helps consolidate sleep at night and waking during the day. With age our exposure to light is reduced, especially for those with physical or mental disabilities that make it more difficult to get out into natural sun light.

Timing of bright light

For those with dementia, a regular timed exposure to bright light in the day time is recommended to help keep the body clock synchronised to the day/night cycle. A good time to get this light exposure is between 9-11am. Research in care home facilities has shown that more regular exposure to bright light during the day has significant effects on sleep timing and quality as well as mood of residents with dementia. If you are exposed to bright light too early or late in the day it can affect your sleep timing. For example bright light later in the afternoon or evening could make it difficult getting to sleep at night. However,, if you get your light exposure very early in the morning you might find you become sleepy earlier than you'd like in the evening.

What if the sun isn't shining?

Even on an overcast day, natural light is still very strong and will have an effect on the body clock. But if the weather is horrid or you are unable to get outside you can still reap the benefits of bright light in your own home. A light box can be used to provide a higher intensity light than normal room lights. You need to sit a few inches away from the light box and can carry on with whatever activity you were doing so long as the light reaches your eyes. This will have the similar effects on the body clock to going outside on a sunny day. Other light tips to try indoors include sitting closer to windows during the day, keeping curtains open to allow natural light into the house, and consider using brighter general lighting in living areas.

BOX 7. Light summary and tips:

- Light is the number one cue for our body clocks to keep in step with the day/night cycle
- The regularity and timing of light exposure is very important
- People with dementia often have poorer sleep timing, so daily scheduled bright light can help bring the sleep/wake cycle back in line
- If you feel sleepy in the day, try going outside into the light or think about brighter lighting options in the home
- If you cannot get enough natural light, consider using a light box for your light exposure
- Keep the curtains open to allow the natural light in during the day
- If you are waking too early, try to reduce your light exposure first thing, for example use heavier curtains in the bedroom to block the early morning light

EXERCISE AND SLEEP

In addition to light, physical exercise is an important time cue to the body clock. The more active people are they less likely they seem to report sleep problems. Therefore regular physical exercise and social activities can be key in keeping your body clock in time and your sleep quality high.

Timing of exercise

Just 30 minutes of physical activity can have a significant effect on your sleep. Also like light, the timing of physical activities is important. A good time to do activities is in the middle of the day and early afternoon. If you perform physical activities late in the day it may make it harder to go to sleep at night. Early afternoon exercise may help dissipate the common symptoms of sundowning (such as agitation and confusion) for those with dementia. It could also reduce stress levels and work up an appetite for your evening meal as well as being an activity you can enjoy together.

Sometimes it may feel as though you don't have the time or energy for exercise, but it is important to allow this time for yourself. This may be a good opportunity to find a local fitness group or take a walk with friends.

If it is difficult to get outside or you are less physically mobile consider some stretches you could do indoors (see Box 8)

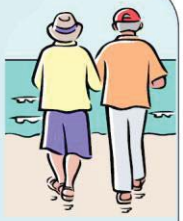
If your fitness level is very low or you have any physical conditions which may affect your ability to perform physical activities (e.g. heart or joint trouble) you should consult your doctor before undertaking any new exercise programmes.

BOX 8.
Suggestions for light exercise:

- A walk around your local park or houses
- Some gardening or housework
- Gentle yoga, pilates or tai chi class
- Activities to music or a video (even from your chair)

More vigorous activities could include:

- Dancing
- Bowls
- Swimming



DIET AND SLEEP

What we eat and when we eat can have a significant effect on being able to go to sleep and stay asleep. When we have reduced sleep, we tend to feel more hungry. This is because we lack energy and our body is less able to regulate our appetite.

Timing of meals

When to eat is important. Going to bed with a full stomach can make it difficult to get to sleep, so it is best to eat a couple of hours before bedtime and make sure that your evening meal isn't too large. Likewise you don't want to lie awake hungry so make sure to have a small snack if needed in the final hour or two before bed. Also consider how much you drink in the evening, especially if you get up a lot in the night for the toilet. Aim to eat and drink enough that you are satisfied but not too much so that you're uncomfortable in the night

Heart burn can be caused by certain foods if eaten later in the day. These foods include onions and garlic, spicy foods, (including curries or foods with mustard or chilli), tomato based foods and fatty foods (such as cheese). Keep an eye on when you eat these foods and whether they have an effect on you. Some individuals may not be so affected.

Caffeine is a stimulant that will make you feel more alert so it is best to avoid caffeinated products in the afternoon or evening. Caffeinated products include:

Product	Caffeine content (mg)
Espresso	250
Coffee (percolated)	110
Instant coffee	75
Black tea	40-60
Soft drinks	34-55
Dark eating chocolate	20
Milk eating chocolate	6
Hot or cold chocolate drink	5- 6

Watch out for other products containing caffeine, even some medications (especially cold remedies) contain caffeine.

Be aware of how much caffeine you consume as it needs time to process. It can take several hours to process the caffeine from just half a cup of coffee. If you decide to stop caffeine, at first you may experience withdrawal effects such as headaches.

Alcohol makes you feel sleepier, however it disrupts the quality of your sleep. Alcohol before bed may help you relax and fall asleep, however it also suppresses REM sleep (dreaming sleep). This can lead to a rebound of REM later in the night, with vivid dreams and fragmented sleep. It takes your body approximately one hour to process each standard size drink you consume. Try to get your blood alcohol level to zero before going to bed.

MOOD AND SLEEP

If you think you may be affected by a mood disorder, please consult your doctor for advice

The way we feel can have an impact on sleep, especially getting to sleep and being able to get back to sleep after a disturbance. Worry, anxiety and depression are common complaints with ageing. On the other hand, sleep deprivation and daytime sleepiness can lead to depression and anxiety due to being less able to cope and live life normally when sleepy. Treatments for mood disorders can also impact sleep (some positively but others negatively), so it is important to recognise when anxiety or depression rears its head and address it with regards your sleeping as well as waking life.

Worry and anxiety

Despite feeling tired at night, worry or anxiety can keep us from getting to sleep. For those with dementia this may be due to feelings of disorientation or not being prepared for sleep. On the other hand, supporters may experience anxiety due to feeling the need to be vigilant 24 hours a day or worrying about the safety of the person they care for.

BOX 9. Tips for relaxing worry at night:

- Set aside some time for relaxing activities before bed (such as a warm bath, reading a book, talking to friends)
- Allocate a time of day for worry, if thoughts of anxiety creep up on you, try to push them aside until that time
- If you wake in the night, focus on calmly going back to sleep rather worrying and 'trying' to get back to sleep
- If you simply cannot get back to sleep at night, try getting up and doing a relaxing activity for a little while before returning to bed
- Make sure if you do get up in the night you keep the environment dimly lit
- Acknowledge that short disruptions are only minor glitches in what could be a an overall good night's sleep

Depression

Unfortunately depression is more common with age, especially when living with a medical condition or supporting a loved one who is unwell. Some people find that feeling 'blue' simply creeps up on them and, like disrupted sleep, can become the norm. Difficulty falling asleep, staying asleep and waking too early can all be caused by depression. On the other hand poor sleep, especially insomnia has been identified as a key predictor of depression. Mood and sleep have a "chicken and egg" like relationship, so it is important to focus on getting the best sleep you can to minimise mood disruption.

Evening and Bedtime Routines

Having a routine before bedtime helps your brain and body to recognise when it is time for sleep. By making some activities a regular night time ritual your mind will more likely associate these activities with feeling tired and promote sleep onset. These activities could then be used in the night if you are having trouble returning to sleep after a disturbance (see Box 8 for some suggestions).

Sleep timing

It is important to get plenty of sleep, however do not force yourself to go to bed if you are not drowsy as you will be less able to fall asleep. If you cannot get to sleep after 20 minutes or so, try getting up and winding down again outside of the bedroom. Beware you do not fall asleep outside of the bedroom! If you have disrupted sleep, acknowledge that catch up sleep may be needed and make some time for this as soon as possible.

BOX 8. Suggestions for evening and bedtime routines:

- A warm shower or bath an hour before bed
- Spend the hour or two before bed in dimmer light
- Try an non-caffeinated warm drink to soothe (e.g. camomile)
- After you are prepared for sleep (e.g. changed into bed clothes and brushed teeth), spend a few minutes relaxing before going to bed
- Dressing in nightclothes etc gives a non-verbal cue that its bedtime for those with dementia. Avoid placing the next day's clothes out as this could cue morning routines during the night
- In bed, rather than thinking about your day or what you need to do, try and focus on your body and breathing to relax
- Sleeping medications should be used conservatively (more details on page 19)

Although life may seem to be too busy to incorporate these sleep rituals, it is important to allow yourself even just 15 minutes to relax before bed. Likewise, try to avoid any exciting or stressful activities prior to bed.

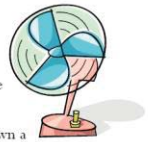
In bed…

It is important to only go to bed when you're tired. If you spend too much time awake in bed, your body and mind begin to associate the bed with being alert and awake which will make it harder to sleep. Avoid any wake time activities (including your relaxing activities) whilst in bed, keeping it as a sleep haven. If you are a clock watcher, try turning the face of the clock away from you as glancing at the time continuously can contribute to worry about not sleeping, making it perpetually hard to get to sleep.

Sleep Environment

It is important to consider the sleeping environment in order to optimise your sleep. Making the bedroom a sleep sanctuary cues the mind and body that this is the place mainly for sleeping. If you share your bed or bedroom consider whether you disturb each other in the night (through movement, trips to the toilet or snoring for example), or whether having one another nearby might have a positive effect on sleep. Likewise, if you share a bedroom with your pets, are they keeping you awake and restricting your space in the bed? Or are they a comfort at night?

Temperature



If the room is too hot or cold this can influence sleep. A hot bedroom can lead to more night awakenings, restlessness and more difficulty getting to sleep. On the other hand, a bedroom which is too cold can also be uncomfortable, leading to disturbed sleep.

Your body temperature is also important to consider. You tend to cool down a little prior to sleep. A hot bath may help to relax you in the evening and the cooling down afterwards can help you to feel suitably tired. However it is best to avoid hot baths immediately prior to bed. Vigorous exercise before bed is likely to raise your body temperature making it more difficult to get to sleep.

Light

Having a dark bedroom can help you to sleep, as light has an activating effect on the brain. If street lights are a problem, you might consider heavier curtains for the bedroom for night time.

For safety reasons you may like to keep some lights on overnight. Consider dim lighting for the bathroom or halls, using night lights near doors, or having a torch at the bedside. This way you don't have to subject yourself or those you live with to bright lights if you get up in the night, which in turn may affect everyone's ability to get back to sleep.

Noise

Noise at night can hinder getting to sleep and cause awakenings. Sudden noises such as traffic, snoring and noisy neighbours can all be problematic and difficult to control. You may consider using ear plugs or moving your bedroom to a quieter part of the house. Another option is covering up the unpleasant noise, for example some people find listening to relaxing music, or sounds of ocean waves helps provide a steady sound to mask the neighbourhood racket.

For safety or comfort reasons, you may want to hear your partner at night. If you are in separate rooms, you might consider using a bell or alarm system if assistance is required. That way those supporting may not need to listen out for every movement.

Safety

When considering adapting the environment, bear in mind the safety issues in your home. Make the house a safe place for those with dementia to wander in at night. Below are some examples of things you may consider trying at night (depending on your situation):

- Dim lighting/night lights in the halls, bathroom or other places which may be accessed during the night
- Blocking or securing stairways
- Turning the gas off
- Locking windows and external doors
- Installing alarms on external doors
- Locking away dangerous implements around the house
- A personal bell or alarm

Respite for those supporting people with dementia is also important. Whether daytime or night, giving supporters some relief of their care-giving responsibilities will allow time to catch up on much needed sleep.

SLEEP AND MEDICATIONS

Medicines to help you sleep

Sleeping pills should be used with caution. Although they can be useful for short periods and in specific circumstances (for example, if suffering from insomnia after bereavement), they should not be relied on to get to sleep on a routine basis.

Sedating medicines taken at night can cause drowsiness or feeling groggy throughout the night and sometimes on into the morning. This has implications for safety (for example falls or driving), performance in the morning (due to the lasting effects of the medication), and ability to cope during the day.

Another important note for taking sleep medications is that they are masking the true reasons for not being able to get good sleep. Sleeping medications should not be taken for long periods (more than a couple of weeks) as it is easy to build up a tolerance and become dependent on them. Therefore it is always important to independently deal with the underlying cause (for example feelings of worry, poor sleep timing or noise) for long term improvements in sleep.

If you are thinking about using sleeping pills or are concerned about the side effects of any medications you are taking, contact your doctor for advice.

Other medications

Many prescribed medications can affect sleep. It is important to read the side effects of your medications and consider whether your sleep has deteriorated since taking the medicine.

For example drugs for asthma, respiratory or heart disease, arthritis, and depression, as well as medications for dementia can have a negative impact on sleep. Such side-effects can include insomnia, drowsiness, or nightmares.

It is worth recognising the effects medicines may have on you. If you do suffer from sleep disruption as a side effect from your medicine, it may be worth consulting your doctor. Changing the time you take medicines may make a difference, or in some cases there may be an alternative for you to try.

BOX 9. Medicine considerations:

- Use sleeping pills with caution – they are best for temporary times of disturbed sleep rather than all of the time
- Be aware of the side effects of sleeping pills and other sedating medications, especially if you need to drive or need to be focused the next day
- If you are having sleeping problems it may be worth considering the side effects of any other medications you are taking
- Although you may not be able to change your medications, it is useful to be aware of their impact on sleep and consider the time of day they are taken

CONCLUDING THOUGHTS

Congratulations! Through reading into the subject of sleep and learning about some key tips, you've taken some of the first steps to improving your own sleep. I hope you have found this booklet useful and you are feeling motivated to focus on your sleep. It may be that you are already doing a lot of what has been mentioned in this booklet. Remember, if you are taking on new routines or changing the environment, it may take some time before you see the changes in your slepe or your partners sleep, so please do persevere. Sleep is vital for functioning during the day, especially as we get older, so the suggestions outlined here may well make a difference to your life with regards to mood, memory, vitality and health.

Use the reference boxes throughout the handbook as a quick reminder of the main points or tips to improve your sleep. For further information consider the reading and internet sites on the next page or contact a member of the research team.

Good luck and pleasant dreams!

FURTHER READING

Books

- The promise of sleep. Dement, C. & Vaughn C. (1999). New York: Delacorte
- Geriatric sleep medicine. Eds. Avidan, A.Y. & Alessi, C. (2008). New York: Informa Healthcare USA
- All I want is a good night's sleep. Ancoli-Israel, S (1996). St Louis: Mosby
- Sleep in the 24-hour society. Gander, P. H. (2003). Lower Hutt: The Open Polytechnic of New Zealand.

The internet

- The National Sleep Foundation has a great website concerning sleep and health for all ages as well as sleep disorders and advice: http://www.sleepfoundation.org/
- Sleep Web is a website providing information on sleep disorders and treatments. http://www.sleepweb.com/
- The Sleep Apnoea Association of New Zealand has a website providing more information on OSA : http://www.sleepapnoeanz.org.nz (phone 094821939)
- "Sleep: A critical but overlooked aspect of dementia" is a website developed by the University of Alberta with the Canadian Dementia Knowledge Translation Network which aims to inform supporters of people with dementia about sleep and what can be done to improve sleep http://www.wix.com/carybrown/sleep-dementia#!
- The American Alzheimer's Association and Alzheimer's Australia have websites with information on sleep: http://www.alzheimers.org.au/services/sleeping.aspx http://www.alz.org/alzheimers_disease_10429.asp

CONTACTS

If you would like further information on the content of this booklet or would like a pamphlet concerning a particular sleep disorder please contact the Sleep/Wake Research Centre:

Research team at the Sleep/Wake Research Centre
Rosie Gibson, MSc
FreePhone: 0800 TBC
Phone: 04 3800635
Email: r.gibson@massey.ac.nz

Professor Philippa Gander, PhD
Phone: 04 3800633
Email: p.h.gander@massey.ac.nz

Alzheimers Wellington is a registered non-profit charity providing support, information and education to anyone in the Wellington region who is affected by dementia:

Alzheimer's Wellington Office
Phone: 04 939 0133
Email: wellington@ alzheimers . org . nz
Address: 55 Hutt Road
Petone
Wellington
5012

Alzheimer's New Zealand National Office
Phone: 04 381 2362
Email: nationaloffice@alzheimers.org.nz
Address: Level 3
Adelphi Finance House
15 Courtenay Place
Wellington

9 Feedback Questionnaire

'SLEEP–WAKE' research centre
'MOE TIKA • MOE PAI'

Sleep of People With Dementia and Those Who Live With Them

Feedback Questionnaire

Thank you very much for taking part in the sleep and dementia study. Your sleep data has been added to our database for analysis. In the meantime I would really appreciate your feedback on the study. Below are some questions and space for you to comment on how you think the study went and what could be done better.

The content of this form is confidential so please don't hesitate to be critical (your comments will help us to redesign this type of study in the future).

Once complete, could you please post the form back to me in the enclosed pre-paid envelope?

---

1. **Do you feel that the information provided to you prior to the study gave you a full picture of what the study entailed?**

   Yes ☐ No ☐

   If you answered no – what information would you recommend including?

2. **Were you satisfied with the answers from the researcher to any queries you had concerning the study?**

   Yes ☐ No ☐ I didn't have any queries ☐

3. **How did you find using the wrist worn sleep monitor?**

   Easy ☐ OK ☐ Difficult ☐

   Any comments about using the monitor?

4. **How did you find completing the sleep diary?**

   Easy ☐ OK ☐ Difficult ☐

   Any comments about the sleep diary?

Continued over page

5. **How did you find completing the questionnaires?**

   Easy ☐ OK ☐ Difficult ☐

   Any comments about the Questionnaires?

6. **Did you complete the 6 week study?**

   Yes ☐ No ☐

   If you did answered No why were you unable to complete the study?

   - Became unwell ☐
   - Moved into respite or permanent care ☐
   - Took too much time/too difficult ☐
   - Didn't realise what the study entailed ☐
   - Dissatisfied with the way the study was being run ☐
   - Other ☐

The following questions are about the sleep therapies you may have tried

How did you find using:	Easy	OK	Difficult	N/A	Any Comments?
7. The light box?					
8. The exercise regime?					
9. The sleep information package?					

10. **Would you recommend these sleep therapies to others?**

    Yes ☐ No ☐ Maybe ☐

    Why/ why not?

Any other comments about the study?

Many thanks and best wishes,

Rosie Gibson
Free phone: 0800 SNOOZE (766693), Phone: 04 3800635, Email: r.gibson@massey.ac.nz

Improving Sleep of People with Dementia and Those who Live with Them Feedback Questionnaire V. 2 09.09.2011

10 Study 4: Information for Participants

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

Rosie Gibson
Sleep/Wake Research Centre
Phone: 04 3800635
Free phone: 0800 SNOOZE (766693)
Email: r.gibson@massey.ac.nz

March 2012

IMPROVING SLEEP OF PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM

Dear ,

Thank you for your interest in participating in the sleep and dementia study. We are trying to improve understanding of the changes to sleep that come with dementia, and to find better ways to manage dementia-related sleep problems for people living in the community.

An information sheet and consent forms are enclosed.

The study is evaluating non-drug techniques to improve the sleep and daytime functioning of people with dementia and their partners who provide care. It will involve monitoring your sleep, completing some questionnaires, and a 5-week trial of three simple therapies: bright light, exercise and sleep education.

If you and your partner agree to take part, please contact me on the phone number or address below and we can organise a time for me to visit you at home to begin the study.

If you have any questions before enrolling or during the study please do not hesitate to contact me on free phone 0800 SNOOZE (76693), or 04 3800635, or by email (r.gibson@massey.ac.nz).

Thank you for your interest in this study. I look forward to meeting you.

Yours sincerely,

Rosie Gibson
Principal Investigator

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

INFORMATION SHEET

IMPROVING SLEEP OF PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM

Thank you for your interest in this study. This information is provided to help you decide whether or not you wish to participate.

WHAT IS THE STUDY ABOUT

Sleep disturbances can be a major problem for people with dementia that can also affect the sleep of partners who support them. One of the factors that results in sleep becoming fragmented for people with dementia is that the circadian body clock (a pacemaker in the brain) becomes progressively weaker in its control of the sleep/wake cycle. (The circadian body clock normally enables us to sleep predominantly at night and be awake and alert during the day, except around afternoon nap time).

The circadian body clock is sensitive to light (through a special input from the eyes that is separate from vision) and it can also be reset by exercise at different times of day. This has lead to the idea that timed exposure to light and timed bouts of exercise could be used to strengthen the output of the circadian body clock. This might help consolidate sleep at night and reduce daytime napping for people with dementia.

This approach has been shown to be helpful for people with more advanced stages of dementia who are living in residential care facilities. In this research, we are interested in finding out whether timed exposure to light and timed bouts of exercise can improve the sleep of people with dementia who are still living at home. If this intervention is successful, it may also have benefits for the sleep and quality of life of supporting family members as well.

WHO IS BEING ASKED TO PARTICIPATE

We are looking for 30 couples to participate in this research, with each couple including one person who has dementia and is living at home, and their partner who supports them. This is a small pilot study so we are only seeking people who live in the greater Wellington Region (including Hutt Valley and Kapiti Coast) and the research would take place in your home.

WHAT IS INVOLVED IF YOU PARTICIPATE

The study takes 6 weeks in total but it all takes place in your home, involves routine tasks, and you would be able to withdraw at anytime should you feel you need to. At the beginning and end of your participation, you would be asked to complete questionnaires. During the first week and the last week you would both have your sleep monitored. This involves wearing a small watch-like device on your wrist that measures movement (an actiwatch – shown in the photo below), and keeping a daily diary of when you sleep. The methods and measures are described in more detail below (a brief 'protocol at a glance' is included on the last page).

- At the beginning of your participation, a member of the research team would come to your home at a suitable time to discuss the project and answer any questions you both may have. They would then give you the questionnaires and show you how to use the actiwatch and complete the sleep diary. If you wish, they can also help you work through the questionnaires.
- During the first week we will ask both of you to wear the actiwatches every day and complete daily sleep diaries.
- At the end of the first week, a member of the research team will visit you again to collect your questionnaires, actiwatches, and sleep diaries.
- For the next 5 weeks you would then be asked to try and add two activities to the daily routine of the person with dementia as often as possible (preferably every day) They are:
    1. **Light:** This involves routine bright light exposure for 30-60 minutes between 9-11 am each morning. You will be lent a light box to try which those with dementia simply sit in front of (sedentary activities such as morning tea, talking on the phone, reading etc can all take place while sitting in front of the light box.) Alternatively, they spend an equivalent amount of time outside in the daylight if that is more convenient.
    2. **Physical Activity:** This involves approximately 30 minutes of exercise around the middle of the day (about 11am-2pm). This could be walking, or any other form of exercise you usually undertake. You will also be lent a DVD of seated exercises which are a good option for rainy days or those who are less mobile.
- You will be given diaries to document when the person with dementia does these activities, as well as when they sleep.
- You will also be given an information booklet on sleep changes with ageing and dementia, with tips for improving your sleep.
- During week 6 of the intervention (your last week in the study) you would both be asked to wear the actiwatches again and to complete the sleep diaries, as well as filling out the final questionnaires. This will enable us to see if anything has changes since the first week of the study.

Equipment can be delivered and collected by the research staff or by courier if more convenient. If you have regular commitments such as work, club, or respite you can still take part in the study.

The light and exercise activities should be used on as many days as possible.

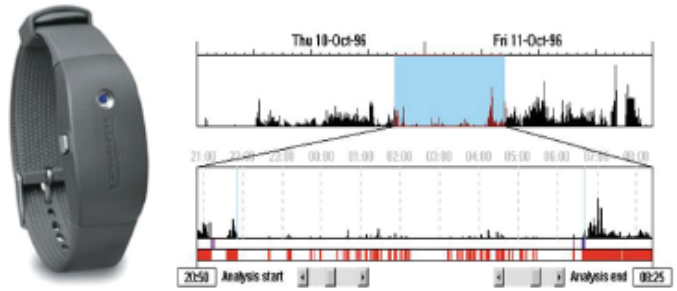
Questionnaires

You will both be given a questionnaire to complete (this should take about an hour). You can work on these together, and a member of the research team can help you work through them if you like.

The questionnaires ask each of you about your age, health, medical history, and usual sleep routine. They also include a standard set of questions to assess mental status and behaviours associated with dementia, and two different sets of standard questions about sleep and sleep quality, mood and lifestyle (one for the person with dementia and the other for their carer). In addition, there is a standard set of questions for the care-giving partner about their role.

Sleep monitoring

The photo below shows an actiwatch, which you would wear on your wrist during the first and last weeks of the study. The actiwatch monitors movement of your wrist, and every minute it records an activity count on an internal memory chip. The photo shows an example of the minute-by-minute activity counts (each vertical black bar represents one minute). The section of the recording that is expanded shows when this person indicated in their diary that they were trying to sleep. The actiwatch also records light exposure. At the end of each week of recording, your activity counts would be transmitted from the actiwatch to a computer and a special program used to decide when you were actually asleep and awake across the week, and how restless you were during sleep. It will also give details on the amount of light exposure you have had throughout the week.



Activities

During weeks 2-6 of your participation, you would be asked to introduce the light and physical activities into your daily routine When the researcher visits you at the beginning of the study, they will work through with both of you what is involved for each activity. While you are trying out the activities (weeks 2-6) you will be asked to note down each day in a diary when you use them. A member of the research team will contact you once a week to check if you need any help and answer any questions that come up. You can also call a free phone number at anytime.

1. Bright light

To boost the cycle of their circadian body clock (which is sensitive to bright light), the person with dementia would be asked to sit in front of the light box for 30-60 minutes each day, between 9-11 am. **Alternatively, spending 30-60 minutes outdoors during this time would have a similar effect.**

The photo to the right shows a light box being used. You will be given an instruction leaflet to go with it.

Frequently asked questions about light therapy:

Do I have to take off my glasses to use the light box?
No, it will work with or without glasses, but should not be used with sunglasses.

Are there any major side effects?
Light therapy is safe when used according to the recommendations. Some people report temporary headaches or nausea, but this usually goes away if you sit a bit further away from the light. You cannot get sunburn because the light box does not produce ultra-violet light, which is the part of the spectrum that causes sunburn.

Can it damage my eyes?
Ultra-violet light is also the part of the spectrum that carries the greatest risk of causing damage to the eyes, and the light box does not produce ultra-violet light.

Is light therapy suitable for everyone?
The use of light therapy for this study may not be suitable for people who have had cataract surgery, or who have some specific eye or skin conditions (such as retinal dystrophies, macular degeneration, porphyria, lupus erythematodes, chronic actinic dermatitis and solar urticaria). It is also not recommended for people who have bipolar disorder. Light therapy should not be used in conjunction with photosensitizing drugs which you might be prescribed for some mental health conditions, heart arrhythmias, rheumatism, or malaria. **Your doctor will be asked to notify us if they feel that using an artificial light box is not appropriate for you, in which case natural light can be used instead.**

Can I use light therapy in the evening?
If you use light therapy too close to your normal bedtime then it may be difficult to fall asleep. This is one reason why the study asks that you use it in the morning.

2. Physical Exercise

Timed bouts of exercise can boost the cycle of the circadian body clock. In addition, regular low-impact aerobic exercise has been shown to improve the amount and quality of sleep, as well as making people feel more alert during the day. The best time of day to do exercise to get these benefits is from mid-morning to early afternoon. Exercising too close to bedtime may make it harder to get to sleep. For this study, you would be asked to do 30-40 minutes of exercise between 11am and 2pm, as often as possible. This could be walking or any other type of exercise you like. The key thing is doing exercise regularly during this time period, not the particular type of exercise you do. If you feel unable to do the exercise component of the study (for example due to health conditions) you can still take part just using the light.

3. Sleep Education

There are a number of strategies that have been tested and shown to improve sleep. These tips are sometimes called 'sleep hygiene education'. The key principle is that activities during the day, and habits around sleep, can have an influence on sleep at night.

As part of the study, you would be given a booklet that describes the basics of sleep and how it changes with ageing and dementia, and some tested tips that can improve sleep (for example, not drinking coffee later in the day, trying to have a regular routine around going to bed, and not having the television in the bedroom). Not everything works for everyone, so you may need to try a few different things.

WILL MY GP BE TOLD I AM IN THE STUDY?
Permission will be sought from you to inform your GP that you are participating in this research. This authorises the researcher to notify your GP about the sleep intervention and study requirements, as well as permission to notify them of any health-related issues that are identified through taking part in the study. This also gives the GP the authority to notify us if for any reason they feel the light box is not suitable for you.

WHAT HAPPENS TO THE DATA
Only research team members from the Sleep/Wake Research Centre and individual GP's will know who participates in the study. Each participant will be given a unique study ID number that will be used to identify their data. Once your participation is complete, no record will be kept that could link you to your data, and only grouped data will be reported. No material that could personally identify you will be used in any reports on the study.

You can choose to have an annotated printout of your sleep/wake patterns as recorded by the actiwatch and the sleep diary, and have the opportunity to discuss it with a member of the research team. In addition, all participating couples will receive a short brochure describing the study findings once the study is complete.

Rosemary Gibson will analyse the data as part of the research for her Doctoral Thesis, and the findings will also be published in a scientific paper. They will help us understand how workable and effective this approach is for improving sleep for people with dementia living at home and for family supporters. If this pilot study is successful, we plan to undertake a larger trial with a more diverse group of people, and to develop the protocols and procedures so the Alzheimers New Zealand community workers could make the therapies available.

YOUR RIGHTS
You are under no obligation to accept this invitation. If you decide to participate you have the right to:

- decline to answer any particular question;
- withdraw from the study at any time;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Note. On completing the study you will be expected to return the equipment to the Sleep/Wake Research Centre. If the trial is successful, light therapy and information packs will be made available through Alzheimers Wellington.

ETHICS
This study has received ethical approval from the Health and Disabilities Ethics Committee (approval number CEN/11/02/01). If you have any queries or concerns regarding your rights as a participant in this study, you may wish to contact an independent health and disability advocate: Free phone: 0800 555 050, Free fax: 0800 2 SUPPORT (0800 2787 7678), Email: advocacy@hdc.org.nz'

COMPENSATION
This is a low risk study which all takes part in the home. Participation is voluntary. The researcher will bring you morning or afternoon tea at the beginning and end of your study. You will also be sent your sleep study print outs when you have finished taking part.

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation, and Compensation Act 2001. ACC cover is not automatic, and your case will need to be assessed by ACC according to the provisions of the Injury Prevention, Rehabilitation, and Compensation Act 2001. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors, such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses, and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

If you have any questions about ACC, contact your nearest ACC office or the research team. You are also advised to check whether participation in this study would affect any indemnity cover you have or are considering, such as medical insurance, life insurance and superannuation.

WHAT DO I DO NOW?
If you both choose to participate after reading this information sheet, please complete the attached consent form and send this back in the pre-paid envelope. If you prefer, the researcher can organise to visit your home to brief you on the study and record your consent on a tape recorder. Only the names and contact details of those who agree to participate will be retained.

Thank you for taking the time to consider being involved with this important research. Please feel free to contact the research team if you have any questions about the study.

CONTACTS

Principal Investigator:
Rosie Gibson, MSc
PhD Candidate
Sleep/ Wake Research Centre
Massey University, Private Bag 756,
wellington
Free phone: 0800 SNOOZE (766693)
Phone: 04 3800635
Email: r.gibson@massey.ac.nz

Research supervisor:
Professor Philippa Gander PhD, FRSNZ
Director, Sleep/ Wake Research Centre
Massey University, Private Bag 756,
wellington
Ph: 04 3800633
Email: p.h.gander@massey.ac.nz

Improving Sleep of People with Dementia and Those who Live with Them Information sheet Version 5 11.11.2011

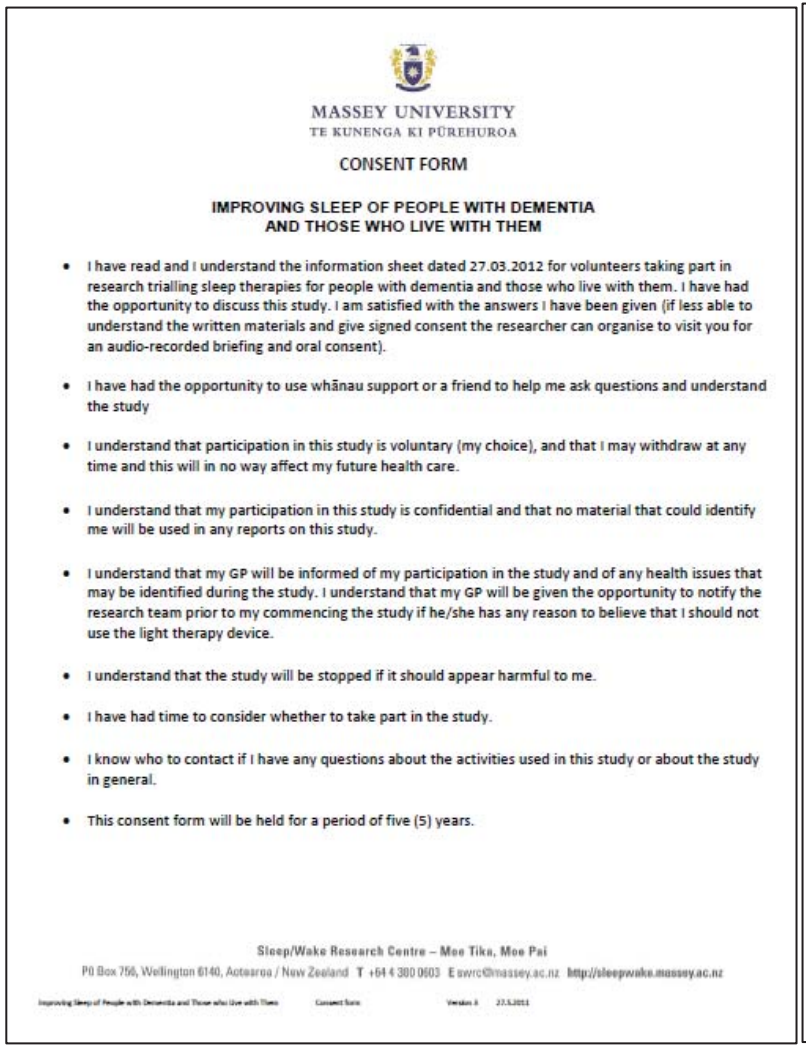
Sleep research protocol at a glance

Week 1	➢ **Questionnaires to complete** ➢ **Both wear the wrist sleep monitor and complete a sleep diary**
Weeks 2-6	➢ **Trial the recommended light and exercise activities for person with dementia** ➢ **Keep a diary of activities and sleep of person with dementia**
Week 6	➢ **Continue using light and exercise activities** ➢ **Both wear sleep monitor and complete a sleep diary** ➢ **Complete questionnaires again**

During the study
If you become unwell, change medications which affect your sleep, have any questions, or wish to withdraw please contact me:

Rosie Gibson, MSc
Principal investigator/PhD Candidate
Sleep/ Wake Research Centre
Massey University, Private Bag 756, wellington
Free phone: 0800 SNOOZE (766693)
Phone: 04 3800635
Email: r.gibson@massey.ac.nz

11 Study 4: Consent Forms



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CONSENT FORM

IMPROVING SLEEP OF PEOPLE WITH DEMENTIA AND THOSE WHO LIVE WITH THEM

- I have read and I understand the information sheet dated 27.03.2012 for volunteers taking part in research trialling sleep therapies for people with dementia and those who live with them. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given (if less able to understand the written materials and give signed consent the researcher can organise to visit you for an audio-recorded briefing and oral consent).
- I have had the opportunity to use whānau support or a friend to help me ask questions and understand the study
- I understand that participation in this study is voluntary (my choice), and that I may withdraw at any time and this will in no way affect my future health care.
- I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.
- I understand that my GP will be informed of my participation in the study and of any health issues that may be identified during the study. I understand that my GP will be given the opportunity to notify the research team prior to my commencing the study if he/she has any reason to believe that I should not use the light therapy device.
- I understand that the study will be stopped if it should appear harmful to me.
- I have had time to consider whether to take part in the study.
- I know who to contact if I have any questions about the activities used in this study or about the study in general.
- This consent form will be held for a period of five (5) years.

Sleep/Wake Research Centre – Moe Tika, Moe Pai
PO Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

Those who have dementia:

I (*full name*) have had the opportunity to discuss this project with my partner and hereby consent to take part in this study.

Note: If less able to understand the written materials and give signed consent please contact the researcher for documents to give consent by statement of partner, alternatively the researcher can organise to visit you for an audio-recorded briefing and oral consent.

Date: _____

Signature: _____

I wish to receive a copy of my sleep data on completion of my trial. Yes ☐ No ☐

I wish to receive a pamphlet summarising the results at the end of the study (approx June 2012). Yes ☐ No ☐

I agree to my GP or other current provider being informed of my Participation in this study/ be involved assessing my suitability for using the light therapy device/the results of my participation in this study. Yes ☐ No ☐

My GP's name and contact details are:

.....

.....

Please use the space below to list the medications you currently take, this is so we can confirm your suitability for light therapy prior to the study beginning.

Medication Name	Dose

Please turn over

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STATEMENT BY PARTNER

Lay title:	Improving sleep of people with dementia and those who live with them
Principal investigator:	Rosemary Gibson
Participant's name:	

I have read and I understand the information sheet dated 27.03.2012 for people taking part in the study designed to improve the sleep of people with dementia and those caring for them. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

I believe that (participant's name) would have chosen and consented to participate in this study if he/she had been able to understand the information that I have received and understood.

I understand that taking part in this study is voluntary and that my family member may withdraw from the study at any time if he/she wishes.

I understand that his/her participation in this study is confidential and that no material which could identify him/her will be used in any reports on this study.

I understand that his/her GP will be informed of their participation in the study and of any health issues that may be identified during the study.

I understand that the study will be stopped if it should appear to be harmful.

I know whom to contact if my family member has any side effects to the study or if anything occurs which I think he/she would consider a reason to withdraw from the study.

This study has been given ethical approval by the Central Health and Disability Ethics Committee. This means that the Committee may check at any time that the study is following appropriate ethical procedures.

I/my family member would like a copy of the results of the study. ☐ Yes ☐ No

I agree to my family member's GP or other current provider being informed of their participation in this study/the results of their participation in this study. ☐ Yes ☐ No

GP's name and contact details:

.....

.....

Sleep/Wake Research Centre – Mae Tika, Moe Pai
PO Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 9003 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

Please use the space below to list the medications your family member takes, this is so we can confirm their suitability for light therapy prior to the study beginning.

Medication Name	Dose

Signed:	Date:
Printed name:	
Relationship to participant:	
Address for results:	

STATEMENT BY PRINCIPAL INVESTIGATOR

I *(name of investigator)* declare that this study is in the potential health interest of the group of patients of which *(name of participant)* is a member and that participation in this study is not adverse to *(name of participant)*'s interests.

Signed: Date:
(Principal Investigator)

12 Example Letters to GP

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

R.Gibson
SLEEP/WAKE RESEARCH CENTRE
Massey University
Wellington
Private Bag 756
Ph: 04 3800635
r.gibson@massey.ac.nz

June 2012

Dear Dr ....

RE: Research participants
'Improving sleep of people with dementia and those who live with them'

Your patient, ... and their partner have agreed to take part in a research project. They were recruited to this project through membership of ..... This research is being run by the Sleep/Wake Research Centre, Massey University, is supported by Alzheimers Wellington, and was reviewed by the Central Regional Ethics Committee (number CEN/10/EXP/33).

The aim of the project is to trial non-pharmacological interventions to improve sleep of people with dementia living in the community. We will also assess the sleep of the partners who act as a carer for those with dementia, and assess daytime functioning, behaviour and quality of life. This research project will not affect your patient's standard of care in any way. We will inform you of any abnormal results with regard to sleep or mood if identified during the protocol.

We will be monitoring sleep for one week before and at the end of the 5-week intervention trial. Sleep monitoring will include using an activity monitor as well as questionnaires and sleep diaries. The trial intervention will include timed daily exposure to bright light (either outdoor light or using an artificial bright light source) and timed daily physical exercise. We will also provide the participants with a booklet covering sleep education and advice.

Participants will have the choice to use a bright light therapy device (Day-Lights™, Uplift technologies incorporated) or natural light (i.e. going outdoors) for approximately 30 minutes per day. The light therapy device delivers a broad spectrum light of 10,000 lux (the equivalent of being outside on an overcast day) and contains no UV. The device is low risk, however there are some contraindications to bright light therapy for which we are screening participants as they enter the study. Participants (and/or carers) have been asked to notify us of any **pre-existing medical conditions of eyes or skin that can show photosensitized reactions to bright visible light, or if they are taking any medications which photosensitize their skin and/or retinal tissues.** In these instances the research study can continue, however light therapy will not use the artificial light source, only regular exposure to outdoor light.

Your patient is due to begin the research protocol on: *30th May 2012 (light therapy from the 8th June)*
The research team have deemed them suitable/ ~~unsuitable~~ for artificial light therapy on the basis of: .....

If for clinical or other reasons you disagree, can you please inform me prior to the start of the research protocol either by returning the attached slip, or by email or telephone, stating whether it is an eye condition, skin condition, a medication issue, or another issue?

If you have any queries in regards to the research please contact me using the above phone number or email address. Alternatively you may wish to discuss this study with Professor Tony Dowell (Department of Primary Health, 04 3855995) who is on the research team.

Yours sincerely,

Miss Rosemary Gibson (MSc, Principle Investigator)

Professor Philippa Gander (PhD, FRSNZ)

Professor Tony Dowell (FRNZCGP General Practice, MbChB))

MASSEY UNIVERSITY
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SLEEP/WAKE RESEARCH CENTRE
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Your patient is due to begin the research protocol on: *30th May 2012 (light therapy from the 8th June)*
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If for clinical or other reasons you disagree, can you please inform me prior to the start of the research protocol either by returning the attached slip, or by email or telephone, stating whether it is an eye condition, skin condition, a medication issue, or another issue?

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Sleep/Wake Research Centre – Moe Tika, Moe Pai
PO Box 756, Wellington 6140, Aotearoa / New Zealand T +64 4 380 0603 E swrc@massey.ac.nz http://sleepwake.massey.ac.nz

If you believe your patient should not use a bright light therapy device please return this slip to:

R. Gibson, Sleep/Wake Research Centre, Massey University, Wellington, Private Bag 756
Or alternatively email the information to r.gibson@massey.ac.nz
Or call 04 3800635

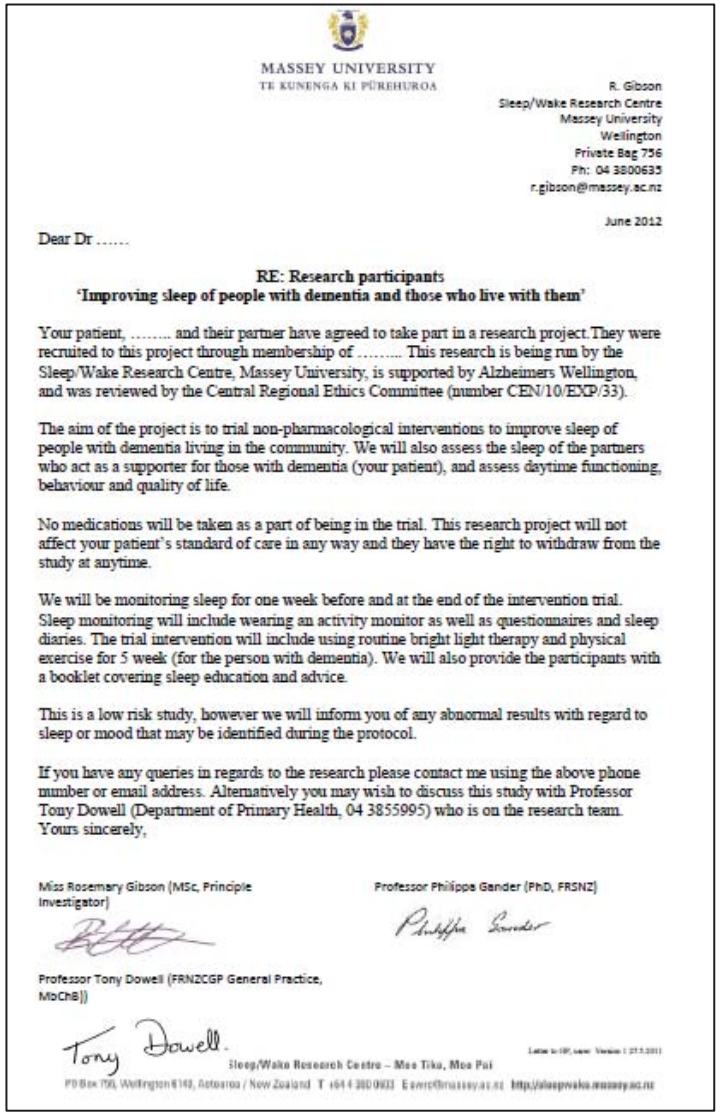
Patient.....(name)

Should not use light therapy device because:	Please tick
Pre-existing eye condition	
Pre-existing skin condition	
Medications	
Other	

Dr's Name (printed) .....

Dr's Signature.....

Letter to GP, PWD Version 1 27.5.2011



MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

R. Gibson
Sleep/Wake Research Centre
Massey University
Wellington
Private Bag 756
Ph: 04 3800635
r.gibson@massey.ac.nz

June 2012

Dear Dr .....

RE: Research participants
'Improving sleep of people with dementia and those who live with them'

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The aim of the project is to trial non-pharmacological interventions to improve sleep of people with dementia living in the community. We will also assess the sleep of the partners who act as a supporter for those with dementia (your patient), and assess daytime functioning, behaviour and quality of life.

No medications will be taken as a part of being in the trial. This research project will not affect your patient's standard of care in any way and they have the right to withdraw from the study at anytime.

We will be monitoring sleep for one week before and at the end of the intervention trial. Sleep monitoring will include wearing an activity monitor as well as questionnaires and sleep diaries. The trial intervention will include using routine bright light therapy and physical exercise for 5 week (for the person with dementia). We will also provide the participants with a booklet covering sleep education and advice.

This is a low risk study, however we will inform you of any abnormal results with regard to sleep or mood that may be identified during the protocol.

If you have any queries in regards to the research please contact me using the above phone number or email address. Alternatively you may wish to discuss this study with Professor Tony Dowell (Department of Primary Health, 04 3855995) who is on the research team.
Yours sincerely,

Miss Rosemary Gibson (MSc, Principle Investigator)

Professor Philippa Gander (PhD, FRSNZ)

Professor Tony Dowell (FRNZCGP General Practice, MbChB))

Tony Dowell.

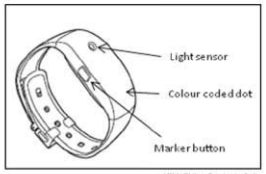
13 Participant Instructions: Sleep Monitoring

'SLEEP~WAKE'
research centre
'MOE TIKA~MOE PAI'

ACTIWATCH AND SLEEP DIARY GUIDE

Information about wearing the actiwatch

The watch you are about to put on contains an activity monitor called an actiwatch. The actiwatch is a small accelerometer with a memory chip and it records movement. The data from the actiwatch is analysed along with the information from the sleep log to determine when and how well you slept. The actiwatch also contains a light monitor which records the intensity of light exposure across the 24 hour day.



AW-2 (Philips Respironics Ltd)

1. Please wear your actiwatch 24 hours a day for the whole week so we can understand your sleep/wake patterns (there is a reminder not to remove it on the watch!).
2. It is important not to mix your actiwatches up - they are colour coded so you and your partner can tell them apart (blue for those with dementia and green for partners).
3. On the side of the actiwatch is a small button. When you push this button, a marker is inserted into the data. The button will not stop or start the watch, which will keep recording the entire time that you have it.
4. Fit the actiwatch around your non-dominant wrist. It should be attached reasonably firmly so that it does not move around your wrist but is comfortable.
5. Please (when possible) push the marker button when you intend to start sleeping (for example after turning the light out) and when you stop trying to sleep. This applies to day and night time sleeps.
6. The watch is water resistant (for up to 30 minutes), so you can keep it on during showers and short baths, but it should be removed when bathing for longer periods or swimming. Please remember to put it back on again after such activities.
7. If you take the watch off for any reason, then please note this in the sleep log. If you forget to put the watch back on at any stage, please put it on as soon as you remember. Do not worry if you accidentally missed some time – we are interested in as much data as possible, so simply note in the sleep log the time when you put the watch back on.
8. Please contact the research team in the unlikely event that you experience skin reddening or inflammation from the watch.
9. Please take care of this equipment. If it is lost or becomes damaged contact the research team as soon as possible for a replacement.

Information about sleep diaries

You have been issued with several weeks of sleep diaries. These are colour coded to differentiate which one belongs to which person (blue and green).

- For the supporters (blue) there are two one week diaries to be completed during the first and last weeks (week 1 and week 6) when wearing the actiwatch.
- For those with dementia (green) there are six one week diaries. Week 1's diary records sleep only, weeks 2-6 record sleep as well as use of the light therapy and physical exercise interventions.

For an example of how to complete them please see the second page of the diaries.

1. Sleep diaries are set out so that each line represents 24 hours, from midnight to midnight on one day.
2. Please write the date for each day in the space provided.
3. We are interested in **any** sleep that is 10 minutes or longer. It does not matter whether this is during the day or night.
4. Please place a mark on the line to indicate a time and then write underneath what the line relates to, with the time in hours and minutes. For the sleep only diaries note the following times:
   - *started trying to sleep* (SS);
   - *finished trying to sleep* (FS);
   - *daytime naps* (NAP);*and*
   - *times when the actiwatch was removed* (OFF) (There are abbreviations listed in the diary for each of these events).
5. On each occasion that you enter a time in the diary, we would also like you to push the marker button on the actiwatch (this backs up the diary data, so don't worry if you forget to press the button sometimes).
6. During the intervention weeks, please also note the times in the green diaries:
   - *using the light box* (LB);
   - being *exposed to natural light* (NL); or
   - *taking part in physical activity* (PA).
7. Each morning please note the quality of your sleep on the scale of 1 (extremely good) to 7 (extremely poor).
8. There is space for you or your partner to write any comments you have at the end of each week. We are particularly interested in anything that may have affected your sleep. This could include changes related to the advice from our booklet, as well as changes to medications, feeling unwell, any unusual, stressful or exciting events, or reasons for changes to your routine sleep times or places (such as having visitors to stay, or an early morning commitment).
9. As you finish each diary, please place it in your coloured file for collection at the end of the study.

We will contact you in a weekly basis to catch up with how you are doing with the diaries and wearing the actiwatches. In the meantime, please don't hesitate to contact me if you have any questions:

Rosie Gibson
Free phone: 0800 SNOOZE (766693)
Phone: 04 3800635
Email: r.gibson@massey.ac.nz

14 Participant Instructions: Sleep Intervention

'SLEEP–WAKE' research centre
'MOE TIKA–MOE PAI'

SLEEP INTERVENTION GUIDE

Light therapy information

The first part of the sleep intervention is light therapy. This involves being exposed to bright light at around the same time every day.
Over the 6-week trial we would like you to get into the routine of getting half an hour of morning light. This can be achieved through going outside into the sunshine, or using the Day-Light™ device.
The key to this therapy is the routine and regular timing - if the routine is maintained then regular light has a boosting effect on your internal clock which can help consolidate your sleep at night.

- Use your light therapy (either natural light or the Day-Light) at the same time each day. We recommended a morning slot of **30 minutes, between 9 -11am**. Pick the most convenient time for you and try to stick to it each day.
- If you also get sunlight other times of day that is fine, but it is important to create a routine of morning exposure.

❖ **Important: When you use the Day-Light or have your dose of outdoor natural light, please note the times in your diary.**

Using natural light

Using natural light to get your timed light exposure is a great way to get outside and enjoy the fresh air as well as the sunshine. Even on an overcast day the strength of the sun will have positive effects on your internal clock. Remember that if you are getting your bright light from the sun, you need to protect your skin a - *Slip* on clothing which covers your skin, *Slop* on the sunscreen, and *slap* on a hat (2011, Health Support Council, www.sunsmart.org.nz).

Using the Day- Light™

You may find it more convenient to get your bright light in the comfort of your own home. The Day-Light is designed to deliver the right amount of light to have therapeutic effects. During the trial you will be lent a Day-Light device to use. The researcher will demonstrate the Day-Light and set it up to the correct height and angle for you. This set up is standard and ensures that you receive the right amount of light. On the next page are some instructions for using the Day-Light.

Day-Light device (Uplift technologies Inc.)

Instructions for using the Day-Light

1. You need to sit 12 inches away from the light. If you sit too close it may hurt your eyes, too far away and you won't get the same therapeutic effects. There is a piece of string attached to your light box to help gauge your distance.
2. Use the light box on the highest setting. The lower setting is only an ambient light, not a therapeutic light source.
3. Avoid using the Day-Light device outside of the recommended time slots (9-11am) unless advised by the research team.
4. You should have your eyes open and face the Day-Light, however it is neither necessary or recommended to look directly into the light. You can keep your glasses on when using, but do not use sun glasses (ultra-violet rays are already filtered out).
5. Whilst using the light box you can take part in sedentary activities such as reading, eating or crafts, just make sure you keep your head raised in order for the light to get to your eyes.
6. Correct and Incorrect ways to use the Day-Light:

CORRECT POSITION
The light should be angled at 15 degrees and at a height that your eyes are in the centre. Sit 12 inches away, where there's space to read, eat etc.

INCORRECT POSITION
If you sit too far away (over 12 inches) you will lower your dosage of light, reducing its effects

INCORRECT POSITION
Do not sit too close to the Day-Light or stare into the light as this might hurt your eyes

INCORRECT POSITION
Although it is fine to read while using the Day-Light, with your head in this position the right amount of light cannot reach your eyes

(Adapted from the Day-Light User Guide, Uplift Technologies Inc)

Safety when using the Day-Light

- As with all electrical devices, do not use the Day-Light device in or near water.
- The Day-Light does not contain UV light, you will not get sunburnt through using it.
- Do not overuse the Day-Light, we recommend 30 minutes each day.
- Do not stare at the light.
- Uplift technologies issue the following disclaimer for using the Day-Light device:
  "The Day-Light is an innovative light supply system – not a medical device. We recommend that you consult a physician before undertaking a bright light therapy regime, especially if you suffer from a mood disorder such as depression, or are on prescription medications, have a history of eye conditions or are under a physician's care for any reason" (Day-Light User Guide, www.day-light.com).

Your GP has been informed that you are taking part in this study and asked to notify us of any pre-existing conditions or medications which may mean the Day-light is unsuitable for you. If in doubt, please contact them to discuss using the light, and remember that natural light has the same effect and can be used as an alternative.

Physical activity information

The second part of the intervention is incorporating regular physical activity into your routine. Like light, exercise can also boost the cycle of the circadian body clock and even low impact exercise can also improve the amount and quality of sleep, as well as making people feel more alert during the day.

The best time of day to do exercise to get sleep benefits is from mid-morning to early afternoon. Exercising too close to bedtime may make it harder to get to sleep.

For this study, we recommend **30-40 minutes of exercise during the window of 11am and 2pm**, as often as possible (preferably every day).

As with the light therapy, there are options for how you get this boost to the body clock. You could go outside for a walk or spend 30-40 minutes doing some yoga style stretches and movement indoors from a recording. Or, of course any other form of exercise that you already do (swimming or dancing for instance). The key thing is doing exercise regularly during the 11am-2pm time period, not the particular type of exercise you do.

The researcher will lend you a "Sit and be fit" (Netfit) DVD as an option for you to use during the study.

- **Important: When you do your physical activities, please remember to note the times in your diary.**

Examples of routine exercise

To get your timed physical activity you could go outside for a good 30-40 minute walk...

Or stay inside and exercises from the "sit and be fit" DVD.

Safety

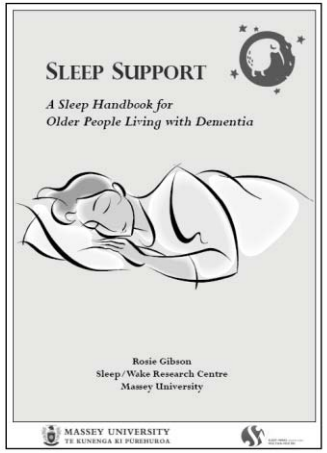
- It is recommended that you always stretch after exercise, so a leaflet with recommended stretches is also in your pack (Push Play Guide to Stretching (Sparc).
- If your fitness level is very low or you have any physical conditions which may affect your ability to perform physical activities (e.g. heart or joint trouble) you should consult your doctor before undertaking any new exercise programmes.

Sleep Support booklet

The third part of your intervention is the sleep support booklet. The first section of this handbook provides an overview of sleep and how it changes during ageing, for people with dementia and their supporters. There is also specific information on common sleep disorders. The second half of the handbook focuses on the factors which can affect sleep and offers advice and tips for activities, routines and changes you may wish to take to help improve the quality of you and your partner's sleep.

It is important to identify what may be causing the sleep problem. For instance it could be the environment, the physiological changes from dementia, or maybe medications you are taking that are at the root of the sleep problem. It is vital to identify the cause of the problem in order to decide which strategies would be best to use.

The booklet is designed for you to be able to dip in and out of so you don't have to read it all at once. It has tips and summaries highlighted throughout.



When incorporating the ideas and suggestions from the Sleep Support booklet, always consider safety within your home, for example making the bedroom dark enough to promote sleep but light enough to find the way to the bathroom.

- **If you try new strategies from the book, note them down in the weekly notes in the sleep diary.** This can help us see if they had any effect on your or your partner's sleep.

Good Luck! Please don't hesitate to contact me if you have any questions:

Rosie Gibson
Free phone: 0800 SNOOZE (766693)
Phone: 04 3800635
Email: r.gibson@massey.ac.nz

15 Fridge Magnet

16 Example of Feedback Letter to Participants

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Rosie Gibson
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June 2012

SLEEP STUDY

Dear .....,
Thank you for taking part in the sleep study. Your data has been added to my database for analysis and will contribute to the richness of this important research. I expect data collection to continue until Mid 2012, after which I will analyse all of the data together. These analyses will increase our understanding of the factors which affect sleep for older people and their families. We will also be able to assess the feasibility and effectiveness of the sleep interventions, through the actigraphy data combined with the diary and questionnaires. Once analysis is complete the results of the study will be made available to you directly from the Sleep/Wake Research Centre

In the meantime, enclosed are your printouts from the Actiwatch monitors that you wore as part of the study. These show the raw daily information which includes activity levels and light as well as times in bed (there is a legend at the bottom). From this data the computer estimates sleep variables which we will use for analysis alongside the questionnaires and diaries.

Looking at the reports you can see that ...gets a reasonable amount of light and activity in the mornings, but is often more rested in the afternoons. I can see you try keep quite routine night bedtimes and rise times (except for a few late nights at the weekend!) You can see on the reports the odd times with movements and/or the light going on which resemble short awakenings or toilet trips in the night, these may be contributing to some the daytime sleepiness/napping. I hope you find these printouts interesting.

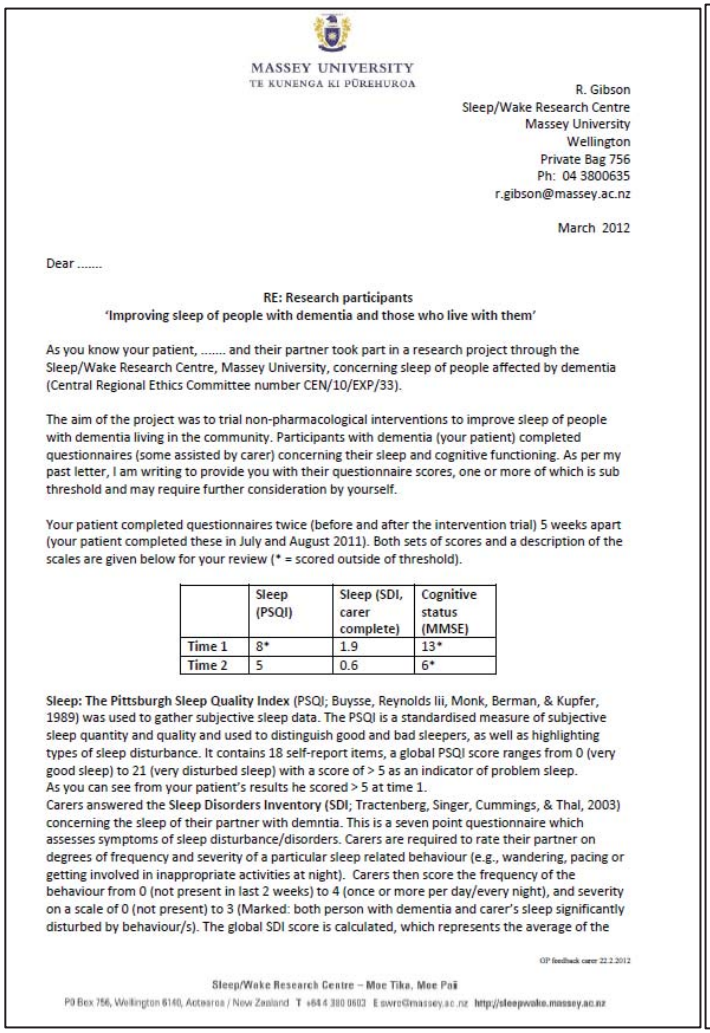
I have also enclosed a short, confidential feedback questionnaire which I would be very grateful if you could complete and send back to me in the enclosed envelope.
If you have any questions please do not hesitate to contact me.

Thank you very much for taking part in this study

Rosie Gibson,

Principal Investigator

17 Example of Feedback Letters to GP



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R. Gibson
Sleep/Wake Research Centre
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March 2012

Dear .....

RE: Research participants
'Improving sleep of people with dementia and those who live with them'

As you know your patient, ... and their partner took part in a research project through the Sleep/Wake Research Centre, Massey University, concerning sleep of people affected by dementia (Central Regional Ethics Committee number CEN/10/EXP/33).

The aim of the project was to trial non-pharmacological interventions to improve sleep of people with dementia living in the community. Participants with dementia (your patient) completed questionnaires (some assisted by carer) concerning their sleep and cognitive functioning. As per my past letter, I am writing to provide you with their questionnaire scores, one or more of which is sub threshold and may require further consideration by yourself.

Your patient completed questionnaires twice (before and after the intervention trial) 5 weeks apart (your patient completed these in July and August 2011). Both sets of scores and a description of the scales are given below for your review (* = scored outside of threshold).

	Sleep (PSQI)	Sleep (SDI, carer complete)	Cognitive status (MMSE)
Time 1	8*	1.9	13*
Time 2	5	0.6	6*

Sleep: The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds Iii, Monk, Berman, & Kupfer, 1989) was used to gather subjective sleep data. The PSQI is a standardised measure of subjective sleep quantity and quality and used to distinguish good and bad sleepers, as well as highlighting types of sleep disturbance. It contains 18 self-report items, a global PSQI score ranges from 0 (very good sleep) to 21 (very disturbed sleep) with a score of > 5 as an indicator of problem sleep.
As you can see from your patient's results he scored > 5 at time 1.
Carers answered the **Sleep Disorders Inventory (SDI**; Tractenberg, Singer, Cummings, & Thal, 2003) concerning the sleep of their partner with demntia. This is a seven point questionnaire which assesses symptoms of sleep disturbance/disorders. Carers are required to rate their partner on degrees of frequency and severity of a particular sleep related behaviour (e.g., wandering, pacing or getting involved in inappropriate activities at night). Carers then score the frequency of the behaviour from 0 (not present in last 2 weeks) to 4 (once or more per day/every night), and severity on a scale of 0 (not present) to 3 (Marked: both person with dementia and carer's sleep significantly disturbed by behaviour/s). The global SDI score is calculated, which represents the average of the

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seven frequency ratings multiplied by the average of the seven severity ratings (0-12) with higher scores indicating higher sleep disturbance.
As you can see from your patients results the SDI score at time 1 was raised, corroborating the heightened PSQI result. The heightened scores on these sleep measures related mostly to some trouble falling asleep and daytime sleepiness

Cognitive functioning: The Mini Mental Status Exam (MMSE, Folstein, Folstein, & McHugh, 1975) was used to assess the sample with regards to cognitive functioning. This is a brief standardised tool containing 11 questions across 5 categories: orientation, registration, attention and calculation, recall, and language. Points for each question are combined to provide a total score of cognitive functioning (on a scale of 0-30) with 21-24 points indicating mild cognitive impairment, 10-20 points indicating moderate cognitive impairment, and ≤9 points indicating severe impairment.
As you can see from your patient's results, he scored within the moderate range of cognitive functioning at time 1, and severe at time 2.

If you have any queries in regards to these scores or the research in general please contact me using the above phone number or email address. Alternatively you may wish to discuss this study with Professor Tony Dowell (Department of Primary Health, 04 3855995) who is on the research team.
Yours sincerely,

Miss Rosemary Gibson (MSc, Principle Investigator)

Professor Philippa Gander (PhD, FRSNZ)

Professor Tony Dowell (FRNZCGP General Practice, MbChB))

GP feedback carer 22.2.2012

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March 2012

Dear .....

RE: Research participants
'Improving sleep of people with dementia and those who live with them'

As you know your patient, ... and their partner took part in a research project through the Sleep/Wake Research Centre, Massey University, concerning sleep of people affected by dementia (Central Regional Ethics Committee number CEN/10/EXP/33).

The aim of the project was to trial non-pharmacological interventions to improve sleep of people with dementia living in the community. The sleep of the supporting partner (your patient) was also assessed, as was mood, cognitive functioning and coping. As per my past letter, I am writing to provide you with their questionnaire scores, one or more of which is sub threshold and may require further consideration by yourself.

Your patient completed questionnaires twice (before and after the intervention trial) 5 weeks apart (your patient completed these in July and August 2011). Both sets of scores and a description of the scales are given below for your review (* = scored outside of threshold).

	Sleep (PSQI)	Anxiety (HADS)	Depression (HADS)	Coping negative (COPE I)	Coping positive (COPE I)	Cognitive status (MMSE)
Time 1	9*	7	7	7	6*	30
Time 2	8*	9*	8*	8	5*	30

* = scored sub threshold

Sleep: The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds Iii, Monk, Berman, & Kupfer, 1989) was used to gather subjective sleep data. The PSQI is a standardised measure of subjective sleep quantity and quality and used to distinguish good and bad sleepers, as well as highlighting types of sleep disturbance. It contains 18 self-report items, a global PSQI score ranges from 0 (very good sleep) to 21 (very disturbed sleep) with a score of > 5 as an indicator of problem sleep.
As you can see from your patient's results she scored > 5 at both times 1 and 2, the heightened scores related mostly to waking in the night, getting up to use the bathroom and having trouble getting to sleep.

Anxiety and depression: The Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983) contains 14 items, 7 concerning anxiety and 7 concerning depression which participants rate. Their responses are scored (0-3) on the basis that a higher score equals more anxiety or depression. Scores are then added for each domain giving a global score of the likelihood of anxiety and depression for each participant (0-7 being normal, 8-10 borderline abnormal, and 11-21 being an abnormally high score).

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As you can see from your patient's scores she scored borderline abnormal for chances of anxiety and depression during her second round of questionnaires.

Carer burden: The COPE Index (Carers of Older People in Europe Index, CI; McKee, et al., 2003) is a 15 item questionnaire which requires carers to rate questions (never – always) with regards to how they currently would describe their feelings. The index is split into 6 questions concerning the negative impact of caring (e.g. Do you feel trapped in your role as a caregiver?) and 5 concerning the positive value of caring (e.g. Do you have a good relationship with the person you care for?).
There are no threshold scores, but for guidance, researcher use scores > 12 to indicate heightened Negative Impact (on a scale of 0-18) or < 12 to indicate reduced levels of Positive Value (on a scale of 15-0) (Roud, H., Keeling, S., & Sainsbury, R. ,2006).
As you can see from your patient's scores, she scored <12 on the positive aspects of caring scale at times 1 and 2.

Cognitive functioning: The Mini Mental Status Exam (MMSE, Folstein, Folstein, & McHugh, 1975) was used to assess the sample with regards to cognitive functioning. This is a brief standardised tool containing 11 questions across 5 categories: orientation, registration, attention and calculation, recall, and language. Points for each question are combined to provide a total score of cognitive functioning (on a scale of 0-30) with 21-24 points indicating mild cognitive impairment, 10-20 points indicating moderate cognitive impairment, and ≤9 points indicating sever impairment.
As you can see from your patient's results she scored above threshold for this domain.

If you have any queries in regards to these scores or the research in general please contact me using the above phone number or email address. Alternatively you may wish to discuss this study with Professor Tony Dowell (Department of Primary Health, 04 3855995) who is on the research team.
Yours sincerely,

Miss Rosemary Gibson (MSc, Principle Investigator)

Professor Philippa Gander (PhD, FRSNZ)

Professor Tony Dowell (FRNZCGP General Practice, MbChB))

Tony Dowell.

18 Software Procedures used to Define Actigraphy Variables

Figure 8.1 shows 48 hours of actigraphy data from one of the participants with dementia. Data shows (from left to right) an excluded period from when the Actiwatch was in transit, followed by some active/wake time, then a marked daytime rest interval (rest #1: 13:46-14:50). After another bout of afternoon activity is the main night time rest interval (rest #2) starting at 19:54 and finishing on the following day at 06:32. On the second day there is another daytime nap (rest #3: 15:05-15:38) followed by an excluded period due to the participant taking his Actiwatch off in the night. The rest intervals were noted in the diary, and the event marker was pushed at the corresponding time (blue arrows). There are also reductions in the participant's activity levels and light exposure around the bedtime, as indicated by breaks in the bottom red line and the absence of the yellow line. The event marker was also pushed at other times during the study however these times did not correspond to the diary or actigraphy data as rest intervals so were ignored (in other cases the event marker was not used at all and so this factor was not considered such a key source of bed and rise times).

These data from *Figure 8.1* gives the participant a night time rest duration of 638 minutes (rest #2, 10.6 hours). The bouts of activity and light exposure at the beginning and end of the rest interval indicate that there is some time when the participant is awake in bed. In the instance of rest #2, the sleep interval begins at 20:38 and ends at 6:22. Therefore the duration of the sleep interval within rest #2 is 584 minutes (9.7 hours).

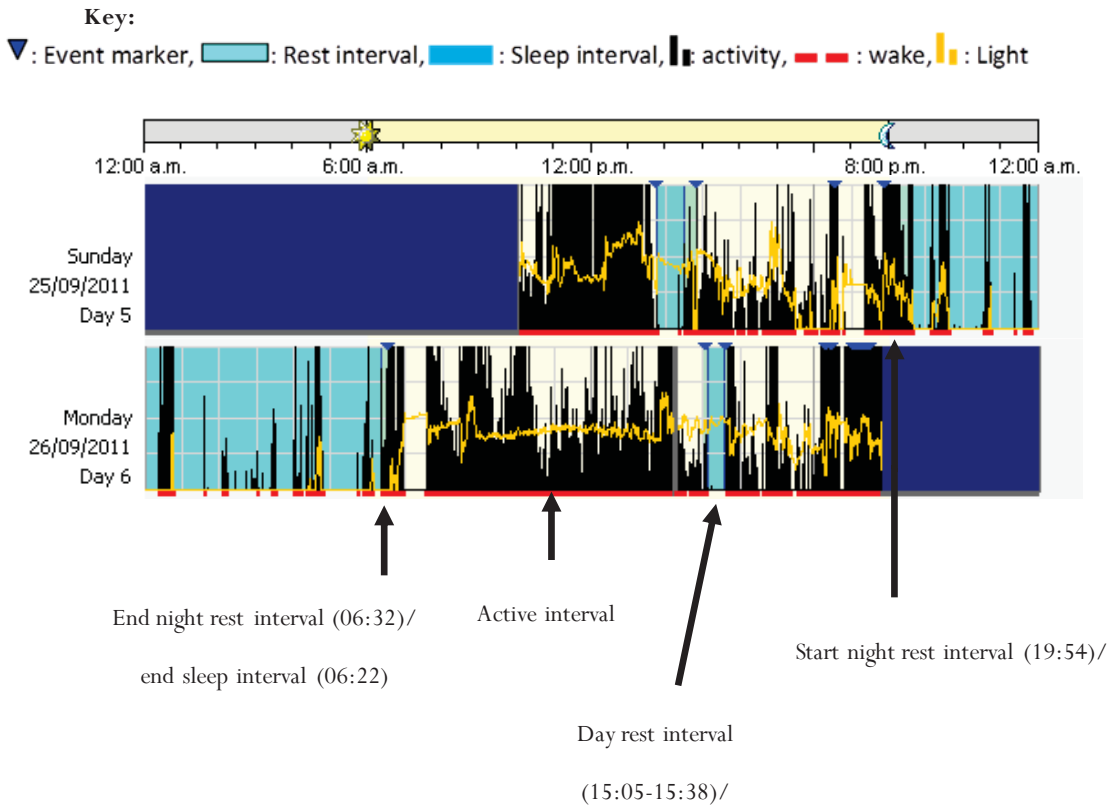

Figure 8.1 A screenshot of 48 hours of actigraphy from a participant with dementia, indicating the variables of interest and their relationships to one another.

19 Case Studies of Pairs who Withdrew

The following case studies are included to help understand the feasibility of conducting research trials for people affected by dementia living in the community. These case studies flow a similar format to those in Chapter 5.2, except the results concerning the PWD and the carers are presented together rather than separately and the field notes are used to summarise factors affecting study compliance and withdrawal rather than sleep etc. between times 1 and 2.

Pair 10 (Nicole and Claude)

Description of Study Experience

Participants.

Pair 10 consisted of a married couple who were living in a retirement village. Nicole was an 80-year-old female with AD, which was diagnosed with approximately a year prior to the study. She had a MMSE score of 21, indicating mild cognitive impairment. Her comorbidities included lung disease (treated), cancer (treated), depression, and arthritis. Her depression and cancer were considered to limit her activities. One of her medications had tiredness listed as a rare side effect (see Appendix 20).

Claude was aged 81 years at the time of the study. He had been providing dementia-related support for his wife for about two years. He was providing care all day and night but had some support from the staff at the retirement village. His comorbidities were cancer (treated), arthritis and back pain, all of which limited his activities. He also reported having hot flushes. Many of the medications Claude was taking had dizziness or somnolence listed as possible side effects, some also listed insomnia, nightmares, or teeth grinding listed (Appendix 20).

Data collection and feedback.

Claude was anxious about committing to the sleep study as Nicole was on a waiting list for moving into permanent care. She also had cataracts which may have been problematic for using the LTD. The researcher reminded them that they could use natural light instead of the LTD and that they could

withdraw from the study at any time. They agreed to take part in the first week of sleep monitoring and then reassess their situation. Nicole signed the consent form for herself.

Questionnaires were completed by Nicole, with the researcher clarifying the questions. They reported that using the Actiwatch was easy for them and they completed the diaries well. Nicole and Claude withdrew from the full study due to Nicole being offered a place in a care facility after the first week. In their feedback form, Claude was apologetic for not completing the full study as he felt they might have found it interesting or beneficial. They were enthusiastic about seeing their actigraphy results.

Results for Pair 10

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.1. Note pair 10 were missing part of the RMBPC and therefore depression and global scores were unavailable from this scale.

Nicole and Claude slept in separate bedrooms. Nicole rated her sleep as "fairly bad", her PSQI score was also high indicating that she had considerable sleep disturbance. This was mostly related to having short sleep duration at night, not being able to get to sleep within 30 minutes, and waking up in the night or early morning (three or more times per week). She also sometimes (less than once per week) had pain which disturbed her sleep. Claude added that she had some episodes of snoring, disorientation or confusion during sleep, general restlessness, as well as sleeping excessively during the day. However he did not rate these behaviours as very severe, hence the low SDI score. Scores from the MMSE and RMBPC indicate that Nicole had some cognitive impairment, including substantial problems with her memory. They both rated her quality of life as mediocre.

Claude rated his sleep as "fairly good". His PSQI score was raised, indicating some trouble sleeping. This was mostly related to not being able to get to sleep within 30 minutes, waking up in the middle of the night, and coughing or snoring (three or more times per week). He also had some less

frequent instances of sleep being disturbed by being too hot, bad dreams, or worrying at night and reported some daytime sleepiness. His HADS scores indicated low risk for anxiety or depression. His positive COPE score indicated an increased risk of carer burden. In his questionnaire he noted that he "often" felt supported in his carer role.

Table 8.1

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 10

Variable	PWD 10	Carer 10
Day sleep (hrs)	0.0	0.3
Night sleep (hrs)	4.0	7.8
PSQI (0-21)	13.0	7.0
SDI (0-12)	0.1	-
Rating of nights' sleep (1-7 median, range)	6.0 (4-7)	2.0(1-4)
MMSE (30-0)	21.0	30.0
QOL-AD PWD (52-13)	34.0	-
QOL-AD carer (52-13)	29.0	-
RMBPC Memory frequency (0-28)	26.0	-
RMBPC Memory carer reaction (0-28)	12.0	-
RMBPC disruption frequency (0-32)	3.5	-
RMBPC disruption carer reaction (0-32)	3.5	-
HADs anxiety (0-21)	-	8.5
HADs depression (0-21)	-	3.0
COPE positive (15-0)	-	9.0
COPE negative (0-18)	-	9.0

Actigraphy data.

There were six complete days of actigraphy data available from Nicole and seven from Claude. The sleep propensity curve using this data shows that they both had consistent bed and rise times at night, however Claude was sleeping less across the 24-hour period compared to Nicole (Figure 8.2).

Nicole's actigraphy data also showed that her average light exposure (between 9-11am) and activity count (between 11am-2pm) were both below the group's 25th percentile, (median lux = 1,345.3, range = 25.1-6,460.8, and median activity count = 14.8, range = 12.0-19.0, see section 5.1.5).

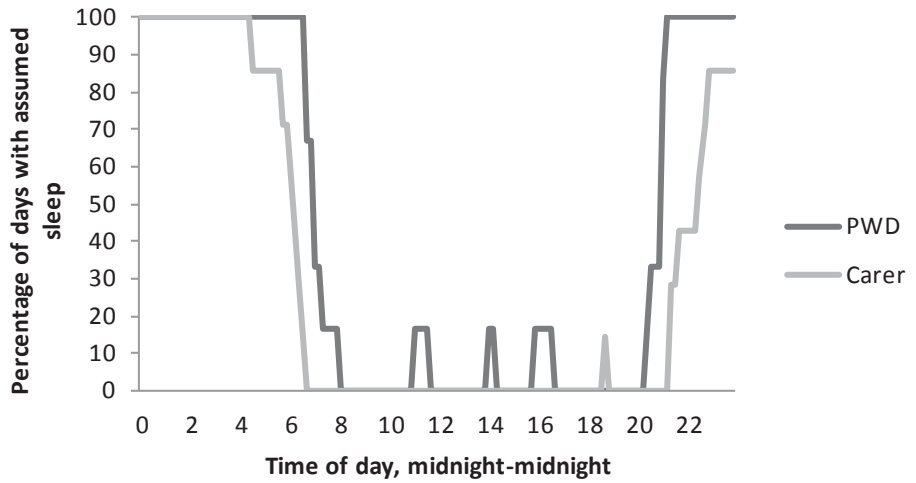

Figure 8.2. Sleep timing of Pair10 at Time 1.

Table 1.58 shows the actigraphic sleep variables for Nicole and Claude at Time 1. Nicole was having at least 10 hours sleep at night with a high sleep efficiency (over 95%) indicating good quality sleep. Claude had more variable sleep timing and less sleep at night compared to Nicole. However he was having a minimum of 6.3 hours sleep and his sleep efficiency averaged 80%.

Table 8.2

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 10

Variable	PWD 10				Carer 10			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	20:30		20:30	(20:15-20:55)	21:41		21:25	(21:00-23:30)
Risetime	07:23		07:13	(06:50-08:15)	06:24		06:15	(05:40-07:30)
Time in bed night, minutes	652.5	(32.0)	642.5	(625.0-720.0)	523.4	(23.9)	520.0	(480.0-555.0)
Total sleep per night, minutes	641.7	(32.4)	637.5	(602.0-705.0)	424.0	(29.3)	417.0	(377.0-477.0)
Sleep efficiency (% sleep night)	98.3	(1.1)	98.4	(96.3-99.7)	81.1	(5.7)	80.2	(73.9-89.0)
Wake time night, minutes	10.8	(6.9)	10.0	(2.0-23.0)	99.4	(31.8)	103.0	(58.0-143.0)
Number of night awakenings	3.2	(1.6)	3.0	(1.0-5.0)	33.1	(9.0)	33.0	(20.0-45.0)
Total sleep per day, minutes	43.3	(14.3)	45.0	(25.0-60.0)	11.0	(5.0)	11.0	(6.0-16.0)

Short summary

Nicole and Claude were potentially ideal subjects for trialling the sleep interventions. They both had disturbed sleep at night and daytime sleepiness. Furthermore Nicole had very low recordings of light and activity at baseline, therefore may have benefitted from the increase the intervention would have provided.

Nicole's sleep duration appeared to be underestimated in her questionnaires as the actigraphy recording showed that she may have been sleeping much longer than she predicted. Claude had variable sleep quantity and quality, he reported some worry affecting his sleep and his questionnaire results indicated that he may have been having some trouble coping in his carer role.

Nicole and Claude managed well with the study protocol, they did not report any problems or have any comments other than showing their interest in the study outcome. They withdrew from the study due to Nicole moving into a care facility.

Pair 11 (Henry and Beverly)

Description of Study Experience

Participants.

Pair 11 were a married couple. Henry was a 72- year-old male with AD, which was diagnosed about five years prior to the study. He had a MMSE score of 13, indicating moderate cognitive impairment. His comorbidities included heart and lung disease, anaemia or blood disease, high blood pressure and arthritis. He received treatment for his heat disease and high blood pressure. The heart disease and arthritis were considered to limit his activities. Four of his medications had tiredness or fatigue listed as possible side effects (see Appendix 20).

Beverly was aged 68 years at the time of the study. She had been providing dementia-related care for her husband for almost five-and-a-half years. She was providing care all day and night but had some support from formal care assistants. Her comorbidities were arthritis (treated) and back pain, these were not considered to limit her activities. One of the medications Beverly was taking had dizziness or

somnolence listed as possible side effects, one also had insomnia listed as a more rare side effect (Appendix 20).

Data collection and feedback.

Beverly was anxious about Henry being able to do physical activity aspect of the intervention but they were keen to take part regardless and would focus on using the light box. Henry signed the consent form himself. Questionnaires were completed in the form of an interview, with Henry and Beverly answering together. Henry appeared to struggle a little with the questions, often repeating the last option given rather than answering for himself. Therefore Beverly's responses were considered more reliable.

During the week of monitoring Henry was taking his Actiwatch off in the night and Beverly was having to frequently check it. She had tried using longer pyjama tops and bandages to keep it on. On one occasion she wrote that it was "too stressful to try and keep it on". It seemed that Beverly's sleep disturbances may have been exacerbated by the study protocol. In the feedback form Beverly noted that the Actiwatch was easy enough for her to use, but Henry considered it like a watch and felt he needed to take it off at night.

Henry was issued with a light box and exercise DVD. However four days into the trial Beverly telephoned to report that he had been admitted to hospital due to heart trouble. He would be there for a week's observations and there would be changes to his medications. They were told to suspend their sleep study until they felt they could decide whether they would continue or withdraw. The following week they withdrew.

In their feedback form Beverly noted that she found the study interesting and would have liked to have finished, however with Henry's trip to hospital she became very tired and lost the energy to support him using the interventions. She noted in the diaries and feedback form that they had tried the light box and found it alright to use although it was a challenge to keep Henry seated in front of it for the full 30 minutes. They had also tried the exercise DVD but found it difficult. The sleep support handbook was considered informative and they were left with it to consider the tips once they had recovered.

Results for Pair 11

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.3. Note pair 11 were missing part of the RMBPC and therefore depression and global scores were unavailable from this scale.

Table 8.3

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 11

Variable	PWD 11	Carer 11
Day sleep (hrs)	0.5	0.5
Night sleep (hrs)	10.0	7.0
PSQI (0-21)	9.0	7.0
SDI (0-12)	0.2	-
Rating of nights' sleep (1-7 median, range)	3.0 (2-4)	4.0 (2-6)
MMSE (30-0)	13.0	25.0
QOL-AD PWD (52-13)	48.0	-
QOL-AD carer (52-13)	29.0	-
RMBPC Memory frequency (0-28)	23.0	-
RMBPC Memory carer reaction (0-28)	3.0	-
RMBPC disruption frequency (0-32)	10.5	-
RMBPC disruption carer reaction (0-32)	19.8	-
HADs anxiety (0-21)	-	4.0
HADs depression (0-21)	-	8.0
COPE positive (15-0)	-	13.0
COPE negative (0-18)	-	4.0

Henry and Beverly slept in the same bed. Henry rated his sleep as "fairly good". His PSQI score indicated that he had some sleep disturbance. This was mostly related to not being able to get to sleep within 30 minutes, waking up in the night or early morning, and getting up to use the bathroom (at least once or twice per week). He took a sleeping tablet once or twice a week. His sleep was also disturbed by occasional coughing or snoring (less than once per week). Beverly's response to the SDI corroborated these sleep disturbances but she did not consider them very severe, hence the low score. In addition, she reported that he had the occasional episode of legs twitching or jerking during sleep.

Scores from the MMSE and RMBPC indicated that Henry had moderate cognitive impairment with some substantial problems with his memory and dementia-related behaviours. The disruptive

behaviours were rated as particularly disruptive by Beverly. Henry rated his quality of life more highly than Beverly did.

Beverly rated her sleep as "fairly good". Her PSQI score indicated some trouble sleeping and her daily diary rating corroborated this. Her sleep was mostly disturbed due to waking up in the middle of the night or early morning, getting up to use the bathroom, or feeling too hot. She also had some instances of not being able to get to sleep within 30 minutes, and trouble sleeping due to pain. Her HADS score was borderline for risk of depression but within the normal range for anxiety. Her COPE scores indicated low risk of carer burden. In her questionnaire she marked that she "often" felt supported in her carer role, she noted that she felt she coped "reasonably well as a caregiver…but like most carers I have my moments".

Actigraphy data

There were four complete days of actigraphy data available from Henry, and seven from Beverly. The sleep propensity curves in *Figure 8.3* show that Henry had a more variable bedtime than Beverly and that he napped more in the day. Table 8.4 shows Henry and Beverly's actigraphic sleep variables at Time 1.This shows that Henry was having at least 6.8 hours sleep per night but he was spending up to 3 hours awake at night which affected his sleep efficiency. Beverly's sleep timing was similar but not as variable as Henry's. She was having more than 7 hours of sleep at night. Her sleep efficiency was above 80%, although she was spending up to 1.8 hours awake at night. She occasionally napped for less than an hour in the daytime.

Henry's actigraphy data also showed that he had a high total light exposure between 9-11am (within the 75th percentile for the entire group, median = 13,492.8 lux, range = 2,168.0-98,865.6). However his average activity count between 11am-2pm was low (within the group's 25th percentile, median = 110, range = 89.6-265.1, see section 5.1.5).

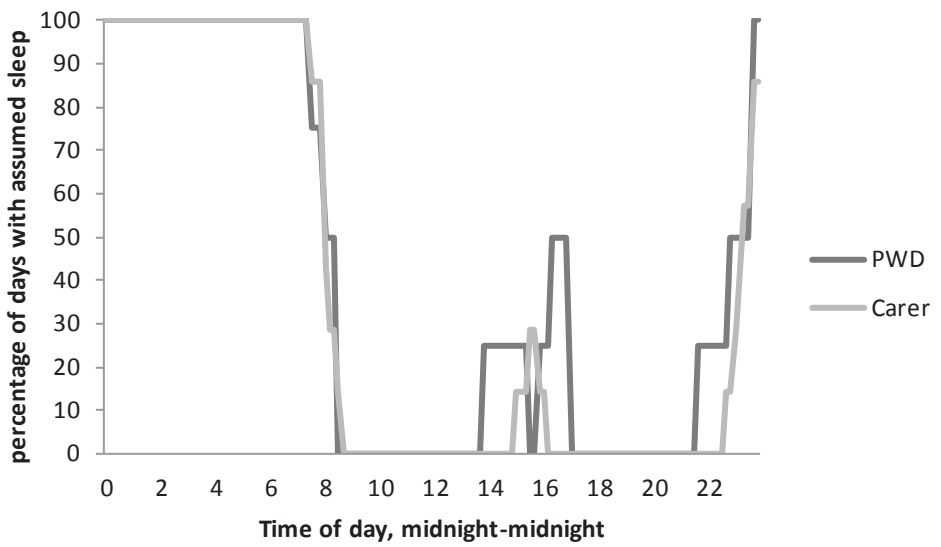

Figure 8.3. Sleep timing of pair11 at Time 1.

Short Summary

Henry and Beverly were potentially ideal subjects for trialling the sleep interventions. They both had disturbed sleep at night, their subjective reports indicated a lot of wakefulness and disruption at night and their actigraphy results corroborated this.

Beverly reported that they were very interested in the study however had some problems with the study protocol including getting Henry to keep his Actiwatch on and to sit in front of the light box for the full 30 minutes. These issues were appearing to concern Beverly and disrupt her sleep further. They withdrew from the study due to Henry becoming unwell and spending some time in hospital. Beverly became tired and reported feeling overwhelmed by the prospect of maintaining the study protocol on top of supporting Henry on his return from hospital.

Table 8.4

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 11

Variable	PWD 11				Carer 11			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	21:37		22:03	(19:54-22:29)	22:13		22:10	(21:36-22:49)
Risetime	7:14		7:27	(6:32-07:30)	7:09		7:02	(6:31-7:50)
Time in bed night, minutes	576.8	(38.3)	564.0	(541.0-638.0)	536.0	(19.0)	540.0	(492.0-556.0)
Total sleep per night, minutes	451.3	(23.1)	462.5	(412.0-468.0)	463.3	(24.9)	452.0	(432.0-502.0)
Sleep efficiency (% sleep night)	78.5	(5.1)	78.4	(71.6-85.4)	86.5	(4.0)	87.4	(80.0-92.3)
Wake time night, minutes	125.5	(36.6)	120.5	(80.0-181.0)	72.7	(22.2)	70.0	(42.0-108.0)
Number of night awakenings	23.5	(2.1)	23.5	(21.0-26.0)	24.6	(4.9)	24.0	(18.0-35.0)
Total sleep per day, minutes	51.2	(14.4)	47.0	(31.0-75.0)	49.0	(6.0)	49.0	(43.0-55.0)

Pair 12 (Sidney and Fiona)

Description of Study Experience

Participants.

Pair 12 were a married couple, Sidney was an 88-year-old man with MCI, which was diagnosed one year prior to the study. He associated his MCI with a stoke he had eight years prior. He had a MMSE score of 29, indicating cognitive functioning within the normal range. His comorbidities included high blood pressure and arthritis. He wasn't treated for either and his arthritis was reported to limit his activities. One of his medications had tiredness or fatigue listed as possible side effects, two others had insomnia or changes to sleep patterns listed as more rare side effects (see Appendix 20).

Fiona was aged 75 years at the time of the study. She had been providing dementia-related care for her husband for 11 months. She was providing care for several hours a day, and had some support from a day care centre which he attended. Fiona had some back pain, no medications were disclosed.

Data collection and feedback.

At the beginning of the study Sidney was a little concerned about the term 'dementia' surrounding the research as he hadn't had an official diagnosis. He was reassured that the main focus of the study was on his sleep and he agreed to take part (he signed the consent form himself). Questionnaires were completed by Sidney with the researcher's guidance, he had them in front of him to read and either verbally answered or filled in the answer himself. Sidney also completed a lot of his own sleep diary (or at least was helping Fiona). She commented on how surprised she was that he could remember so many details.

At the beginning of week one, Sidney swapped his Actiwatch onto his dominant wrist due to some bruising on his left, however then transferred it back again. This may have affected the reliability of his actigraphy results.

Sidney was issued with a light box and DVD. He struggled with walking as had an amputated limb, so he was enthusiastic about the DVD option for exercise. During the first week of the intervention

they reported getting on well, and had used the light box. However, Fiona was due to go away and Sidney would be going into respite care for a long weekend. He did not take the light or DVD, but agreed to continue his diaries while he was away. Sidney got out of the habit of keeping his diaries or using the interventions after his time in respite. He did not use the light box for the whole week. The researcher reassured them that there was still three weeks of trial time if they wanted to continue. However Sidney decided that there was little point as he felt he slept well and had "had enough" of the protocol therefore they withdrew from the study. When the equipment was collected it seemed that Sidney had been using the light box at random times of day rather than during the 9-11am period. He liked how bright it was and used it for reading.

Fiona noted that future studies might want to consider using the term "neurological conditions" instead of "dementia" to avoid feeling of stigma surrounding the language. She also reported that the diaries were sometimes confusing, especially when completing them retrospectively. She thought that the study protocol and completing the questionnaires could be difficult for people with cognitive impairment. Sidney also commented that he found the diary and questionnaire a challenge. If he did not complete them soon after the events it became more difficult for him to recall the correct answer. On the feedback form Sidney noted that he did not complete the trial as he didn't realise what the study entailed and how much time it would take to complete.

Results for Pair 12

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.5. Sidney rated his sleep as "fairly good", his PSQI score was borderline for having a sleep disturbance. This was due to sometimes waking in the night or getting up to go use the bathroom. He would also occasionally have trouble sleeping due to being too hot (one or twice per week), bad dreams, or legs twitching (but less than once per week). His daily diary ratings of his sleep were average quality. No dementia-related sleep disturbances were recorded by Fiona on the SDI. Sidney's

MMSE and RMBPC indicate that he had normal cognitive functioning but some problems with his memory, he and Fiona both rated his quality of life as mediocre.

Fiona rated her sleep as "fairly good". Her PSQI score was within the normal range, however her average daily diary rating was raised indicating some poor nights. In her questionnaire she reported that her sleep was occasionally disturbed by getting up to go to the bathroom (three or more times per week), or feeling too hot (once or twice per week). She would also have occasional instances of not being able to get to sleep within 30 minutes, having bad dreams, or daytime sleepiness (less than once per week). Her HADS and COPE scores were within the normal ranges, indicating that she was at low risk for anxiety, depression, or carer burden. In her questionnaire she noted that she "often" felt supported in her role as a caregiver.

Actigraphy data.

There were seven complete days of actigraphy data available from both Sidney and Fiona. The sleep propensity curves in *Figure 8.4* shows that Sidney and Fiona appeared to have stable sleep timing with the occasional short nap.

Table 8.6 shows Sidney and Fiona's actigraphic sleep variables recorded at Time 1. This shows that they went to bed around the same time, however Sidney was sometimes having short sleep duration at night (as little as 5.2 hours). He would spend over an hour awake whilst in bed at night, therefore his sleep efficiency was sometimes less than 80%. He had the occasional nap in the daytime of approximately one hour. Fiona was having more sleep at night (a minimum of 6.4 hours) than Sidney. She spent an average of 43 minutes awake at night, however her sleep efficiency was over 85% indicating good quality sleep in general. She had two instances of going to bed in the daytime but only one in which she slept for 29 minutes indicating that she did not regularly take naps.

Sidney's actigraphy data also showed that he had a high total light exposure between 9-11am (above the 75th percentile for the entire group, median = 8,120.5 lux, range = 2,022.1-27,344.7). However his average activity count between 11am-2pm was low (less than the group's 25th percentile, median = 92.9 counts, range = 66.7-124.3 see section 5.1.5).

Table 8.5

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 12

Variable	PWD 12	Carer 12
Day sleep (hrs)	1.5	0.4
Night sleep (hrs)	8.5	7.5
PSQI (0-21)	5.0	4.0
SDI (0-12)	0.0	-
Rating of nights' sleep (1-7 median, range)	4.0 (2-5)	4.0 (2-5)
MMSE (30-0)	29.0	30.0
QOL-AD PWD (52-13)	36.0	-
QOL-AD carer (52-13)	30.7*	-
RMBPC Memory frequency (0-28)	15.0	-
RMBPC Memory carer reaction (0-28)	12.0	-
RMBPC depression frequency (0-36)	5.0	-
RMBPC depression carer reaction (0-36)	9.0	-
RMBPC disruption frequency (0-32)	0.0	-
RMBPC disruption carer reaction (0-32)	0.0	-
RMBPC global frequency (0-96)	20.0	-
RMBPC global carer reaction (0-96)	21.0	-
HADs anxiety (0-21)	-	8.4
HADs depression (0-21)	-	7.0
COPE positive (15-0)	-	12.0
COPE negative (0-18)	-	5.0

* = Item imputation used due to missing data from <20% of the component scores.

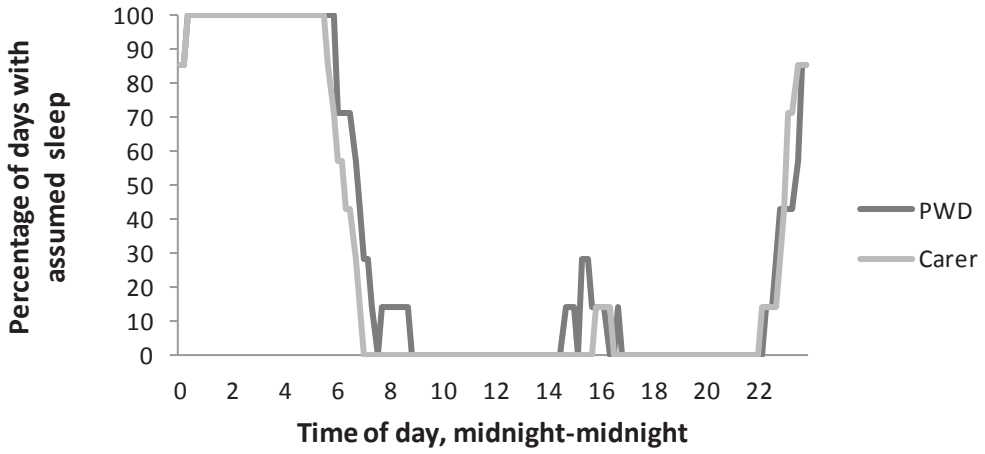

Figure 8.4. Sleep timing of Pair 12 at Time 1.

Table 8.6

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 12

Variable	PWD 12				Carer 12			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	22:58		22:45	(22:05-00:15)	22:53		22:45	(22:00-23:58)
Risetime	6:50		6:46	(6:00-07:30)	06:23		06:20	(05:45-07:00)
Time in bed night, minutes	472.3	(51.0)	495.0	(391.0-535.0)	449.6	(42.4)	450.0	(382.0-510.0)
Total sleep per night, minutes	380.3	(46.0)	405.0	(312.0-437.0)	402.4	(33.4)	401.0	(347.0-443.0)
Sleep efficiency (% sleep night)	80.5	(3.1)	81.7	(74.7-84.6)	89.6	(1.5)	90.1	(86.9-91.1)
Wake time night, minutes	92.0	(16.2)	90.0	(72.0-118.0)	47.1	(10.7)	43.0	(35.0-67.0)
Number of night awakenings	24.0	(5.9)	22.0	(15.0-36.0)	21.1	(2.1)	21.0	(18.0-25.0)
Total sleep per day, minutes	59.3	(31.2)	63.5	(19.0-98.0)	13.3	(12.0)	11.0	(0.0-29.0)

Short Summary

Sidney and Fiona appeared to be outside of the range of subjects who would benefit from the interventions. Sidney's cognitive impairment as well as sleep troubles were mild, and he seemed to lead a busy lifestyle. Fiona was also a reasonably good sleeper and did not have to provide full time care to Sidney. Therefore they had less motivation for him to trial the treatments.

As he was cognitively able, Sidney wanted to take on a lot of the study protocol himself. However, Fiona reported that he had some issues remembering the finer details which made completing the diaries and using the light box at the correct times of day more difficult. Sidney and Fiona withdrew from the study due to Sidney going into short-term respite care then losing interest in the study. He did not consider his sleep problematic enough to justify continuing with the study protocol which he was finding more time consuming than expected.

Pair 13 (Maria and Felix)

Description of Study Experience

Participants.

Pair 13 were a married couple. Maria was an 83-year-old woman with VaD which was diagnosed approximately seven years prior to the study. She had a MMSE score of 20, indicating mild cognitive impairment. She also had high blood pressure which she was treated for. One of her medications had inability to sleep listed as a rare side effect (see Appendix 20).

Felix was aged 82 years at the time of the study. He had been providing dementia-related care for his wife for 6.5 years. He was providing care all day and night but had some support from family members. His comorbidities included diabetes as well as polymyalgia and atrial fibrillation. These were all treated and not considered to limit his activities. He took eight medications which had sleep-related side effects listed, including fatigue, somnolence or insomnia (see Appendix 20).

Data collection and feedback.

At the beginning of the study Felix disclosed that they didn't really have many sleeping problems. He considered his own sleep as poorer than Maria's and he didn't associate his sleep problems with his carer role. However he was keen to take part and support the research with regards to trialling the methodology. Maria signed the consent forms herself, her questionnaires were completed in the form of an interview, with the researcher completing the paperwork as she read long.

During the week of actigraphy monitoring Felix had a night in hospital due to chest pains. Maria's daughter was supporting Maria with her dementia-related care as well as the study protocol. She reported that they were getting on well. Due to this disruption they had an additional three nights of actigraphy monitoring, so that the time surrounding Felix's time in hospital could be excluded from analyses.

Maria and Felix withdrew from the study at the end of the first week due to not considering their sleep problematic enough to warrant the trial. They were also already practicing a lot of the recommended activities, e.g. having long walks in the sunshine. So they didn't feel they would get any further benefit from the light box or DVD. They indicated that using the Actiwatch, diaries and questionnaires was easy. No other comments or feedback were given.

Results for Pair 13

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.7.

Maria slept in the same room but not the same bed as Felix. She rated her sleep as "very good", and her PSQI score was within the normal range. She would occasionally get up to go to the bathroom (but less than once per week). Felix's score on the SDI corroborated that Maria had good sleep. He only reported that she would sometimes wake a little earlier than habit but this wasn't considered problematic.

Maria's MMSE and RMBPC scores show that she had minor cognitive impairment, with some memory problems and mood disruption. She and Felix both rated her quality of life well.

Table 8.7

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 13

Variable	PWD 13	Carer 13
Day sleep (hrs)	0.0	0.3
Night sleep (hrs)	8.0	5.5
PSQI (0-21)	3.0	8.0
SDI (0-12)	0.0	-
Rating of nights' sleep (1-7 median, range)	2.0 (2-2)	4.0 (2-4)
MMSE (30-0)	20.0	29.0
QOL-AD PWD (52-13)	39.0	-
QOL-AD carer (52-13)	35.0	-
RMBPC Memory frequency (0-28)	19.0	-
RMBPC Memory carer reaction (0-28)	4.0	-
RMBPC depression frequency (0-36)	9.0*	-
RMBPC depression carer reaction (0-36)	4.5*	-
RMBPC disruption frequency (0-32)	0.0	3.0
RMBPC disruption carer reaction (0-32)	0.0	4.0
RMBPC global frequency (0-96)	28.1	13.0
RMBPC global carer reaction (0-96)	8.6	5.0
HADs anxiety (0-21)	-	3.0
HADs depression (0-21)	-	4.0
COPE positive (15-0)	-	13.0
COPE negative (0-18)	-	5.0

* = * = Item imputation used due to missing data from <20% of the component scores.

Felix rated his sleep as "fairly good". However his PSQI score indicated some trouble sleeping. This was mainly due to waking up in the night or early morning, getting up to go to the toilet, or bad dreams (once or twice per week). He also had the occasional episode of waking disorientated or confused. His HADS' and COPE scores were within the normal ranges, indicating that he was at low risk for anxiety, depression or carer burden. In his questionnaire he noted that he "always" felt supported in his role as a caregiver.

Actigraphy data.

There were seven complete days of actigraphy data available from both Maria and Felix . The sleep propensity curves in *Figure 8.5* show that they had consistent sleep timing at night and no daytime naps.

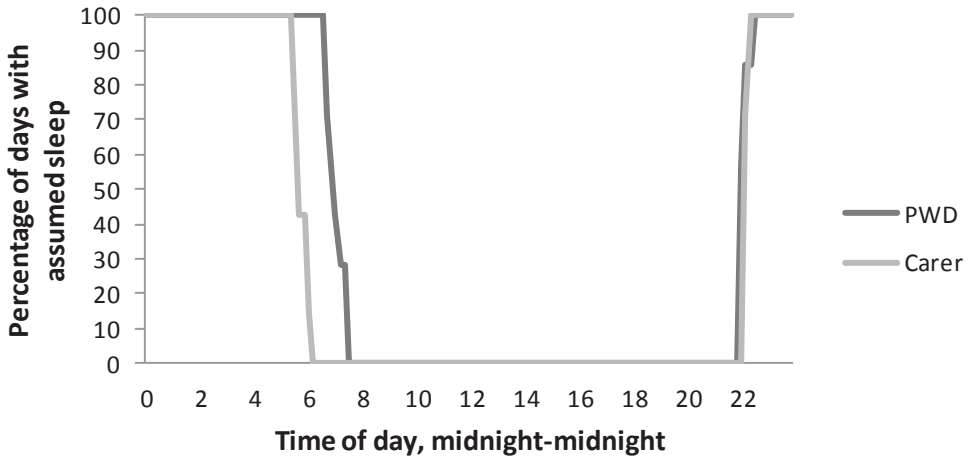

Figure 8.5. Sleep timing of Pair 13 at Time 1.

Table 8.8 shows Maria and Felix's actigraphic sleep variables at Time 1. This confirms that Maria had consistent sleep timing and shows that she was having at least 8.5 hours sleep at night. She had good quality sleep as indicated by her sleep efficiency at night being over 90%. Felix's had a shorter sleep duration than Maria and would spend up to an hour and a half awake at night. However he still had a high sleep efficiency (over 85%) indicating reasonable sleep quality.

Maria's actigraphy data also showed that she had reasonable amount of total light exposure (median= 6,514.5 lux, range = 3,609.4-9,325.4) during the 9-11am interval, as well as average activity counts (median = 132.1 counts, range = 91.1-151.5) between 11am-2pm. These were within the 50th percentile for the entire group (see section 5.1.5).

Table 8.8

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 13

Variable	PWD 13				Carer 13			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	21:59		22:00	(21:45-22:13)	21:59		22:00	(21:45-22:13)
Risetime	7:18		7:23	(6:55-7:44)	5:54		5:58	(5:30-6:10)
Time in bed night, minutes	559.6	(22.2)	550.0	(535.0-587.0)	475.3	(12.5)	480.0	(460.0-491.0)
Total sleep per night, minutes	534.6	(20.0)	526.0	(511.0-569.0)	425.7	(18.5)	436.0	(398.0-444.0)
Sleep efficiency (% sleep night)	95.6	(1.9)	96.3	(92.5-97.4)	89.5	(2.2)	90.4	(86.5-92.5)
Wake time night, minutes	25.0	(11.2)	20.0	(14.0-44.0)	49.6	(9.8)	46.0	(36.0-63.0)
Number of night awakenings	15.3	(5.7)	13.0	(8.0-24.0)	23.3	(5.4)	22.0	(17.0-34.0)
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	5.7	(4.5)	3.0	(2.0-12.0)

Short Summary

Maria and Felix appeared to be outside of the range of subjects who would benefit from the interventions. Maria's cognitive impairment was mild and she did not have any sleep problems. Felix did have trouble sleeping but he reported that this was unrelated to his carer role. Felix spent some time in hospital which disrupted their actigraphy study. Due to Maria's lack of sleep problems and their routine already including plenty of light and exercise, Maria and Felix decided that they did not want to complete the trial.

Pair 14 (Georgina and Lisa)

Description of Study Experience

Participants.

Pair 14 were mother and daughter. Georgina was an 89-year-old woman who was temporarily living with her daughter and her family, while the family organised care assistance for her in her home town, overseas. She had an undefined type of dementia, which was diagnosed approximately two years prior to the study. Her MMSE score was 23 which was within the normal range of cognitive functioning. Her comorbidities included heart disease (treated) and high blood pressure, she also reported that she had numb legs which limited her activities. She frequently took a sleeping tablet which understandably had sedating effects as well as having other side effects concerning sleep. Georgina also took another medication which had fatigue, insomnia as well as abnormal dreams listed as possible side effects (see Appendix 20). Lisa said that her mother reported sleeping better while staying with them due to not having to worry about her own housekeeping.

Lisa was aged 55 years at the time of the study. She had been providing dementia-related care for her mother for six months. She was providing care for several hours a day, whilst also working a 40-hour job, and caring for her teenage children. She had support from her husband. She had treated cancer which wasn't considered to limit her activities. Lisa also took a sleeping medication regularly which had sedating effects as well as having other side effects concerning sleep (see Appendix 20).

Data collection and feedback.

At the beginning of the study Lisa had some concerns as Georgina was not fully aware of her dementia diagnosis, so she did not want the research process to be too distressing. However they both had sleep problems and were interested in the study. The researcher assured Lisa that the home visit would be approached very carefully, and additional time and support would be made available for Georgina.

Georgina signed the consent form for herself. She seemed anxious and her behaviour was somewhat defensive towards the researcher. Therefore Lisa remained present when she was completing the MMSE. The questionnaires were completed together as an interview, with Georgina reading the questions and the researcher marking her answers.

Lisa reported that Georgina was a little upset after the initial home visit and appeared to consider the researcher as a clinician despite her seeming to understand what the research was about. She refused to wear the Actiwatch on the first night. Lisa commented that Georgina would forget why she had to wear the Actiwatch and remove it, then would appear paranoid when reminded to put it back on again.

They were issued with a light box and DVD however Georgina resisted using either at first. Lisa reasoned that this was due to an association her mother had made with the research process which she considered clinical and threatened by. They were reminded that they could withdraw at any time. Lisa had been offering the light box as an option for Georgina but not forcing its use. She reported that Georgina had been having more walks which they logged, apparently she was more interested in activity in general.

They continued to trial the interventions as Lisa believed that her mother would "come around". Over the subsequent weeks it was reported that Georgina was having more walks outside and they felt there may have been some improvement to sleep. She also reported having nice dreams more often. However she still refused to use the light box. Lisa struggled to maintain the diaries after week one, commenting that she lost the motivation without having the Actiwatch to remind her to complete them.

Towards the end of the trial Lisa was concerned that she did not want to upset her mother by going through the sleep monitoring process and questionnaires again. She was due to move back to her own home overseas, and she didn't want to disrupt the process. It was agreed that Lisa could complete the

questionnaires with Georgina and initialise wearing the Actiwatches again. However Georgina refused to wear the Actiwatch at the end of the trial. Lisa felt that the study was tainted by her experience at the onset and did not want to add to their family's burden by pursuing the study any further.

In the feedback form Lisa commented that, despite being made fully aware of what the study entailed, she hadn't realised how much of a negative impact completing the questionnaire would have on her mother. There was a resistance to answering the questions, Lisa associated this with the thought and concentration required as being too challenging as well as confronting. She also associated the negative impact of the research with paranoia which Georgina typically exhibited with regards to clinical processes rather than the researcher's approach. She commented that she was very happy with the way the researcher approached them and their concerns. However, recommended that future research should focus more with the engagement of PWD with the trial itself, rather than the assessments as the initial "testing" process could be off-putting or met with hostility.

Results for Pair 14

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.9.

Georgina slept in a separate room form Lisa. She rated her sleep as "very good" and her PSQI score was within the normal range. She would get up to go to the bathroom (three or more times per week), and occasionally have trouble sleeping due to bad dreams. Lisa's score on the SDI indicated that Georgina had some additional issues with her sleep including snoring, getting up in the night, waking others, as well as sleeping excessively in the day (however most of these activities were occurring less than once per week and were not considered severe, hence the low score). Although Georgina's MMSE score was within the normal range, her RMBPC indicated that she had some considerable problems with her memory, as well as some disruptive behaviours related to dementia. Lisa reported Georgina's quality of life as lower than she did.

Table 8.9

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 14

Variable	PWD 14	Carer 14
Day sleep (hrs)	0.0	0.3
Night sleep (hrs)	9.5	8.0
PSQI (0-21)	4.0	9.0
SDI (0-12)	0.5	-
Rating of nights' sleep (1-7 median, range)	Missing	Missing
MMSE (30-0)	23.0	Missing
QOL-AD PWD (52-13)	43.6	-
QOL-AD carer (52-13)	33.6*	-
RMBPC Memory frequency (0-28)	14.0	-
RMBPC Memory carer reaction (0-28)	2.0	-
RMBPC depression frequency (0-36)	5.0	-
RMBPC depression carer reaction (0-36)	4.0	-
RMBPC disruption frequency (0-32)	9.0	-
RMBPC disruption carer reaction (0-32)	8.0	-
RMBPC global frequency (0-96)	28.0	-
RMBPC global carer reaction (0-96)	14.0	-
HADs anxiety (0-21)	-	3.0
HADs depression (0-21)	-	1.0
COPE positive (15-0)	-	14.0
COPE negative (0-18)	-	5.0

* = Item imputation used due to missing data from <20% of the component scores.

Lisa rated her sleep as "fairly bad". Her PSQI score indicated some trouble sleeping, which was mainly associated with not being able to get to sleep within 30 minutes, waking in the night or early morning, getting up to use the bathroom, and having bad dreams (once or twice per week). She regularly took sleeping medication (three or more times per week), and reported that she had trouble sleeping because she was stressed and restless. Her HADS and COPE scores were within the normal ranges, indicating that she was at low risk for anxiety, depression or carer burden. In her questionnaire she noted that she "often" felt supported in her role as a caregiver.

In their questionnaires, Lisa commented that her mother was coping with her dementia and sleeping a lot better since moving into their family home. Prior to this she was living on her own in a big house with les stimulation and without reminders to maintain a routine for healthy eating and sleeping. She noted that Georgina was more calm staying with them, and would keep to a routine, eat well, and take her medications on time. All of these factors were considered to be helping her sleep through the night.

Actigraphy data.

There were six complete days of actigraphy data available from Georgina and seven from Lisa. The sleep propensity curves in *Figure 8.6* show that they both had consistent sleep timing at night however a more variable rise time. Neither of them napped in the day.

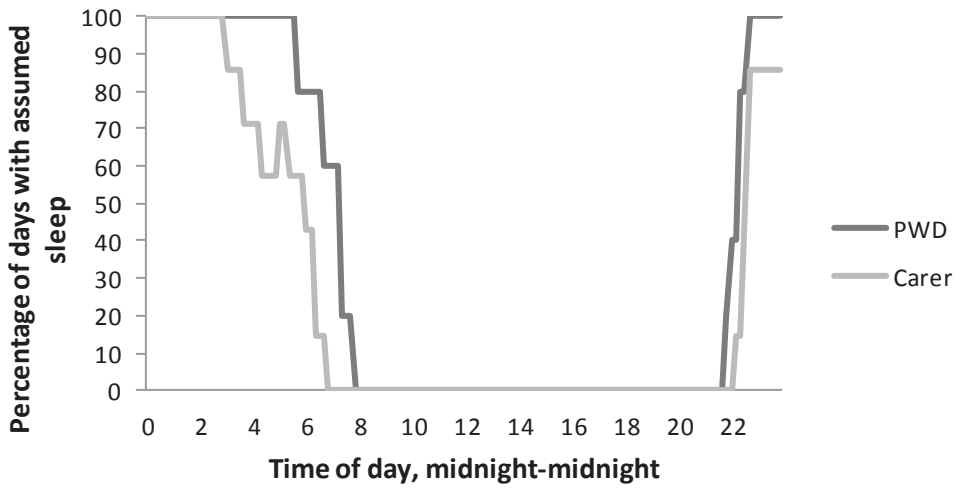

Figure 8.6. Sleep timing of Pair 14 at Time 1.

Table 8.10 shows Georgina and Lisa's actigraphic sleep variables at Time 1. This shows that Georgina was having at least 7 hours sleep at night. She had reasonable quality sleep as indicated by her average sleep efficiency at night being 85%, however some night she was spending over 2 hours wake and therefore her sleep efficiency was variable. Lisa's sleep was poorer than Georgina's. Her average sleep duration at night was less than 6 hours, and she would, on average, spend over 2 hours awake at night. Therefore her sleep efficiency was often reduced.

Table 8.10

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 14

Variable	PWD 14				Carer 14			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	22:18		22:13	(21:55-22:45)	18:54		22:10	(00:05-22:58)
Risetime	7:38		7:35	(7:02-08:11)	06:44		06:25	(05:45-08:10)
Time in bed night, minutes	559.8	35.0	567.0	(497-611)	504.3	(93.6)	495.0	(375.0-659.0)
Total sleep per night, minutes	470.2	26.2	482.0	(423.0-498.0)	372.4	(51.9)	354.0	(295.0-443.0)
Sleep efficiency (% sleep night)	84.2	(5.5)	85.3	(74.6-90.1)	74.9	(9.2)	72.5	(64.3-89.9)
Wake time night, minutes	89.7	34.4	84.0	(49.0-144.0)	131.9	(63.7)	141.0	(47.0-235.0)
Number of night awakenings	14.8	2.9	16.0	(10.0-18.0)	30.1	(7.2)	31.0	(19.0-41.0)
Total sleep per day, minutes	0.0	(0.0)	0.0	(0.0-0.0)	0.0	(0.0)	0.0	(0.0-0.0)

Georgina's actigraphy data also showed that she had high total light exposure between 9-11am (median = 9,053.0 lux, range = 2,231.4-3,0299.3) as well as average activity counts between 11am-2pm (median = 365.2 counts, range = 305.7-371.1). These were within the 75th percentile for the entire group (see section 5.1.5).

Short Summary

Georgina and Lisa were an interesting pair who had many barriers to completing the research protocol successfully. Georgina was living temporarily with her daughter and her family. Her anxiety around clinical processes and her not being fully aware of her dementia diagnosis made the consent process and data collection difficult. Her hostility towards the researcher transferred onto any activities associated with the research. Lisa was working full time as well as caring for her mother and teenage family. Although her questionnaire scores were within the normal range, she admitted feeling stressed, and she felt that this affected her sleep and their study compliance.

Georgina was issued with the LTD and DVD however refused to use them. Lisa reported that this was due to the clinical association her mother had made to the research. They withdrew from the study prior to the Time 2 monitoring, as Georgina was due to move back into her own home and Lisa and her family did not want to create any further stress on the family at this difficult time.

Pair 15 (Edward and Rose)

Description of Study Experience

Participants.

Pair 15 were a married couple. Edward was a 73-year -old male with VaD, which was diagnosed two years prior to the study. He had a MMSE score of 18, indicating moderate cognitive impairment. His comorbidities included high blood pressure, anaemia or blood disease, cancer, depression, back pain, and a prostate problem. He received treatment for all of these conditions except his depression. Most of them were considered to limit his activities. Three of his medications had tiredness or fatigue listed as possible side effects (see Appendix 20).

Rose was aged 55 years at the time of the study. She had been providing dementia-related care for her husband for one year. She was providing care all day and night and was in the process of organising some additional support. No comorbidities or medications were disclosed.

Data collection and feedback.

Edward and Rose had recently moved into the region, due to downsizing their business and home in order to manage Edward's dementia and be closer to family. At the beginning of the study, Rose commented on how bad Edward's sleep was, giving the example that he would go to his room for a nap and return five minutes later believing he had slept for a long time. They would often be awake at night and Edward was also sleeping a lot during the day. She was very keen for them to try a non-pharmaceutical alternative to treating his sleep problems.

Edward signed the consent form for himself but his daughter also signed a statement by partner form due to his severe cognitive impairment. Questionnaires were completed interview style, with Edward reading the questionnaire and Rose helping him to answer. Four days into actigraphy monitoring Edward had a fall (suspected seizure) and was admitted to hospital. They suspended their sleep monitoring at this time due to the hospital environment and changes to medications.

On Edward's return from hospital, Rose commented that his sleep had become much worse. She was finding it so hard to manage she had some sleeping tablets prescribed for Edward but they didn't seem to be having any effect. She was still very enthusiastic about taking part in the trial and the researcher agreed that, if the baseline questionnaire data could be repeated, then they could still trial the interventions. However the following week, Edward moved into a care facility and they withdrew from the study. Rose expressed her disappointment at not having the opportunity to try the interventions. She was given the sleep support handbook. She mentioned that she would endeavour to help Edward exercise and get more sunlight whenever possible.

Results for Pair 15

Questionnaire and diary data.

Questionnaire and diary data concerning subjective sleep ratings as well as indicators of cognitive functioning, quality of life, dementia-related behaviours, plus carer anxiety, depression or problems coping can be found in Table 8.11

Table 8.11

Questionnaire and Diary Data at Time 1 Concerning PWD and Carer 15

Variable	PWD 15	Carer 15
Day sleep (hrs)	1.5	0.0
Night sleep (hrs)	10.0	8.0
PSQI (0-21)	4.0	10.0
SDI (0-12)	2.0	-
Rating of nights' sleep (1-7 median, range)	3.0 (3-3)	4.0 (2-6)
MMSE (30-0)	18.0	29.0
QOL-AD PWD (52-13)	39.0	-
QOL-AD carer (52-13)	23.0	-
RMBPC Memory frequency (0-28)	28.0	-
RMBPC Memory carer reaction (0-28)	6.0	-
RMBPC depression frequency (0-36)	14.0	-
RMBPC depression carer reaction (0-36)	18.0*	-
RMBPC disruption frequency (0-32)	10.0	-
RMBPC disruption carer reaction (0-32)	12.0	-
RMBPC global frequency (0-96)	52.0	-
RMBPC global carer reaction (0-96)	35.8	-
HADs anxiety (0-21)	-	4.0
HADs depression (0-21)	-	2.0
COPE positive (15-0)	-	6.0
COPE negative (0-18)	-	9.0

* = Item imputation used due to missing data from <20% of the component scores.

Edward and Rose slept in the same bed. Edward rated his sleep as "fairly good". His PSQI score was within the normal range however he had trouble sleeping because of waking in the night or early morning, getting up to use the bathroom, coughing or snoring, pain or being woken by Rose (three or more times per week). Rose also noted that he had occasional apnoeas. Her SDI score indicated that Edward had some additional, dementia-related sleep disturbances. These included episodes of disorientation or confusion, wandering and disruptive behaviours at night, as well as sleeping excessively in the day. She typically rated these as moderate-marked and she found them distressing.

Scores from the MMSE and RMBPC indicate that Edward had moderate cognitive impairment with some substantial problems with his memory. He also had some symptoms of depression and disruptive behaviours which seemed to have a marked effect on Rose. Edward rated his quality of life more highly than Rose did.

Rose rated her sleep as "fairly bad". Her PSQI score indicated some trouble sleeping and her daily diary rating corroborated this. Her sleep was mostly disturbed by being able to get to sleep within 30 minutes, waking up in the middle of the night or early morning, getting up to use the bathroom, feeling too hot, and being woken by Edward (three or more times per week). She also had some instances of restless legs (three or more times per week). Her HADS scores were within the normal range indicating low risk for anxiety or depression. However, her positive COPE score was reduced, indicating increased likelihood for carer burden. In her questionnaire she indicated that she "sometimes" felt supported in her carer role.

Actigraphy data.

There were three complete days of actigraphy data available from Edward, and four from Rose. The sleep propensity curves in *Figure 8.7* show that they both had variable sleep timing, Edward napped more often than Rose.

Table 8.12 shows Edward and Rose's actigraphic sleep variables at Time 1. This shows that over the three days prior Edward being admitted to hospital, he was having an average of 5.5 hours sleep at night. He was spending between 3.5 and 6.7 hours of the night awake, therefore his sleep efficiency was very low. Rose's sleep timing was also variable. She was having over 6.5 hours of sleep at night, however was spending up to 1.9 hours awake, therefore her sleep efficiency sometimes affected.

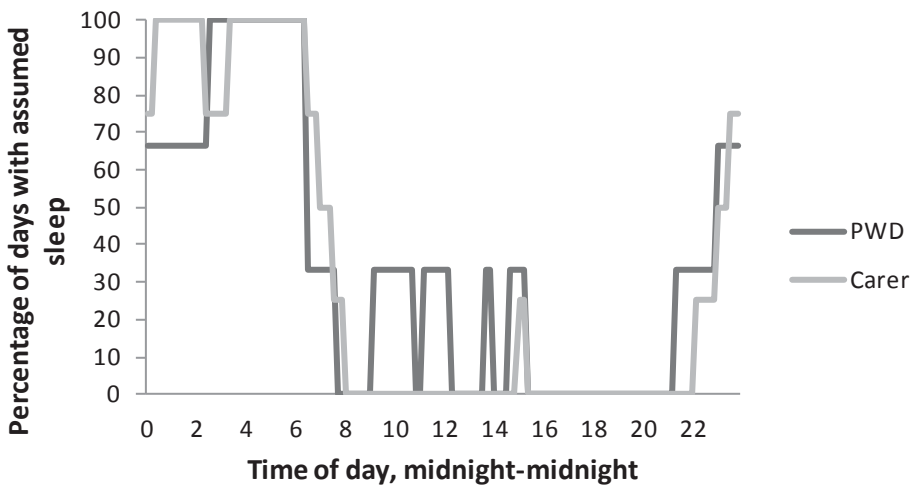

Figure 8.7. Sleep timing of Pair 15 at Time 1.

Edward's actigraphy data also showed that he had reasonable exposure to bright light during the 9-11am timeframe (within the 50th percentile for the group , median = 1,520.4 lux, range = 673.2-2,367.5), however his average activity count between 11am-2pm was in the lower 25th percentile (median = 111.5 counts, range = 85.5-174.0, see section 5.1.5).

Short Summary

Edward and Rose were potentially ideal subjects for trialling the sleep interventions. They both had disturbed sleep at night. Edward had very short and fragmented sleep with dementia-related wandering and disruptive behaviours during the night. Rose's sleep disturbances were related to caring for Edward as well as symptoms of insomnia.

Rose was very enthusiastic about trialling something non-pharmaceutical for Edward's condition. However Edward had a fall and was admitted to hospital, at which point his dementia deteriorated and medications changed. Soon after this episode, he moved into a care facility.

Table 8.12

Actigraphic Sleep Outcomes at Time 1 Concerning PWD and Carer 15

Variable	PWD 15				Carer 15			
	Mean	(SD)	Median	(Range)	Mean	(SD)	Median	(Range)
Bedtime	21:00		20:30:00	(20:30-22:00)	23:04		23:30	(21:40-00:10)
Risetime	7:27		7:00:00	(7:00-08:20)	07:22		07:20	(07:00-08:00)
Time in bed night, minutes	626.7	(69.4)	630.0	(540.0-710.0)	498.0	(50.0)	510.0	(440.0-560.0)
Total sleep per night, minutes	342.3	(32.3)	332.0	(309.0-386.0)	433.4	(28.1)	446.0	(396.0-471.0)
Sleep efficiency (% sleep night)	55.4	(8.4)	61.3	(43.5-61.5)	87.4	(5.2)	90.0	(80.0-92.4)
Wake time night, minutes	284.3	(83.8)	244.0	(208.0-401.0)	64.6	(32.0)	44.0	(34.0-112.0)
Number of night awakenings	59.7	(11.5)	61.0	(45.0-73.0)	24.0	(6.8)	19.0	(18.0-35.0)
Total sleep per day, minutes	57.8	(23.2)	54.0	(34.0-89.0)	60.0	(0.0)	60.0	(60.0-60.0)

20 Medications Being Taken by all PWD and Carers at Time 1

Table A.1

Medications and their Sleep-Related Side Effects: Taken by the PWD Who Completed the Trial (IDs1 -9) and Those who Withdrew (IDs 10-15)

Medication	Sleep-related side effects																
	General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Cardiovascular:																	
Acebutolol																✓	
Bendroflumethiazide												✓			✓		
Cilazapril	fatigue, tiredness					✓								✓			
Dabigatran								✓									
Digoxin	Tiredness												✓				
Doxazosin †	fatigue, somnolence																✓
Duride	Tiredness		✓														
Felodipine ^	fatigue, tiredness		✓		✓								✓				
Furosemide ^	drowsiness, restlessness, lethargy				✓			✓					✓			✓	
Hyzaar	Fatigue				✓												
Inhibace Plus †	Fatigue										✓						
Isosorbide mononitrate								✓									
Lipitor	Tiredness, weakness, insomnia		✓		✓												
Metoprolol	fatigue, tiredness	insomnia, nightmares	✓		✓	✓		✓			✓		✓				✓
Quinapril or Accupril	tiredness or weakness, drowsiness							✓					✓				
Simvastatin	fatigue insomnia	Nightmares				✓		✓			✓						
Terazosin	Somnolence			✓							✓						
Warfarin					✓											✓	

^ rarely has side effects associated with photosensitivity † possible side effects associated with photosensitivity

Table A.1 Continued. (PWD medications)

Medication	Sleep-related side effects: General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pain/inflammation relief:																	
Asprin			✓	✓		✓					✓						
Panadol/paracetamol														✓			✓
Prednisone	insomnia, hallucinations						✓										
Mood, cognition or behaviour:																	
Aricept/donepezil	drowsiness, fatigue, tiredness, insomnia					✓	✓		✓							✓	
Sinemet	somnolence, sudden sleep onset episodes, hallucinations, nightmares						✓										
Tegretol ^	fatigue, drowsiness																✓
Venlafaxine †	insomnia, nightmares, teeth grinding, drowsiness, fatigue		✓														
Zopiclone	sleepiness, disrupted sleep architecture									✓						✓	
Hormonal:																	
Levothyroxine		Insomnia												✓	✓	✓	

^ rarely has side effects associated with photosensitivity † possible side effects associated with photosensitivity

Table A.1 Continued (PWD medications)

Medication	Sleep-related side effects: General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Other																	
Cetirizine	Fatigue	somnolence, insomnia						✓									
Flixotide inhaler		disturbed sleep		✓													
Folic acid			✓														
Laxative							✓							✓			
Loperamide		tiredness										✓					
Loratadine		fatigue					✓										
Metamucil										✓							
Omeprazole ^		somnolence, insomnia						✓			✓			✓			
Total			7	3	6	5	5	8	1	2	6	2	5	6	3	6	4

^ Rarely has side effects associated with photosensitivity, † possible side effects associated with photosensitivity

Table A.2

Medications and their Sleep-Related Side Effects: Taken by the Carers Who Completed the Trial (IDs1-9) and Those who Withdrew (IDs 10-15)

	Sleep-related side effects																	
Medication	General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Cardiovascular:																		
Amlodipine			✓															
Atenolol	fatigue	nightmares				✓												
Atorvastatin	fatigue, insomnia														✓			
Bendroflumethiazide			✓			✓												
Cilazapril	fatigue, light headedness, tiredness, weakness				✓													
Digoxin	Tiredness														✓			
Duride	Tiredness														✓			
Felodipine	fatigue and tiredness																	
Furosemide	drowsiness, restlessness, lethargy														✓			
Metoprolol	tiredness, fatigue	insomnia, nightmares	✓		✓										✓			
Quinapril or Accupril	tiredness, weakness, drowsiness		✓															
Simvastatin	fatigue, insomnia	nightmares			✓													

Table A.2 Continued (Carer Medications)

			Sleep-related side effects														
Medication	General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pain/inflammation:																	
Asprin					✓												
Celebrex	Fatigue											✓					
Diclofenac		drowsiness, insomnia and nightmares					✓										
Panadol/paracetamol																	
Prednisone	insomnia, hallucinations			✓											✓		
Mood, cognition or behaviour:																	
Clonazepam	somnolence, transient fatigue, sleep disturbances, nightmares												✓				
Epilim	Drowsiness		✓														
Venlafaxine	insomnia, nightmares, teeth grinding, drowsiness, fatigue				✓										✓		
Zopiclone	sleepiness, disrupted sleep architecture															✓	
Hormonal:																	
Calcitriol	somnolence – acute														✓		
Levothyroxine		insomnia									✓						
Remifemin							✓										
Tamoxifen																✓	
Thyroxin		insomnia														✓	

Table A.2 Continued (Carer Medications)

		Sleep-related side effects															
Medication	General	Less common	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Other:																	
folic acid															✓		
Losec		somnolence, insomnia											✓				
Metformin hydrochloride		somnolence													✓		
Methotrexate	fatigue, drowsiness														✓		
Omeprazole		somnolence, insomnia													✓		
Telfast	drowsiness, fatigue	insomnia, sleep disorders					✓										
Total			5	1	5	2	3	0	0	0	1	1	2	0	12	3	0

21 Actograms Concerning Pairs who Completed the Trial

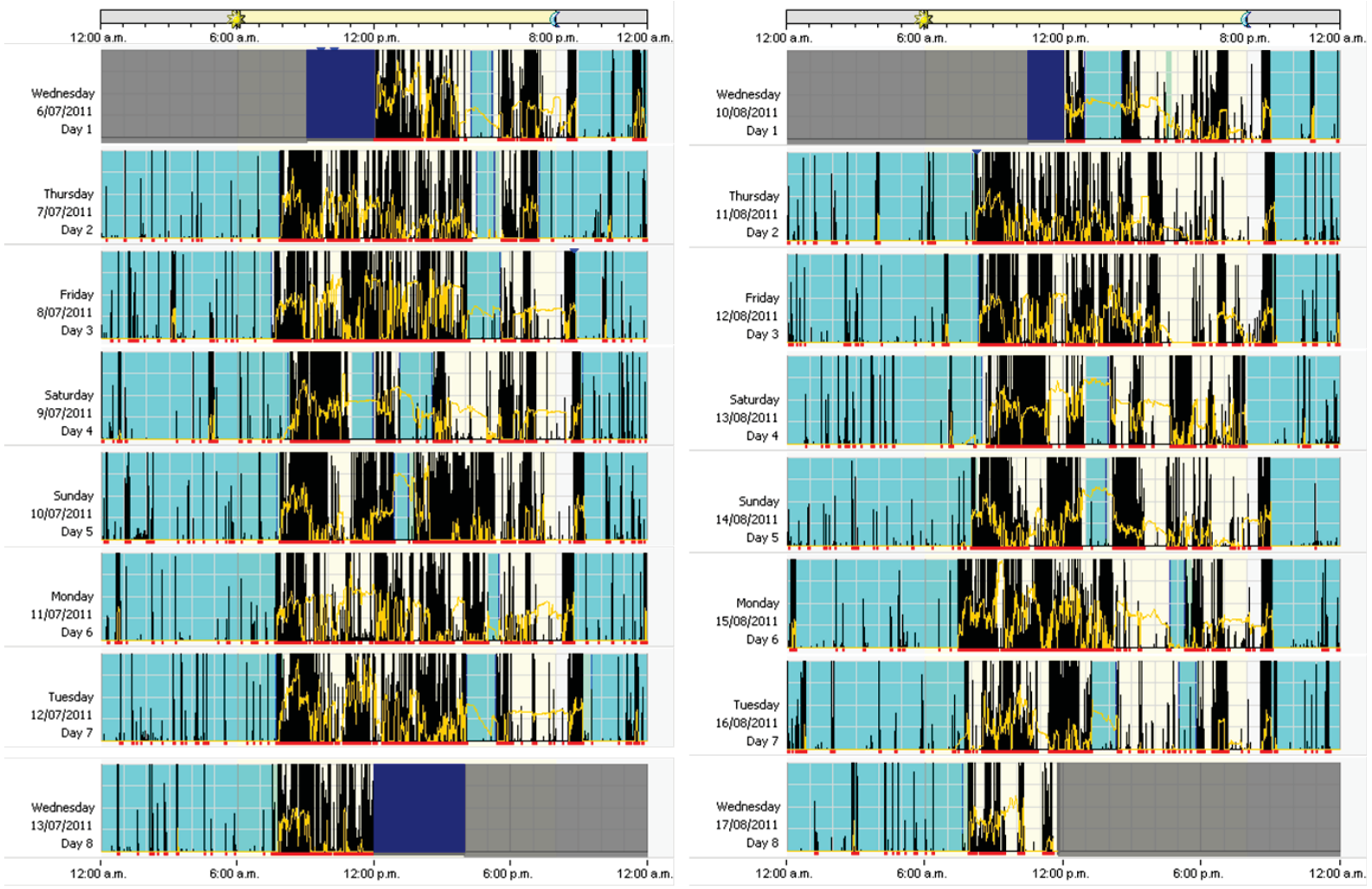

Figure A.8. Activity and light exposure for PWD 1 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux).

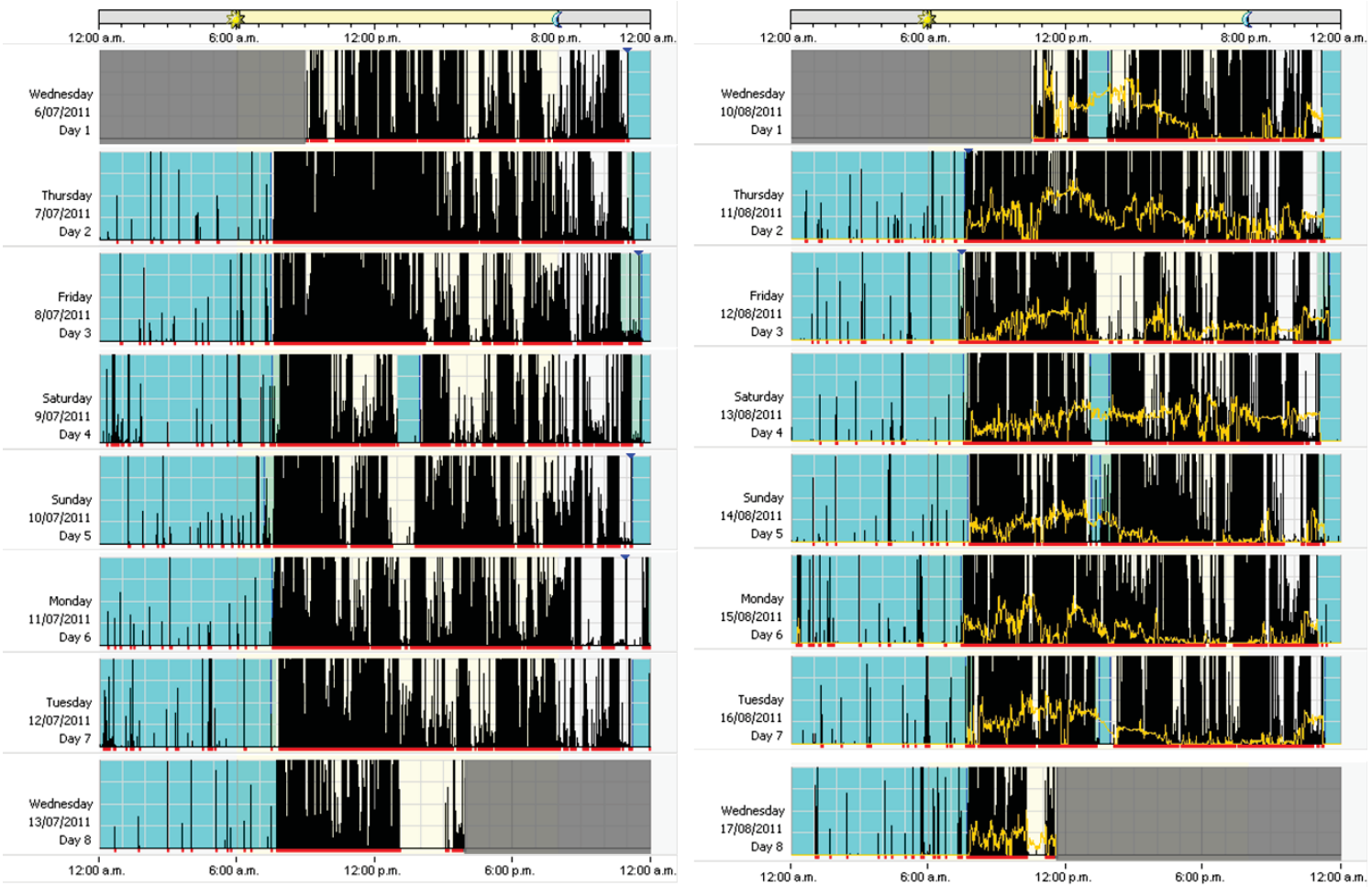

Figure A.9. Activity and light exposure for carer 1 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux) (missing light information due to equipment failure).

▼: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

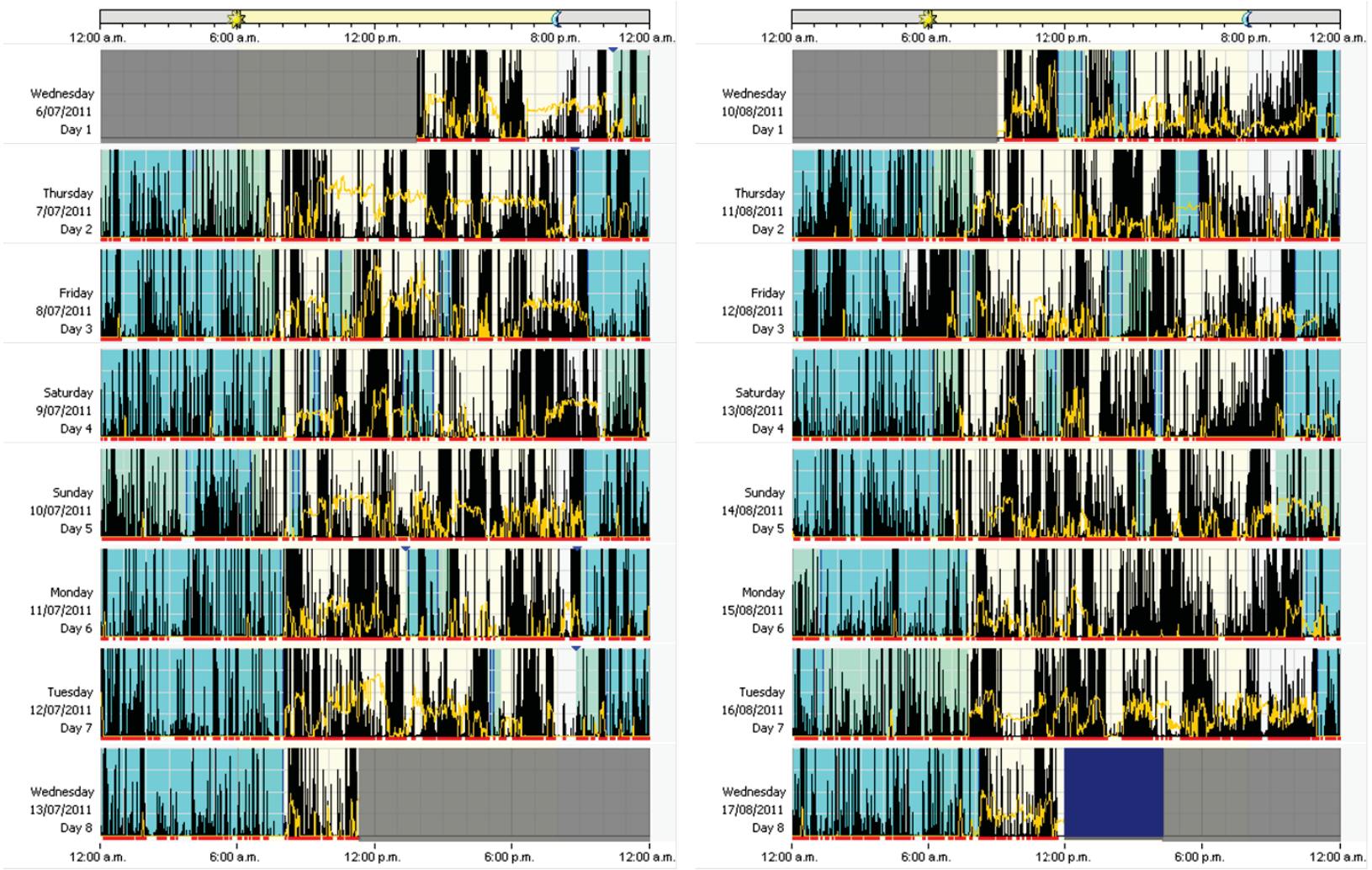

Figure A. 10. Activity and light exposure for PWD 2 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux).

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬: wake, ▮: Light

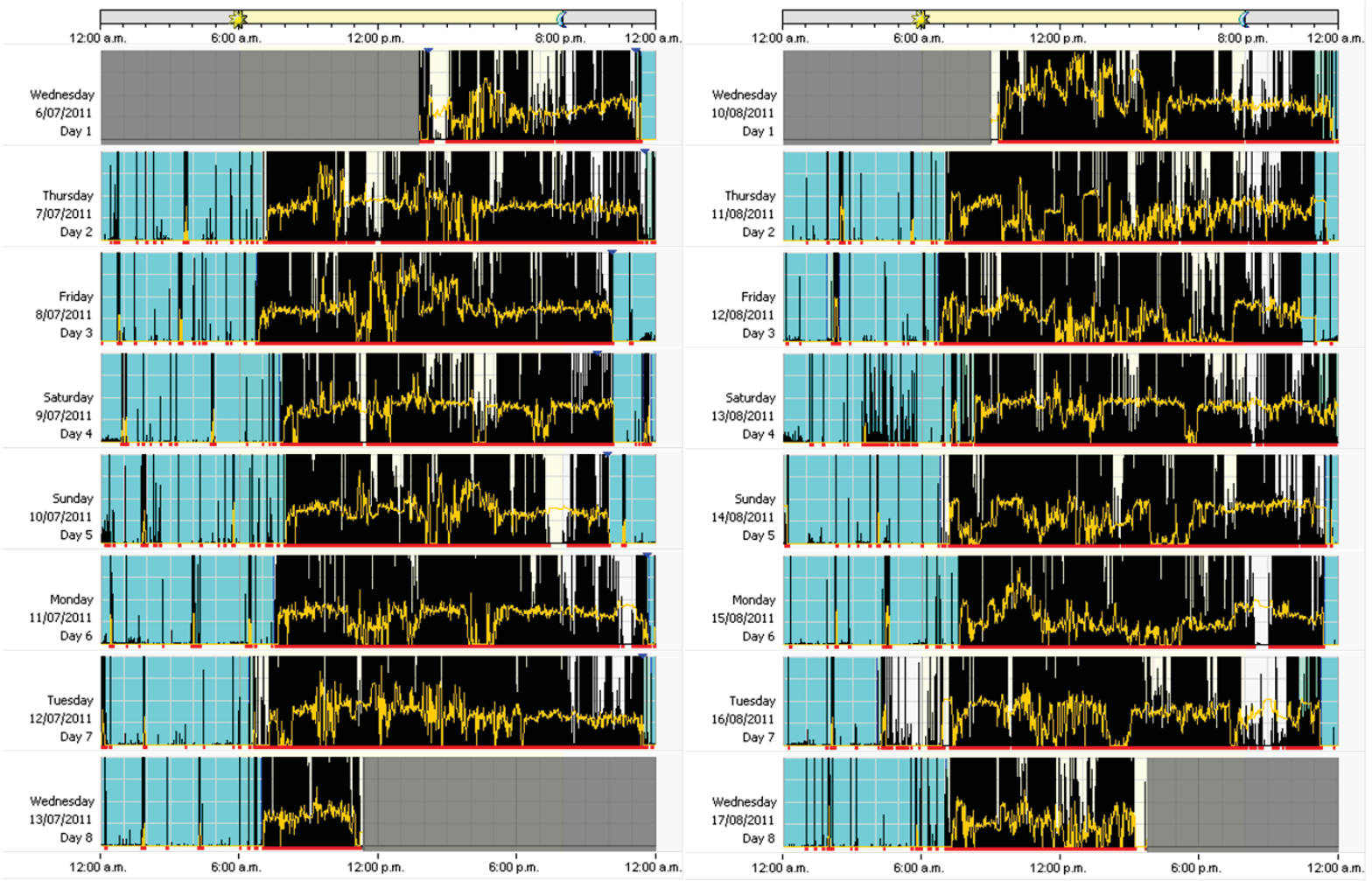

Figure A.11. Activity and light exposure for carer 2 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux).

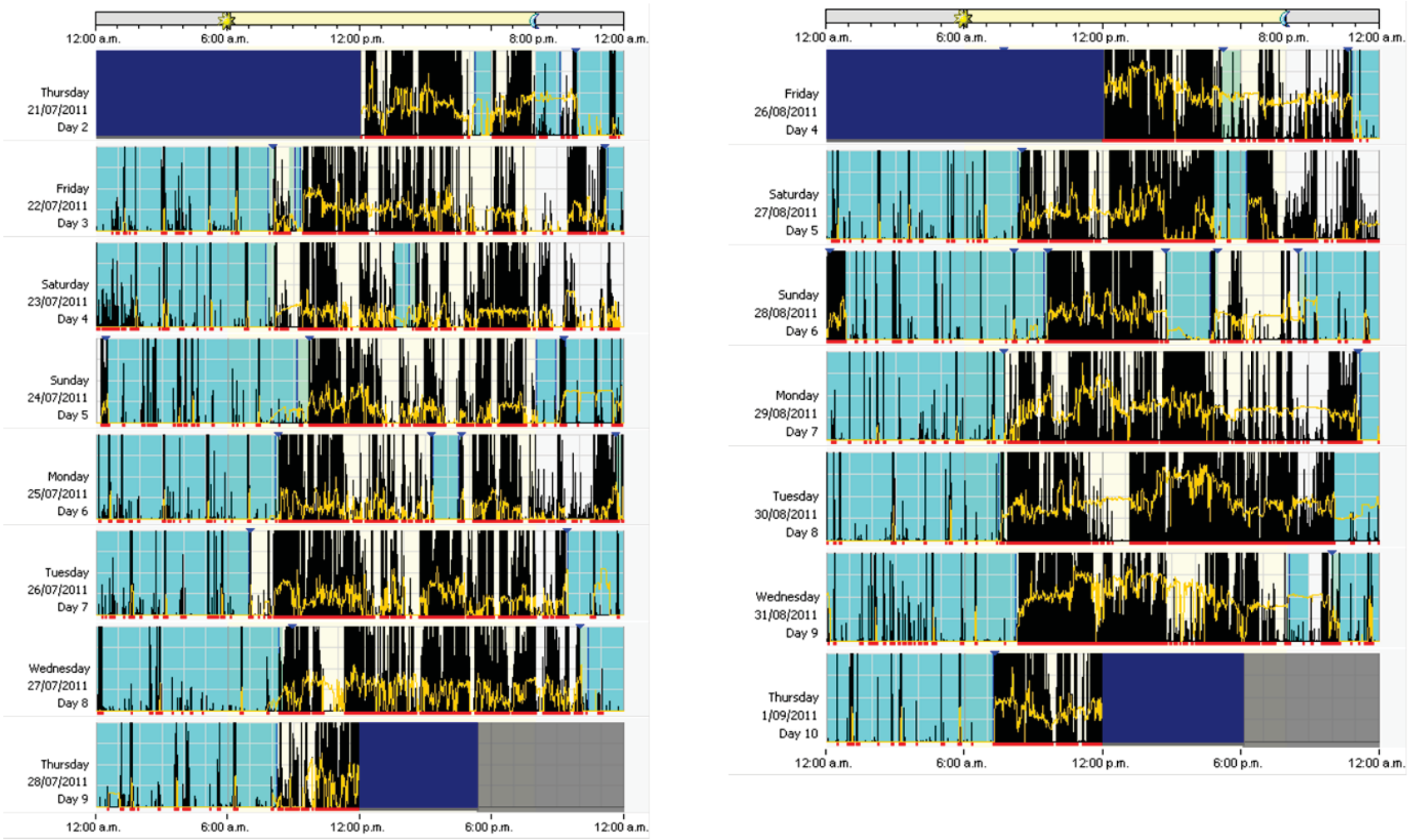

Figure A.12. Activity and light exposure for PWD 3 at times 1 (left, 7 days) and 2 (right, 6 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux.

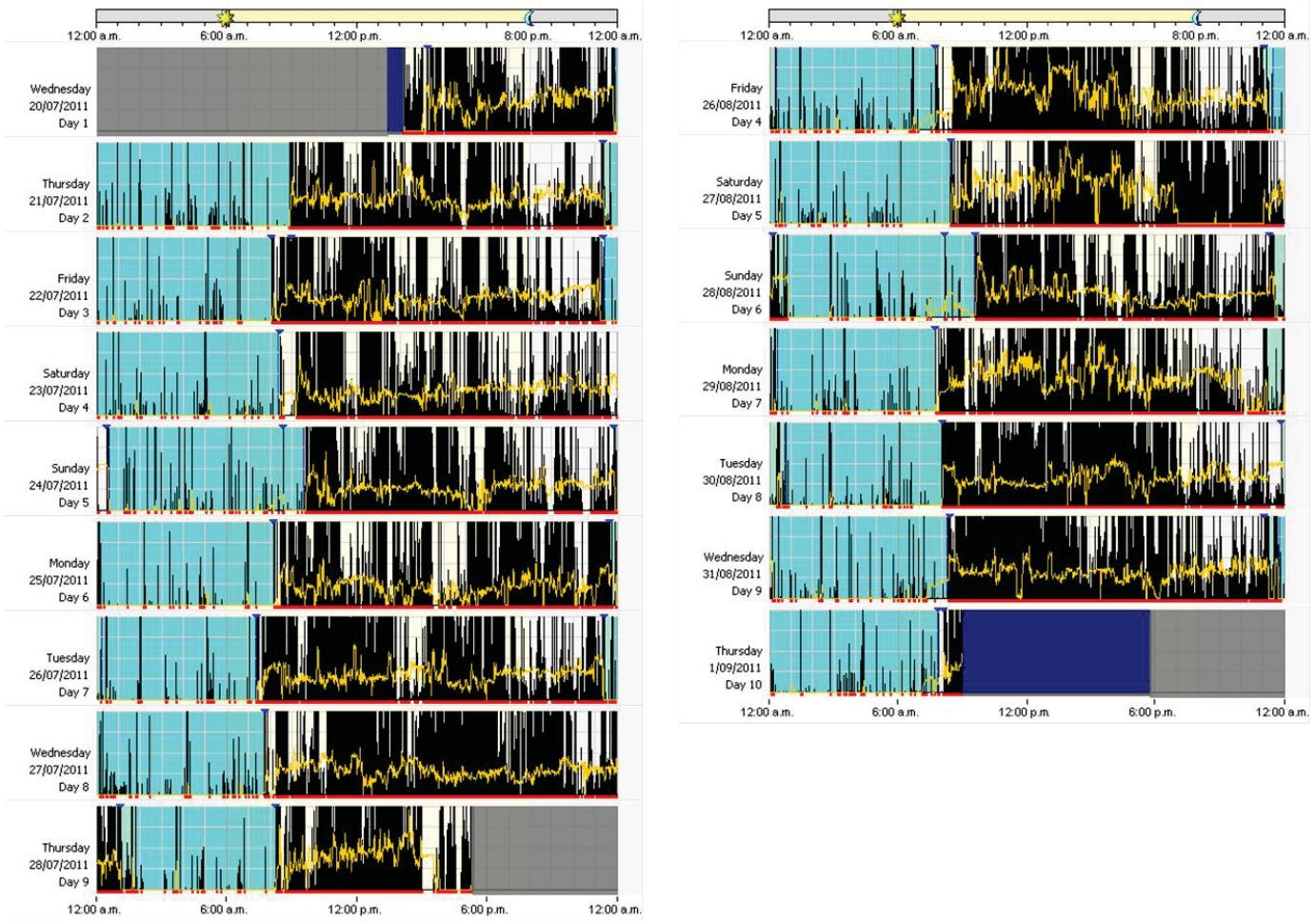

Figure A. 13. Activity and light exposure for carer 3 at times 1 (left, 8 days) and 2 (right, 6 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0 lux.

: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

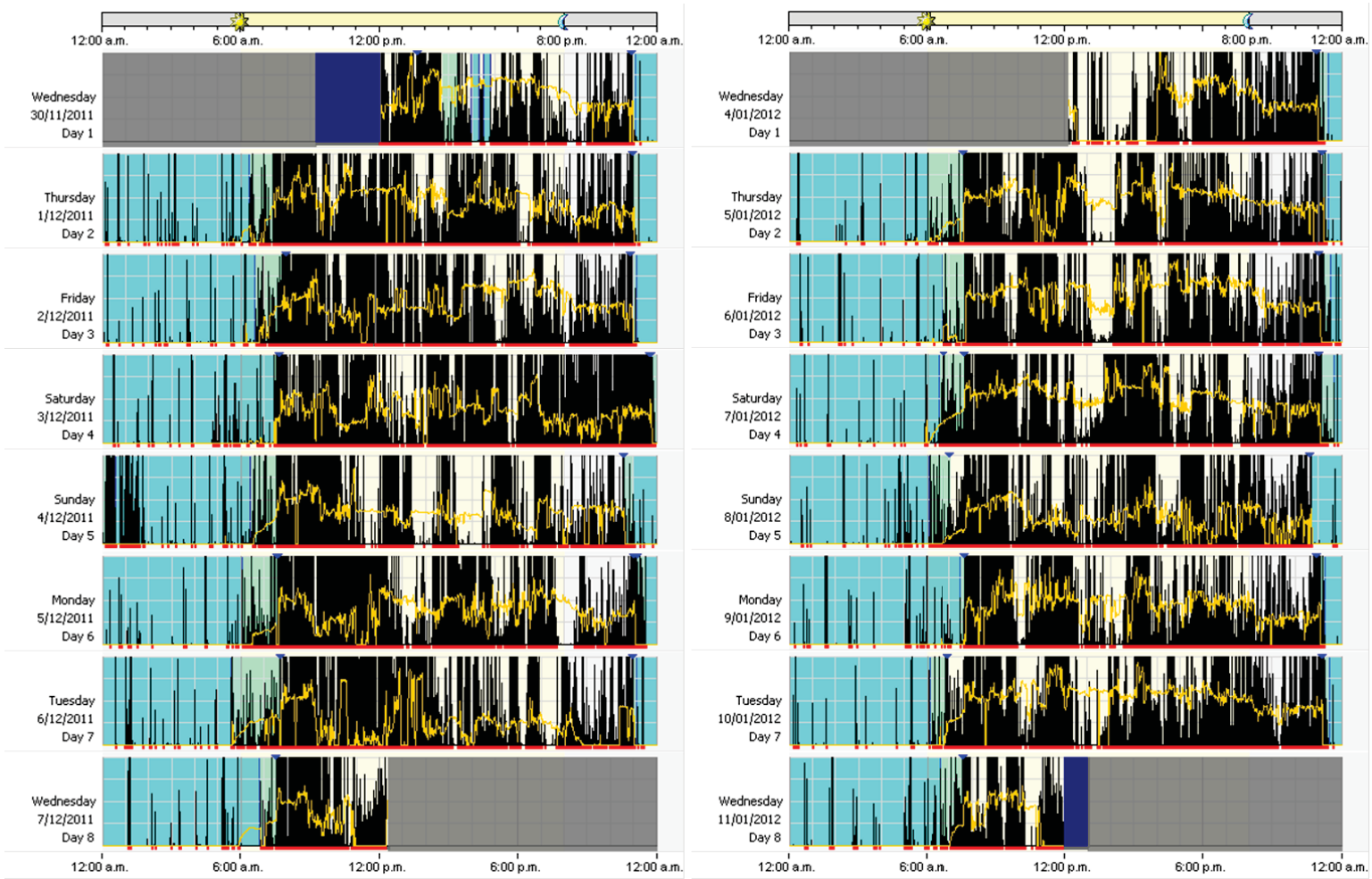

Figure A.14. Activity and light exposure for PWD 4 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

▼: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

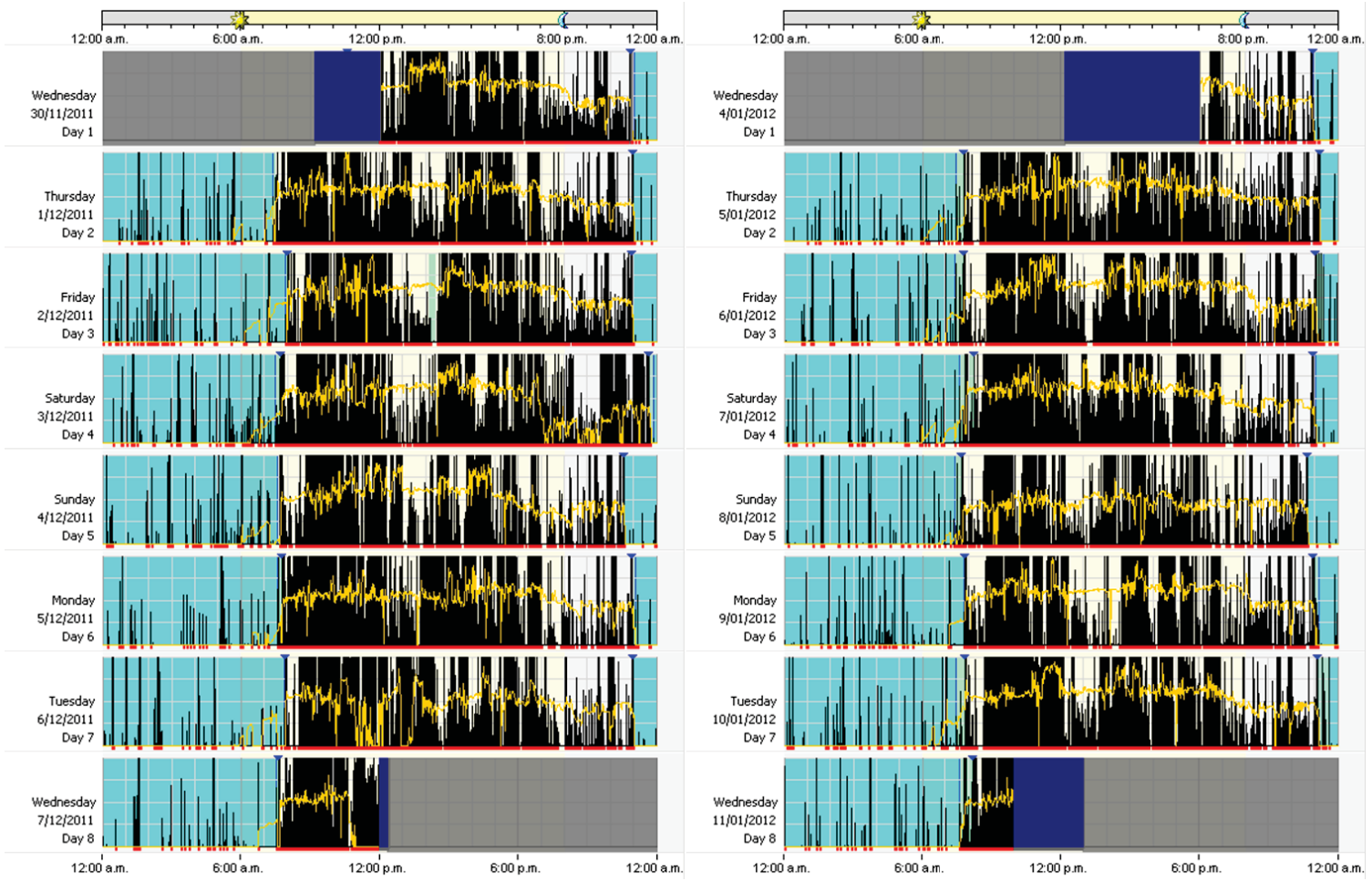

Figure A.15. Activity and light exposure for carer 4 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬: wake, ▮: Light

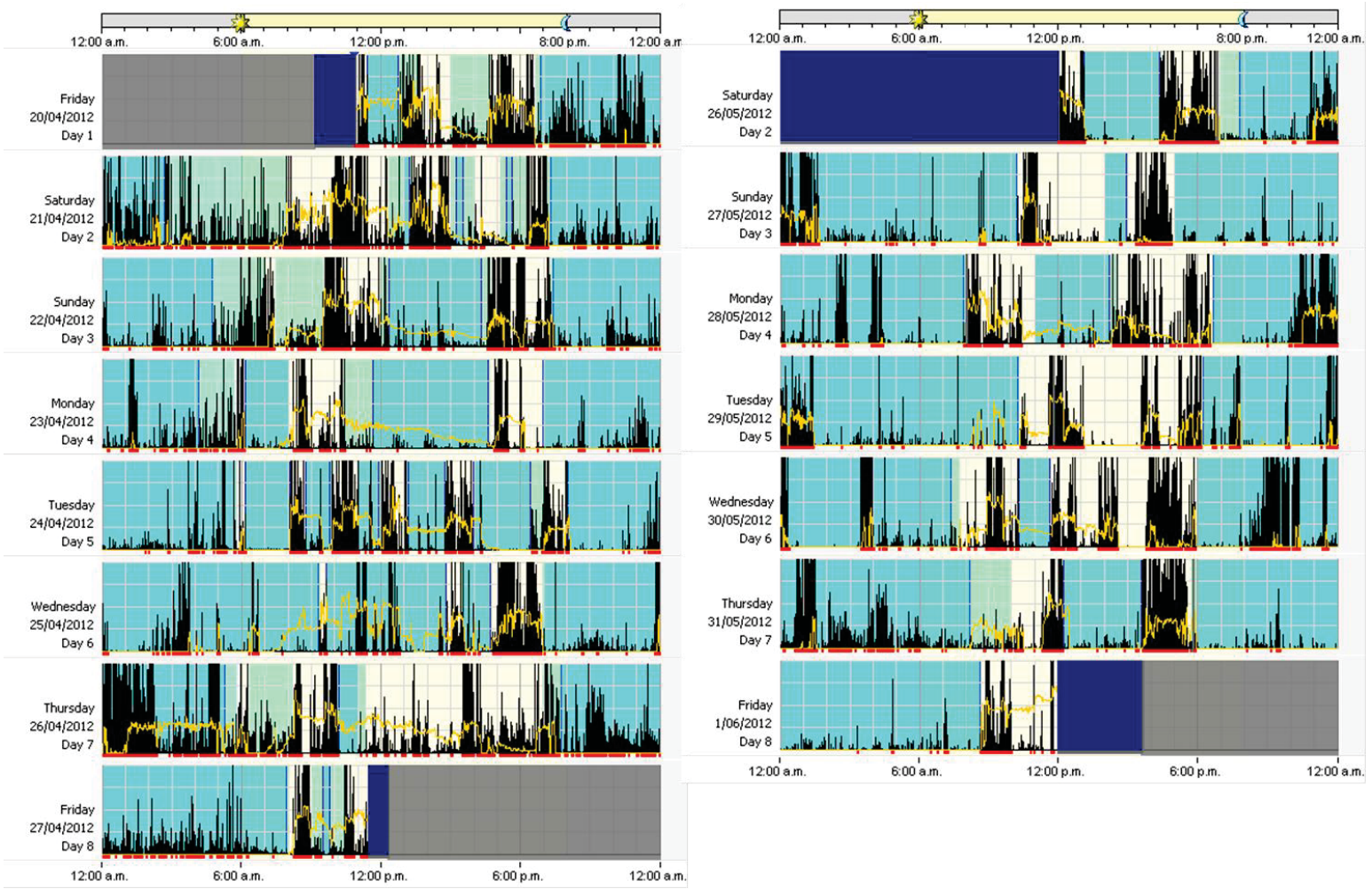

Figure A. 16. Activity and light exposure for PWD 5 at times 1 (left) and 2 (right),7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

▼: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

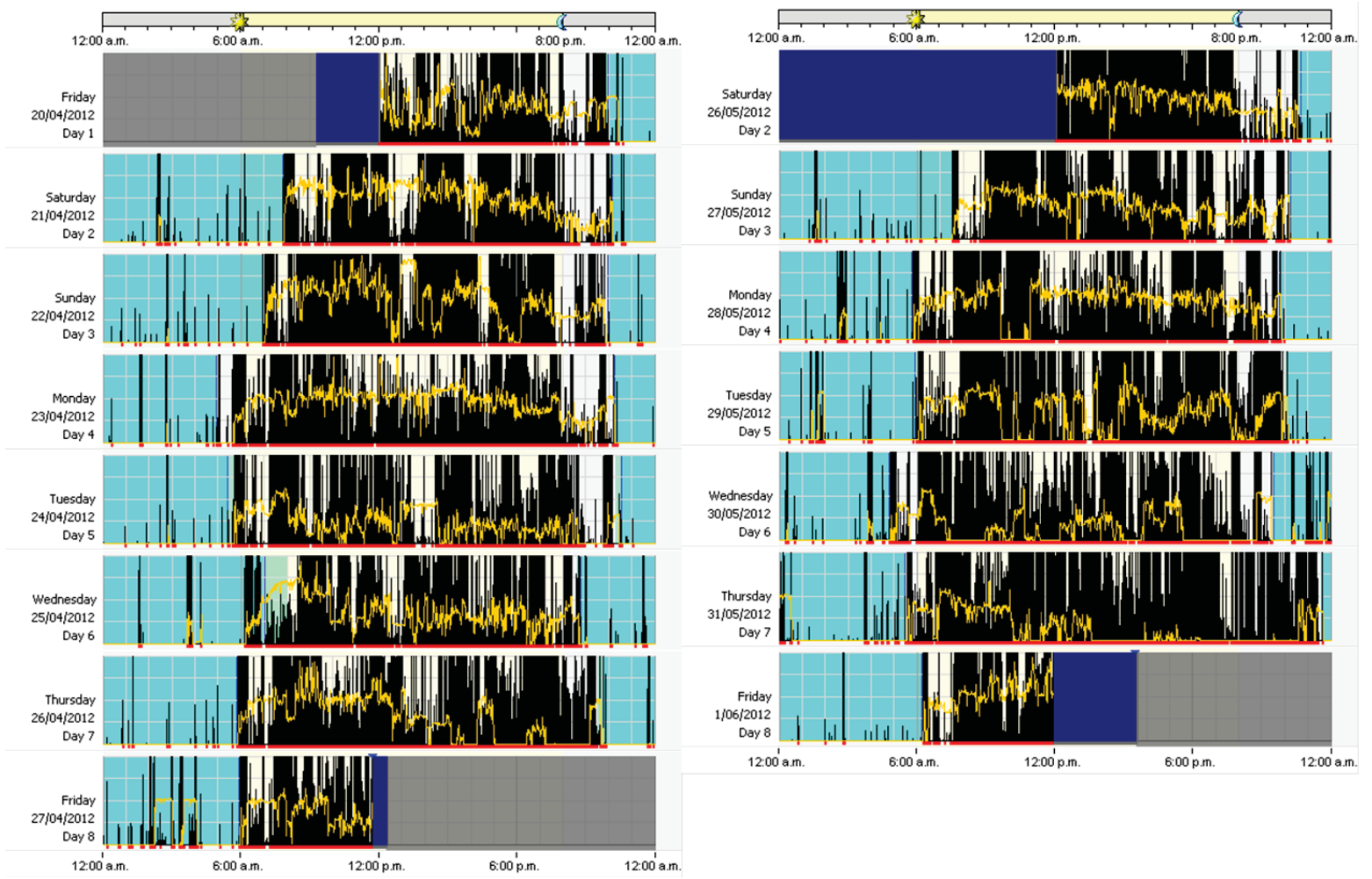

Figure A.17. Activity and light exposure for carer 5 at times 1 (left, 7 days) and 2 (right, 6 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

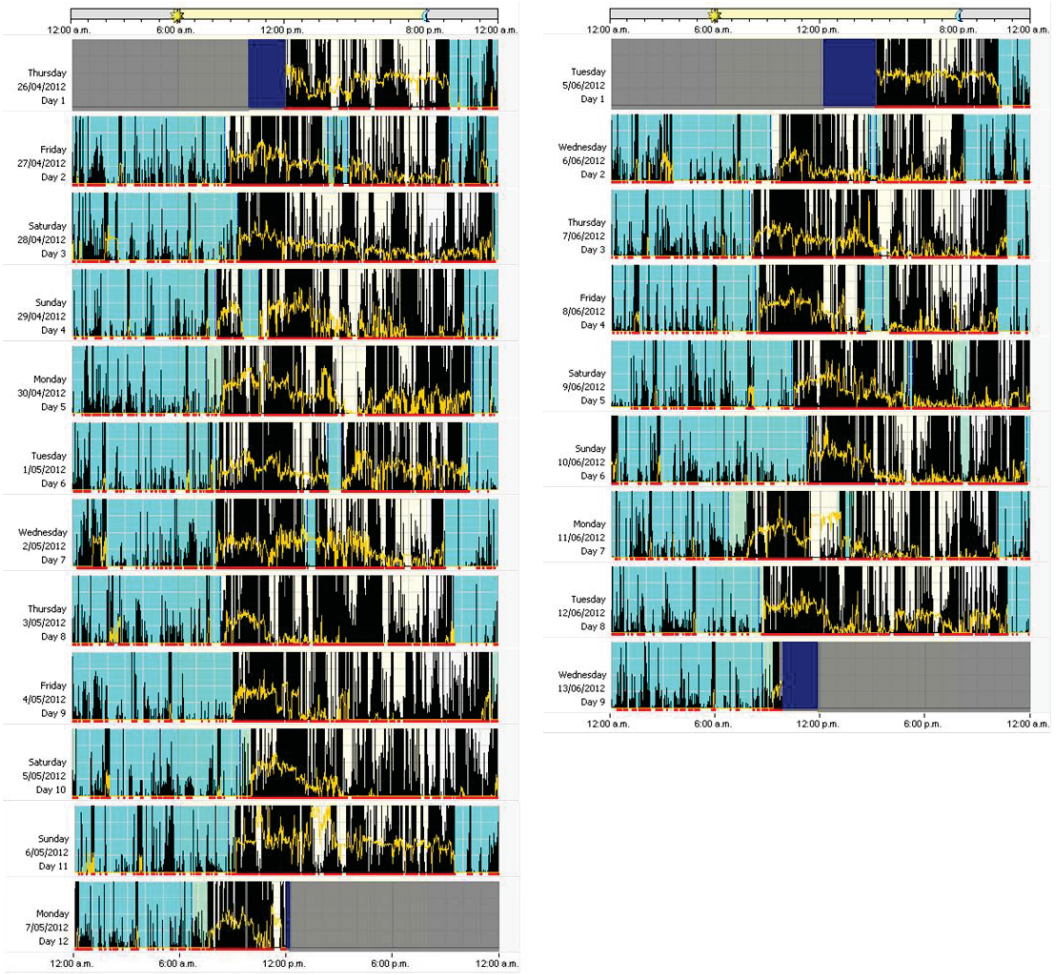

Figure A.18. Activity and light exposure for PWD 6 at times 1 (left, 11 days) and 2 (right, 8 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0.

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬ ▬: wake, ▮: Light

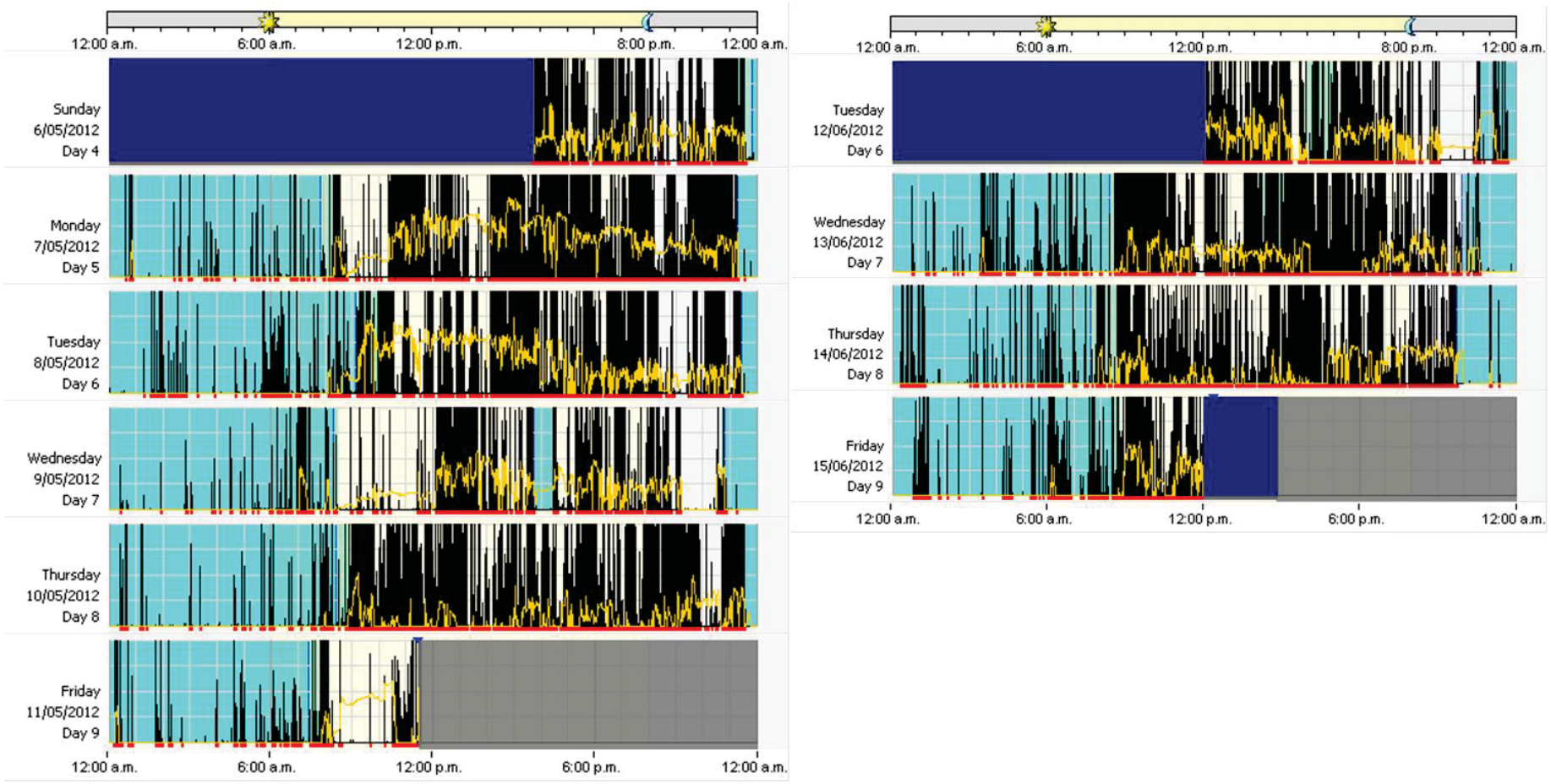

Figure A.19. Activity and light exposure for PWD 7 at times 1 (left, 5 days) and 2 (right, 3 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0.

▼: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

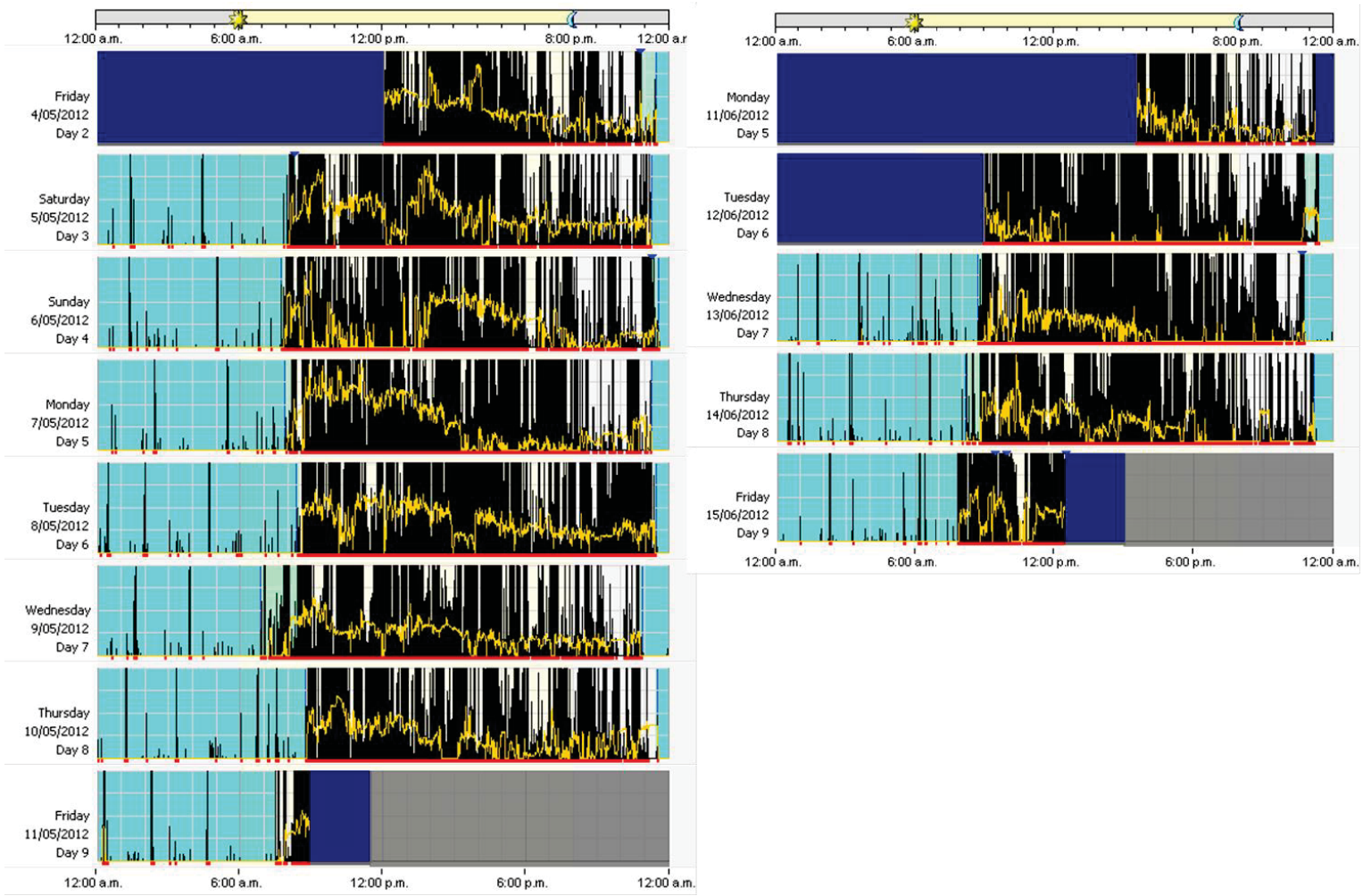

Figure A.20. Activity and light exposure for carer 7 at times 1 (left, 7 days) and 2 (right, 3 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0.

▼: Event marker, : Rest interval, : Sleep interval, : activity, : wake, : Light

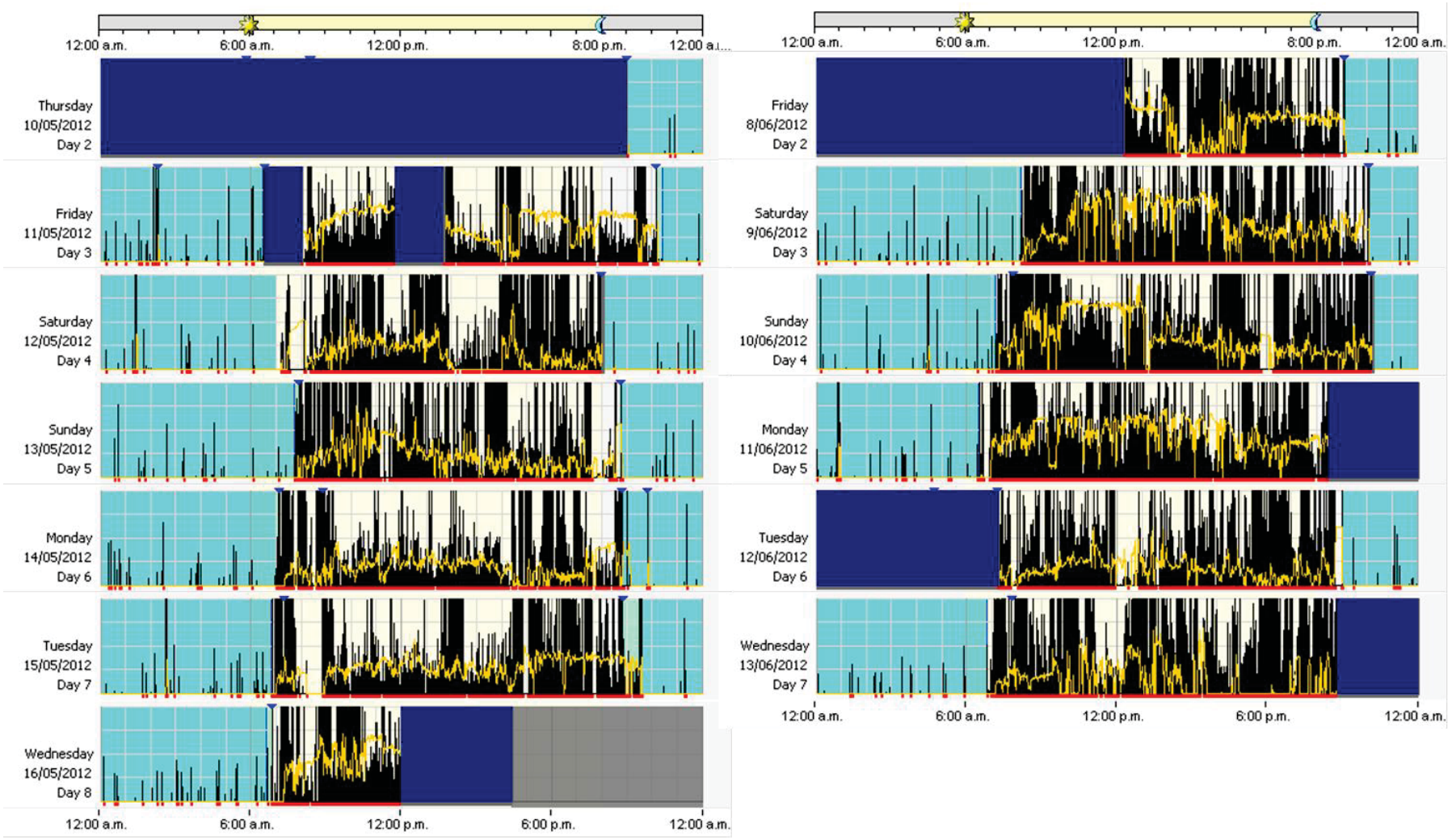

Figure A.21. Activity and light exposure for PWD 8 at times 1 (left, 6 days) and 2 (right, 4 days), activity scale: 0-200 counts, white light scale: 0.01–50,000.0.

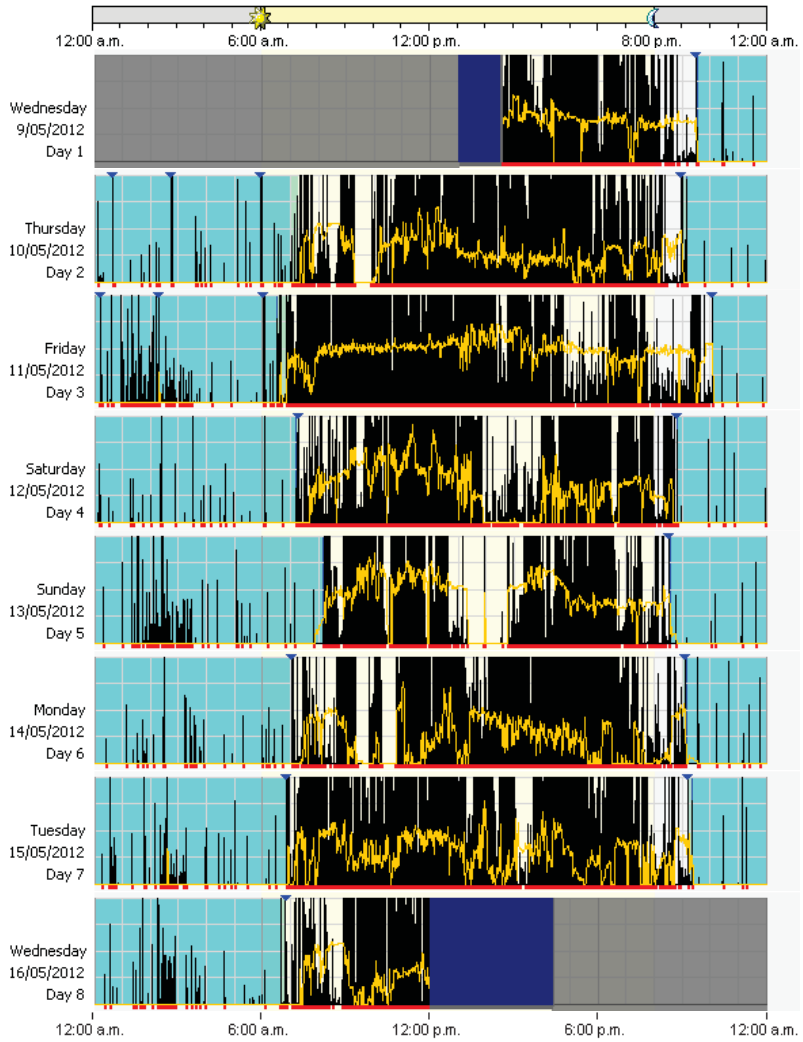

Figure A.22. Activity and light exposure for carer 8 at time 1 (7 days, no Time 2 data available), activity scale: 0-200 counts, white light scale: 0.01–50,000.0.

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬: wake, ▮: Light

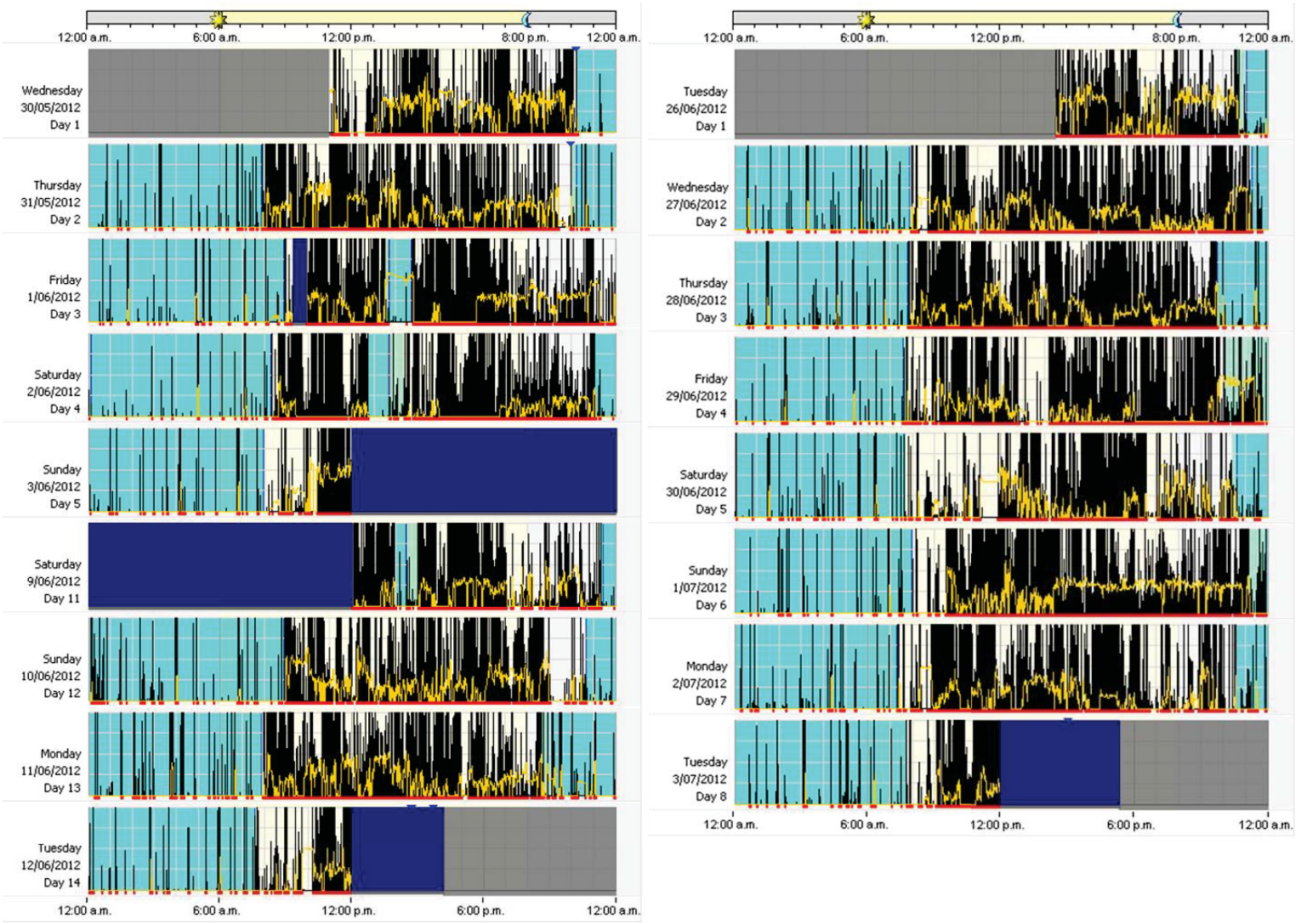

Figure A.23. Activity and light exposure of PWD 9 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬: wake, ▮: Light

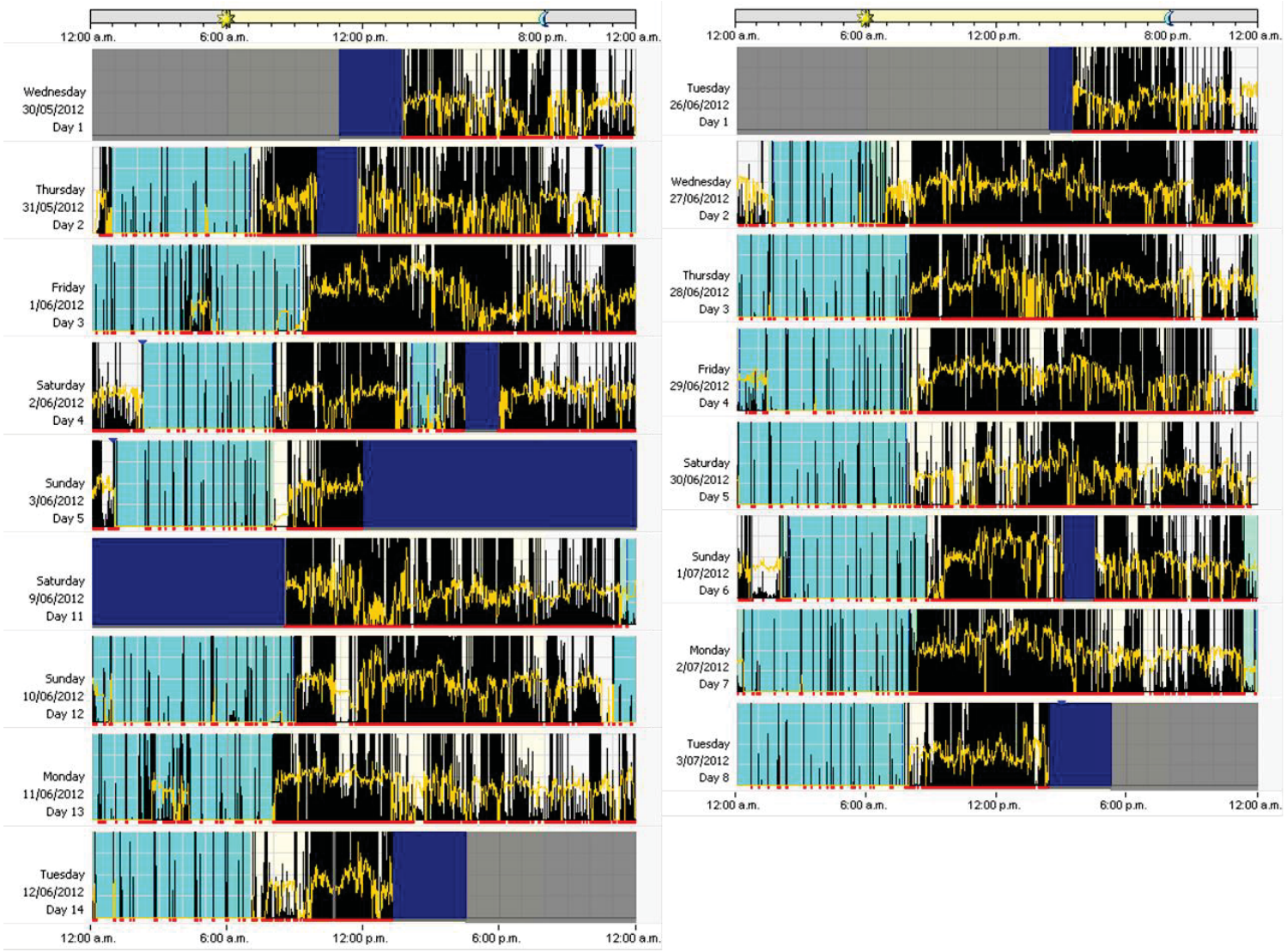

Figure A.24. Activity and light exposure of carer 9 at times 1 (left) and 2 (right), 7 days (activity scale: 0-200 counts, white light scale: 0.01–50,000.0).

▼: Event marker, ▭: Rest interval, ▬: Sleep interval, ▮: activity, ▬ ▬: wake, ▮: Light

22 Additional Results Concerning Pairs who Completed the Trial

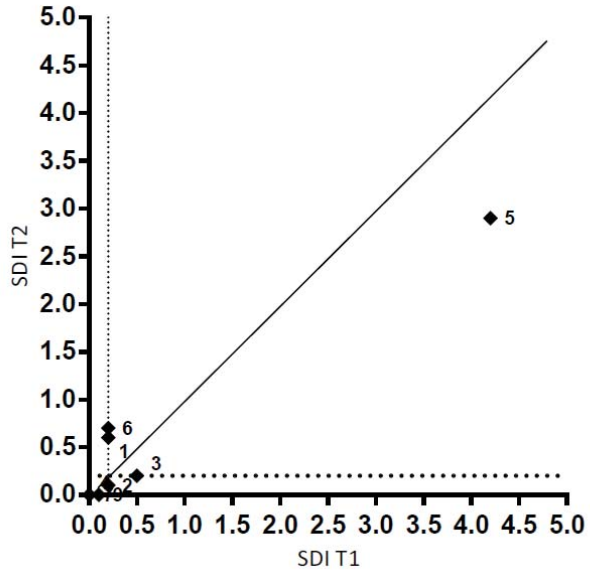

Figure A.25. Comparison of global SDI scores of PWD between Times 1 and 2.

Including line of no change, higher scores indicate greater sleep disturbance, group median at Time 1 = 0.2 (dotted lines). Note PWD 8's SDI data was missing.

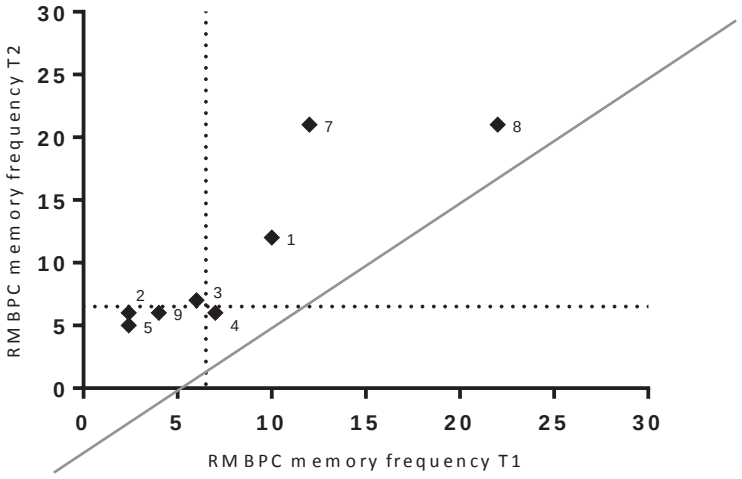

Figure A.26. Comparison of RMBPC scores related to the carers reaction to memory-related behaviours of PWD between times 1 and 2.

Including line of no change. Higher scores indicate greater reaction, group median at time 1 = 6.5 (dotted lines). Note no data is presented for carer 6 due to missing data.

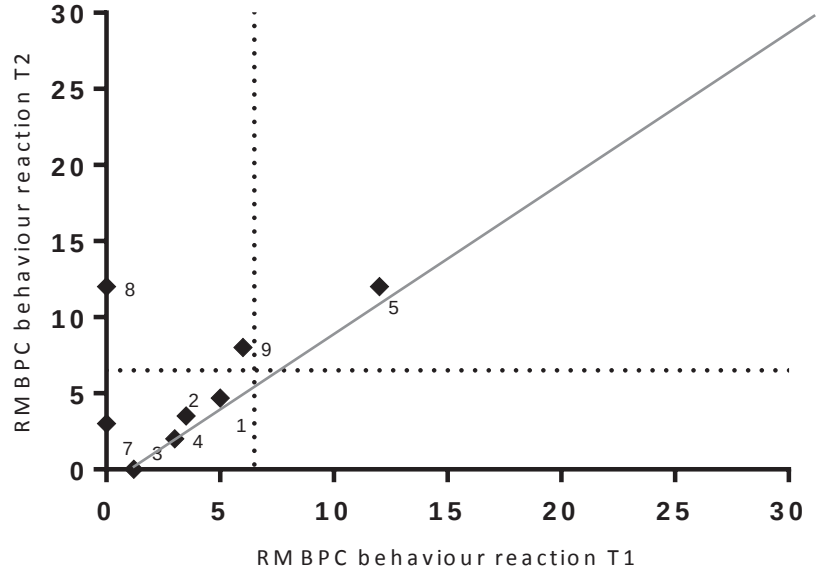

Figure A.27. Comparison of RMBPC scores related to the carers reaction to dementia-related behaviours of PWD between times 1 and 2.

Including line of no change. Higher scores indicate greater reaction, group median at time 1 = 3.5 (dotted lines). Note no data is presented for carer 6 due to missing data.

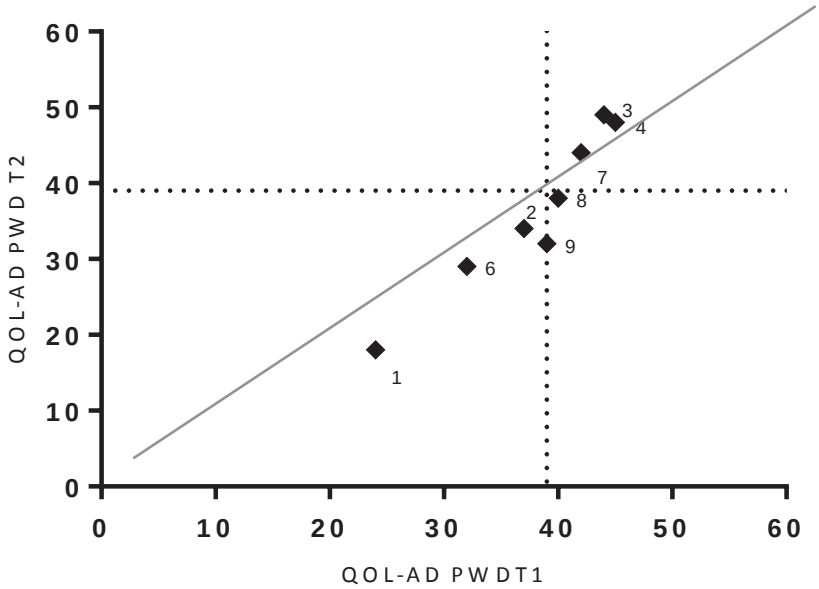

Figure A.28. Comparison of QOL-AD scores as rated by the PWD between times 1 and 2.

Including line of no change. Higher scores indicate greater QOL, group median at time 1 = 39 (dotted lines). Note no data is presented for PWD 5 due to missing data.

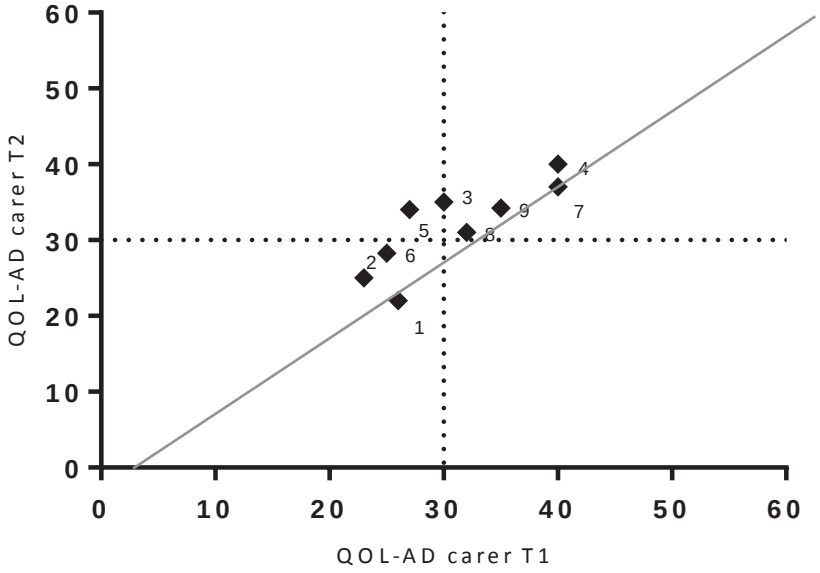

Figure A.29. Comparison of QOL-AD scores as rated by the carers between times 1 and 2.

Including line of no change. Higher scores indicate greater QOL, group median at time 1 = 30 (dotted lines).

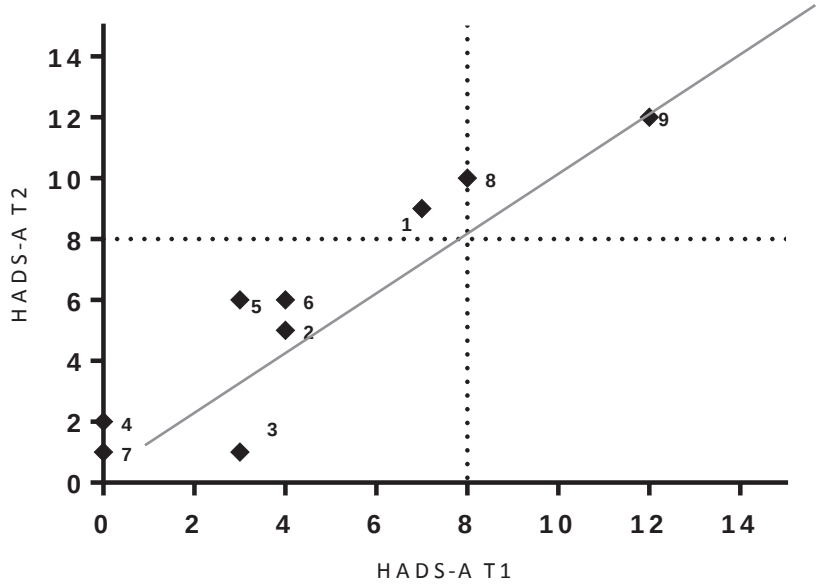

Figure A.30. Comparison of HADS-anxiety scores of carers between times 1 and 2.

Including line of no change. Scores ≥8 = increased likelihood for anxiety (dotted lines).

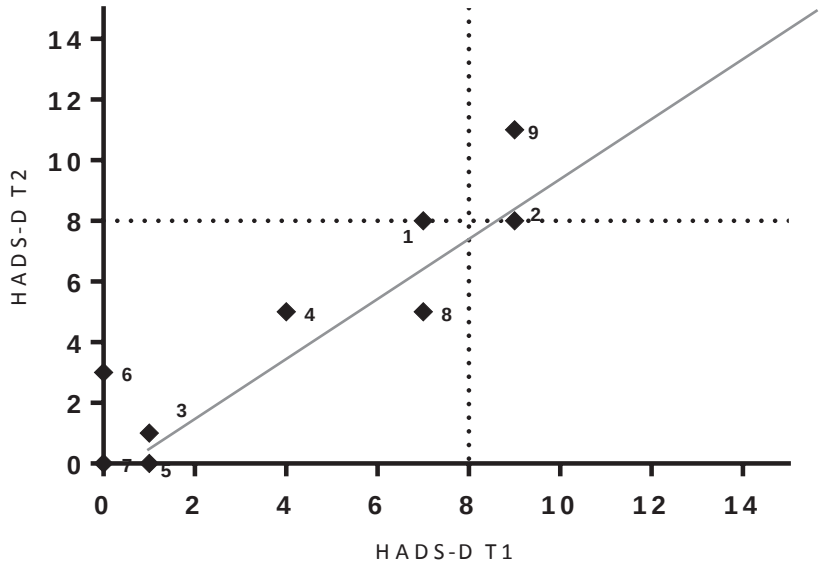

Figure A.31. Comparison of HADS-depression scores of carers between times 1 and 2.

Including line of no change. Scores ≥8 = increased likelihood for depression (dotted lines).

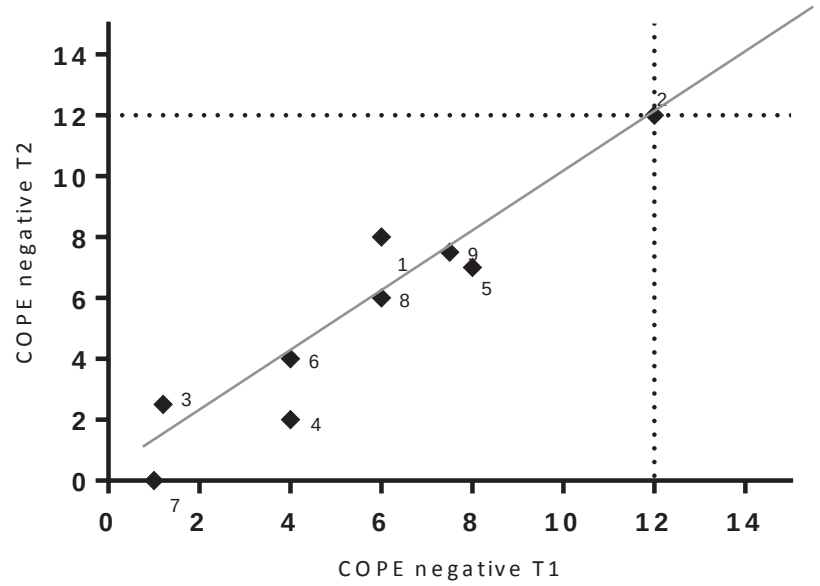

Figure A.32. Comparison of negative COPE scores of carers between times 1 and 2.

Including line of no change. Scores >12 = increased likelihood for carer burden (dotted lines).

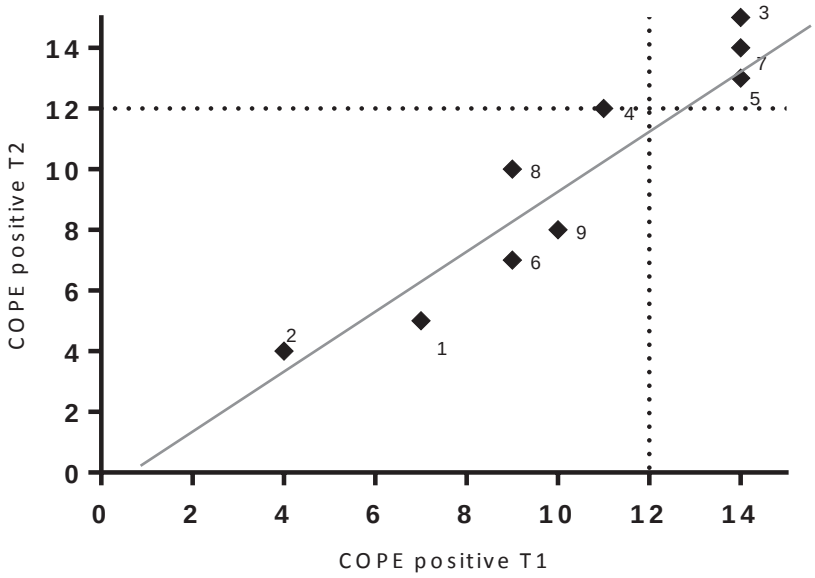

Figure A.33. Comparison of negative COPE scores of carers between times 1 and 2.

Including line of no change. Scores <12 = increased likelihood for carer burden (dotted lines).