Traumatic Injury and Dementia in New Zealand: A Palmerston North Hospital Case-Control Study

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Virginia Westerberg
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Abstract

Little is known about the relationship between traumatic injury (TI) and dementia. The increasing prevalence of both conditions in the world and in New Zealand (NZ) drove the Author to want to investigate whether the pathophysiological consequences of major trauma of any kind - mostly due to falls in the dementia population - and not just traumatic brain injury (TBI), may result in dementia.

Both TI and dementia constitute major health and socio-economic problems contributing to long-term disability worldwide and have important implications for health service delivery and for medico-legal compensation issues. The first specific objective was to determine whether dementia was associated with an increased risk and incidence of trauma in the past and whether such an association might be explained by the injuries or by medical comorbidities. The second specific objective was to identify whether there were any differences in the mechanisms of injury and type of discharge from hospital between cases and controls. The research was a non-experimental, retrospective, hospital-based, case-control study. Cases and controls were selected from the Palmerston North Hospital (PNH) acute admissions database and were matched in terms of exposure to traumatic injury, sex, age, ethnicity, and recorded comordibites. Statistical and epidemiological analyses were done using Raosoft^{R} and MedCalc^{R} softwares.

All medical conditions were operationally defined using the current World Health Organization's International Classification of Diseases (ICD-10). The results showed that a history of TI was more frequently found in cases with dementia than in the controls. Patients with dementia and TI were more likely to have preexisting comorbidities and were more unlikely to be discharged to their previous habitual residence. The findings strongly indicate that the brain is affected by the way the body responds to TI both locally and systemically. The conclusion was that the direct and indirect consequences of TI, mostly due to falls, could constitute a plausible risk factor for the development or progression of dementia but that further research is needed to assess what type of trauma and what type of dementia could be involved in the association, one that is likely to be multifactorial in the elderly population.

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Chapter 1. An Overview of the Topics

The aim of this chapter is to briefly explain the title of this work outlining concepts that will be considered in more detail in subsequent chapters. In other words, chapter one deals with the operational definitions of traumatic injury, falls, dementia, and the New Zealand Central Region to which Palmerston North belongs.

1.1. Traumatic Injury

The World Health Organization (WHO) (2013) classifies human causes of death in three groups: Group I: Communicable diseases, Group II: Non-communicable diseases, and Group III: Injuries. Non-communicable diseases are the leading cause of death in developed countries, followed by injuries. Injuries – also known as traumatic injuries (TI) or simply trauma- include blunt trauma, motor vehicle accidents, and falls. The definition of injury in this work is based on that of the Australian Modification of the 10th revision of the WHO's International Classification of Diseases and Related Health Problems (ICD) (WHO, 2013). The ICD codes are used by the NZ Ministry of Health (MOH) and its affiliate institutions to code mortality and hospitalisation data (MOH, 2013).

The Global Burden Disease - a collaboration among the WHO, the World Bank and the Harvard School of Public Health - latest report shows that over 10% of the estimated 60 million deaths in the world were due to some form of traumatic injury (Institute for Health Metrics and Evaluation, 2013). However, it should be noted that because the WHO cannot possibly take into account wars or natural disasters in their predictions,

the figures may consistently be an underestimate of the actual number of deaths by traumatic injuries.

Traumatic injury is any form of anatomical damage caused by an external force (WHO, 2004). Regardless of gender, ethnicity, or socio-economic status, traumatic injury is a leading cause of death worldwide, accounting for almost 6 million of them each year. Unintentional and intentional traumatic injuries are, respectively, the fifth and seventh causes of death in the world. However, deaths due to trauma are only part of the problem as millions more are left with the effects of disabilities for the rest of their lives (WHO, 2009). Traumatic injury may be classified by severity and location but it may also be classified by demographic data (such as age, gender, and ethnicity) or by the type of force responsible for the trauma (blunt, blast, penetrating) (Marino, 1998). Clinically, and for research and data analysis purposes, injury can also be classified using the Barell matrix, which is based on the WHO's International Classification of Diseases (ICD). This diagnostic classification results from the International Collaborative Effort on traumatic injury statistics. The purpose of the matrix is to standardise the classification of trauma so that the international healthcare and research community are on the same page when describing the topic (Centre for Disease Control and Prevention [CDC], 2013).

1.1.1. Falls.

The WHO (2012) defines a fall as an event resulting in an individual unwillingly coming to rest on the ground, floor, or other lower level. Although fall-related injuries can be fatal they are most often non-fatal (Stewart & Allen, 2007). Among older adults, the leading cause of traumatic injury is falls. With an aging world population, both the

number of falls and the cost to treat fall-related traumatic injuries are likely to increase (WHO, 2012). In NZ one in three adults aged 65 and older falls each year (ACC, 2014). Of those who fall, up to one third sustain moderate to severe injuries that permanently limit their ability to move about or live independently, thus increasing their risk of an early death. Older adults are hospitalised for fall-related injuries five times more often than they are for injuries from other causes (WHO, 2012). In NZ almost two million injuries were reported in 2012 (ACC, 2014), half of which happened in a residential setting.

Residential-related trauma is almost three times higher than that of sports-related injuries, over five times that of work-related injuries, and 34 times the number of traffic accident injuries nationwide (ACC, 2014). Traumatic injuries requiring hospitalisation often take a toll on the health and economy of the affected individuals and their care-giving relatives. The cost is also borne by the general public. Each traumatic injury claim in NZ costs around $1,200, resulting in approximately two billion dollars being paid by taxpayers per year in traumatic injury-related costs (ACC, 2014).

1.2. Dementia

1.2.1. Definition of dementia.

Dementia is a gradual impairment of cognitive and behavioural functioning due to structural and pathophysiological changes in the brain that end up hampering the individual's ability to cope at personal, family, professional, and community levels (Braunwald et al., 2008). Dementia is the most prevalent mental illness among individuals over 75 years of age. About 50,000 New Zealanders have dementia (Alzheimer's NZ, 2015), representing roughly 1% of the population.

According to the WHO (2013) and to the Mayo Clinic (2014), dementia should not be considered a specific disease but a syndrome – or conglomerate of signs and symptoms- affecting memory, though process, and social skills severely enough to interfere with activities of daily living.

1.2.2. Types of dementia.

Dementias can be classified by aetiology, prevalence, age of onset, brain location, and genetic factors (Braunwald et al., 2008; Sadock & Sadock, 2014). The database on which this work is based was composed mostly of cases classified by brain location, that is, cortical vs. subcortical dementias. Cortical dementias involve the bodies of neurons that specialise in memory, language, perception, thought, and attention. Examples of cortical dementias are Alzheimer's disease (AD), dementia with Lewy bodies (DLB), fronto-temporal lobar degeneration (FTLD), and dementia pugilistica or chronic traumatic encephalopathy (CTE) (Braunwald et al., 2008; Sadock & Sadock, 2014).

Subcortical dementias affect the neuronal bodies in the grey nuclei located deep in the brain and in the sub-cortical white matter tracts. Among the most common subcortical dementias are metabolic dementia and Parkinson's disease dementia (Braunwald et al., 2008). Vascular dementia can be both cortical and / or subcortical depending on the location and extent of the ischaemic changes. Simultaneously or not, people can have more than one type of dementia (Braunwald et al., 2008). The present study focuses on the most commonly diagnosed dementias: cortical dementias.

1.3. The New Zealand Central Region

1.3.1. Introduction.

The regions in NZ were created by the central government. New Zealand has two tiers of local government: 11 regional councils and 67 territorial authorities (Local Government Commission, 2014). The country is further divided into 16 regions administered by 11 regional councils. With regard to health, the sector has been restructured several times over the years. The latest restructuring took place in 2010, when a new legislation led to the creation of 20 District Health Boards (DHBs). The MidCentral DHB (MCDHB) is the one on which the current study is based. The DHBs oversee the health and disability services of their respective communities. Of the members of the DHBs, seven are elected by local residents and up to four more may be appointed by the Minister of Health (Local Government Commission, 2014).

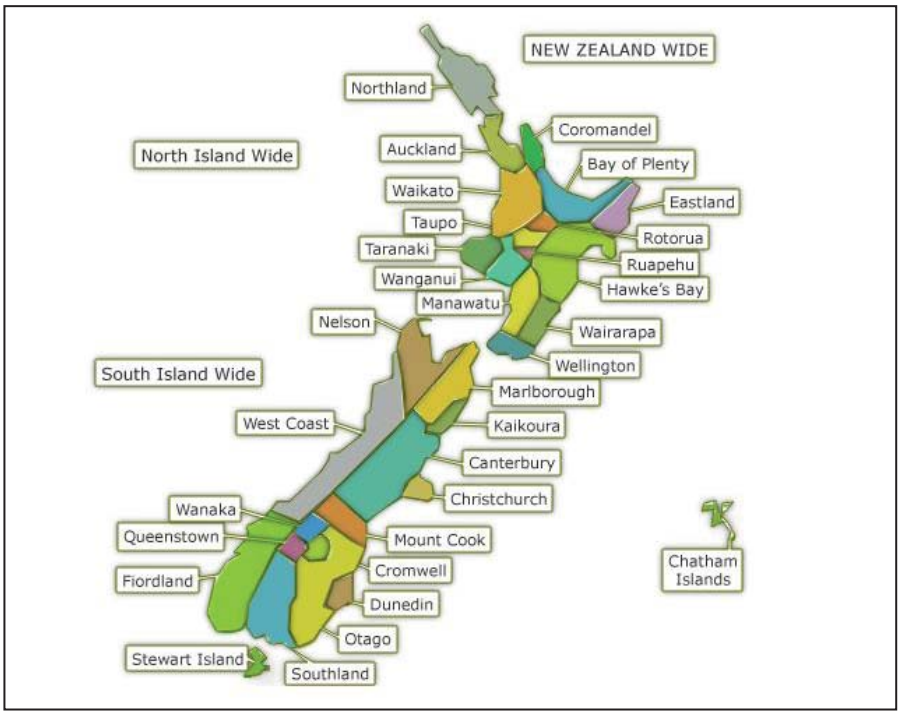

Figure 1. New Zealand Regions
Note. From Land and Search and Rescue (LANSAR), 2013

With regard to the MCDHB district, it stretches across the centre of the North Island from the west to the east coast and is characterised by the Tararua and Ruahine ranges that run across the centre of the district. The MCDHB - of which the PNH is the largest health services provider - serves the following districts: Horowhenua, Tararua, Otaki and Kapiti Coast, Manawatu and Palmerston North. The current NZ population of approximately 4,540,000 people is estimated to increase by one person every eight minutes. The population in the MidCentral Region is about 165,000 people, more than half of which live in Palmerston North City (Statistics NZ, 2014). A broader, more manageable, form of land – and therefore, population - division used by the government to assign Police Districts is that of Regions (NZ Government, 2002). The latter classification, which is depicted in Table 1 below, will be used for the statistical analysis of demographics in this research.

Table 1

New Zealand Regions Classification

NZ ISLAND	REGION NUMBER	REGION NAME	AREA
NORTH (Regions 1-4)	Region 1	Northern	Auckland Northland North Shore-Waitakere Counties-Manukau
	Region 2	Midland	Waikato Bay of Plenty
	Region 3	Eastern	Gisborne Hawke's Bay
	Region 4	Central	Taranaki Rangitikei Manawatu Wanganui Horowhenua Wairarapa Wellington
SOUTH (Regions 5-7)	Region 5	Tasman	Marlborough Nelson West Coast
	Region 6	Canterbury	Canterbury
	Region 7	Southern	Otago Southland

Note. Adapted from New Zealand Government, 2002

As part of the Ministry of Health (MOH), the DHBs are responsible for the provision of funds for health and disability services in their district. Statistics NZ is the original source for all the population data – like the census - shown in the DHB reports (MCDHB, 2009). The 2006/07 NZ Health Survey is the fourth and latest national population-based health survey carried out by the MOH (MCDHB, 2007). The 2011 Census was not held on 8 March 2011 as planned, due to the Christchurch earthquake on 22 February 2011. This means that the demographic and health data in some of the available sources used in this research may be at least eight years old and not as precise as desirable.

1.3.2. The Central Region: Structure and demographics.

Approximately 840,000 people live in the Central Region, representing 20% of the NZ population (CCDHB, 2008). The Central Region is expected to experience a considerable growth in population over the next 20 years. Given that other demographic characteristics of the region are similar to the NZ average (Statistics NZ, 2014), the data of this study may be used as national estimates by proxy. The Central Region is composed of six DHBs: Whanganui, MidCentral, Capital & Coast, Hutt Valley, Wairarapa and Hawke's Bay. Capital & Coast is the largest DHB in the region with a population of almost 278,000; the smallest is Wairarapa with a slightly under 40,000 inhabitants. Because the PNH is one of the largest trauma centres in the Central Region, patients from all six DHBs are often referred to this 300-bed hospital for acute, secondary, and some tertiary services (CCDHB, 2008). To put these data in context, Figure 2 on page 8 shows the population distribution of the NZ Central Region.

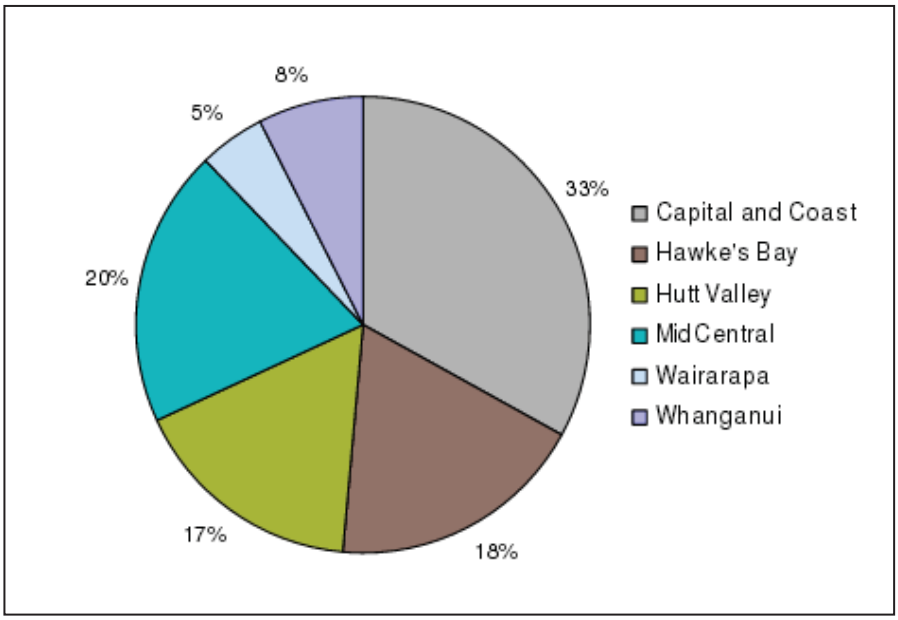

Figure 2. Population Distribution in the CCDHB Area
Note. From Statistics New Zealand, 2006

1.3.2.a. *Gender.*

More males are born than females in NZ, but males have higher mortality rates than females at all ages, particularly between the ages of 20 and 30 (Statistics NZ, 2007). That said, the resulting male to female ratio in the Central Region is approximately 1:1, with slightly more women than men - approximately 51% and 49% respectively (CCDHB, 2013). Because NZ is a developed country, non-communicable diseases account for the highest rates of mortality in the population. In particular cardiovascular diseases are the main cause of death in developed countries. Men are more at risk but women catch up with the death rates after menopause. All in all, women tend to live longer than men in NZ (Statistics NZ, 2007).

1.3.2.b. *Age.*

The age group distribution in the Central Region is very similar to that of NZ (see Table 2). Whereas the proportion of individuals under 15 years of age is almost the same for all the DHBs in the Central Region, the proportion of older people (65+) varies considerably among them (CCDHB, 2008). Wairarapa's proportion of older people is the highest at 16.5%, whereas with 10.5% Capital & Coast has the lowest proportion of senior citizens. The NZ average proportion of individuals who are older than 65 years of age is 12.2% (Statistics NZ, 2007).

Table 2

Population Distribution by DHB and Age Group

DHB	<14	15-24	25-44	45-64	65-84	>85	TOTAL (%Central Region)
Capital & Coast	53,450 (19.2%)	44,310 (15.9%)	89,440 (32.2%)	61,710 (22.2 %)	25,690 (9.2 %)	3,390 (1.2%)	277,990 (33.1 %)
Hawke's Bay	34,740 (22.8%)	19,220 (12.6 %)	39,050 (25.6 %)	38,520 (25.2%)	18,610 (12.2%)	2,450 (1.6%)	152,590 (18.2%)
Hutt Valley	31,550 (22.4%)	19,520 (13.9%)	40,420 (28.7%)	33,500 (23.8%)	14,190 (10.1%)	1,750 (1.2%)	140,930 (16.8%)
Wairarapa	8,285 (20.9%)	4,435 (11.2%)	9,255 (23.4%)	11,085 (28.0%)	5,780 (14.6%)	710 (1.8%)	39,550 (4.7%)
Wanganui	14,040 (22.0%)	8,320 (13.0%)	15,560 (24.3%)	16,025 (25.1%)	8,825 (13.8%)	1,160 (1.8%)	63,930 (7.6%)
MidCentral	34,630 (21.1%)	25,340 (15.5%)	42,270 (25.8%)	38,790 (23.7%)	20,380 (12.4%)	2,550 (1.6%)	163,960 (19.5%)
Central	176,695 (21.1%)	121,145 (14.4%)	235,995 (28.1%)	199,630 (23.8%)	93,475 (11.1%)	20,010 (1.4%)	838,950 (20.1%)
New Zealand	21.2%	14.5%	28.2%	23.9%	10.8%	1.4%	

Note. Adapted from Statistics New Zealand, 2006

While the population of NZ is still young, the number of younger people is projected to decrease considerably in the near future, with the population of over 65 years of age steadily increasing nationwide (Statistics NZ, 2007). One of the main reasons for this trend is the increase in Life Expectancy (LE). LE is an indicator of population health

and reflects mortality from all causes at all ages. As shown in Table 3, LE for males in the Central Region is 77 years and for females is 81 years, similar to the national averages. With regard to ethnicity, the Māori population have a considerably lower LE than the NZ European and Asian population (Statistics NZ, 2007).

Table 3

Life Expectancies at Birth from Gender by Broad New Zealand Region

	LIFE EXPECTANCY	
GENDER	**MALES**	**FEMALES**
Region 4	77.09	81.04
Region 3	76.38	81.11
Region 2	77.73	82.28
Region 1	77.43	82.01
New Zealand	77.14	81.63

Note. Adapted from Statistics New Zealand, 2007

People over 65 years are also high users of secondary care services with 35% of all hospitalisations in the Central Region being for people over 65 years (CCDHB, 2008). Approximately 6% of all people in the Central Region over the age of 65 live in residential facilities. In the Central Region, the volume of potentially avoidable hospitalisations in the 65-74 age group has increased by 16% in the period from 2001 to 2006. The main non-communicable causes of avoidable hospitalisations in the 65-74 age group are cardiovascular disorders, chronic obstructive respiratory disease, diabetes, mellitus, respiratory infections, stroke, and cancer-related symptoms. Cardiovascular disorders and cancers remain the leading causes of death (Statistics NZ, 2007).

Falls are the leading cause of hospitalisations as the result of injury and one of the top three causes of injury related death in NZ (Statistics NZ, 2013). Osteoarthritis and rheumatoid arthritis are common disabling conditions amongst older people that

compound on and worsen other age-related ailments. Depression, anxiety disorders, and substance use disorders (multiple medications and alcohol abuse) put great strains on primary and secondary health services with comorbidities being often more frequent in older age groups (Statistics NZ, 2006, 2014).

1.3.2.c. *Ethnicity.*

Like many developed countries attracting growing numbers of migrants, the ethnic composition of NZ keeps changing over time. Based on the latest census, the population of the Central Region is mostly of European descent (69%), with 16% Māori, 10% Asian, and 5% Pacific people (CCDHB, 2008). Ethnicity is difficult to operationally define with any degree of reliability (Statistics NZ, 2015). One of the main reasons is that there is confusion between concepts like ethnicity, race, ancestry, and citizenship. Another reason is that ethnicity changes over time (ethnic mobility) and some people may provide different ethnic group answers in longitudinal surveys and administrative databases (Statistics NZ, 2015). In that regard, over 10% of New Zealanders identify with more than one ethnic group (Statistics NZ, 2006) and may struggle when asked to give a single answer about their ethnicity. For all of the mentioned reasons, the current study did not consider this variable in the final statistical analysis.

Chapter 2. Health Status of the Central Region

The main purpose of this chapter is to put the health status of the population of interest in this study in context. This will allow comparisons to be made between the study sample health findings and those of the general population. In order to do that, two questions need to be answered: What are the main reasons for hospital admissions in the Central Region, and how are they different in aetiology, number and / or outcome from those in other regions in the country? This chapter will also explain the concept of avoidability in terms of morbidity and mortality rates.

2.1. Hospitalisations

The Central Region has the same mortality causes than the NZ average but with slightly higher mortality rates, with up to 75% of deaths under the age of 75 being classified as avoidable. Avoidability refers to deaths that are potentially preventable with early diagnosis and treatment. Most avoidable deaths are related to cardiovascular disease, diabetes, cancers (lung, breast, colorectal, prostate), chronic pulmonary diseases (COPD, asthma), and suicide. The causes of death for Māori people (see Table 4) are often related to lifestyle factors like diet, sedentarism, and substance use. Māori people have three times the avoidable mortality rates compared to Europeans (CCDHB, 2008).

Table 4

Major Causes of Death Ranked by Age-Standardised Mortality Rates

	MALES	FEMALES
EUROPEAN	Ischaemic heart disease	Ischaemic heart disease
	Suicide	Breast cancer
	Lung cancer	Cerebrovascular disease
	Cerebrovascular disease	Lung cancer
	Colorectal cancer	Colorectal cancer
MĀORI	Ischaemic heart disease	Ischaemic heart disease
	Lung cancer	Lung cancer
	Diabetes	Chronic obstructive pulmonary disease
	Suicide	Diabetes
	Traffic accidents	

Note. From Statistics New Zealand, 2006

Because the present study is based on acute hospital admissions and comorbities in both cases and controls, it is relevant at this point to highlight the main causes of hospitalisations in the Central Region.

The leading cause of hospitalisations in the Central Region are infections of respiratory, cutaneous, dental, gastrointestinal and / or otorhinolaryngological origin. Ambulatory sensitive hospitalisation rates in the Central Region have been on a mildly increasing trend since 2004 (CCDHB, 2008). Among the causes of hospitalisation frequently leading to admission are complications from non-communicable diseases and injuries, the former being more prevalent than the latter. Non-communicable diseases include chronic conditions like cardiovascular disorders (CVD), responsible for over 40% of all deaths in the Central Region. CVD mortality rates are in a declining trend due to improved primary prevention (especially quitting smoking) and improved therapeutic quality and compliance. Although the health gap between the Māori and the European population is declining, the former still have three times the mortality rate for CVD. CVD hospitalisation rates increased in the Central Region in 2006 but not in other NZ regions.

The Central Region acute myocardial infarction (AMI) hospitalisation rates are significantly higher than the national average (CCDHB, 2008). The second place in number of hospitalisations, and a common comorbidity of the dementia patients in this study, is diabetes mellitus (DM). Diabetes is a condition in which there are high levels of glucose in the blood. Type 1 DM is an autoimmune genetic disorder and, therefore, it is not preventable (Braunwald et al., 2008). It accounts for 10% of diabetes cases and deaths and 30% of diabetes hospital admissions. Type 1 DM has lower mortality and

hospital admission rates, but higher use of primary and associated health services and pharmaceuticals than type 2 DM. Type 2 DM is an acquired metabolic disorder that is largely preventable with lifestyle changes, mostly diet and exercise (Braunwald et al., 2008). It accounts for 90% of diabetes cases and deaths, and 70% of diabetes hospital admissions. The condition is more prevalent among the Māori and the general older population. Type 2 DM hospital admissions are significantly lower in the Central Region than in NZ (CCDHB, 2008).

The third cause of hospitalisation of interest in the Central Region is respiratory disease. Avoidable respiratory disease deaths are 95% due to COPD and 3% due to asthma. The Central Region COPD hospitalisation rates are significantly lower than the national average, whereas asthma admissions are similar to those of the rest of NZ. Premature mortality rates of all respiratory conditions are over 40% for Māori adults and 6% for Europeans. Māori and Pacific people have higher COPD and asthma hospitalisation rates. COPD admissions are generally increasing across NZ, and most involve adults of over 45 years of age, with four out of five patients being over 65 years of age (CCDHB, 2008).

Renal failure is another condition to consider among the most common causes of hospital admissions. Major risk factors for chronic kidney disease (CKD) are diabetes and hypertension, both very prevalent all over NZ. Although only a small proportion of patients with CKD will end up developing end-stage renal failure (ESRF) (Kidney Health NZ, 2014), the latter condition accounts for most of the burden of renal failure-related of hospital services, health expenditure and quality of life impact. There are two main nephrology specialist centres in the region – Palmerston North and Wellington –

both providing renal dialysis for ESRF patients. The number of dialysis patients, mostly elderly ones, is increasing faster for PNH than for the Wellington Hospital. The number of patients benefiting from home-based dialysis in the Central Region exceed those receiving dialysis in hospitals (CCDHB, 2008; Kidney Health NZ, 2014).

Finally, the fifth most frequent cause of hospital admissions in the Central Region, marginally before cardiovascular disorders when it comes to mortality rates, is cancer. The age-standardised rates (ASR) for all-cancer admissions is currently about six. All-cancer hospitalisations and mortality are higher for Māori people (CCDHB, 2008). In the Central Region, the top five female cancer diagnoses between 2000 and 2004 were breast, colorectal, melanoma, lung, and uterus. The top five male cancer diagnoses are prostate, colorectal, lung, melanoma, and bladder. All ethnicity ASR for prostate cancer registrations are projected to decrease steadily due to the implementation of screening programmes. Hospitalisation rates are higher for the - arguably - more symptomatic types of malignant neoplasms such as colorectal cancer (CCDHB, 2008).

2.2. Mortality Rates from Non-Communicable Diseases

Injury is the leading cause of lost years of life in those aged under 45 in NZ, the main cause being traffic accidents. The leading cause of traumatic death among elderly people are falls. That said, when it comes to overall mortality rates and co-morbidity factors in trauma cases, non-communicable diseases take the lead. Mortality rates in the Central Region (shown in Table 5 on page 17) are all generally decreasing. The Capital and Coast DHB has the lowest mortality rates and these are lower than the NZ rates. The other five DHBs generally have mortality rates higher than the NZ average (Statistics NZ, 2007). The most common causes of mortality are: cardiovascular

disorders (mostly acute myocardial infarction and ischaemic heart disease) 33%, cancers (gastrointestinal, lung, melanoma, prostate, breast) 19%, chronic respiratory diseases (COPD and asthma) 6%, stroke 5%, diabetes mellitus 3% (CCDHB, 2008).

Cardiovascular disorders and cancer account for over one in three deaths in the Central Region (CCDHB, 2008). Secondary diabetes is an important cause of mortality among Māori and Pacific People. Mortality rates are higher for men at all ages. On a more positive note, avoidable mortality has been declining across NZ and specifically across the Central Region with an almost 30% decline. Avoidable mortality rates in the Central Region still do tend to be slightly higher than other regions with Capital & Coast DHB being the lowest (CCDHB, 2008).

Almost 80% of all avoidable deaths occur in those aged 45-74 years, dominated by cardiovascular disease, cancers and diabetes. Avoidable death rates are much lower in younger age groups. Reductions in avoidable death rates are caused predominantly by reductions in preventable mortality amongst 65-75 year olds. The major causes of avoidable mortality for all ethnic groups and all ages are cardiovascular diseases followed by cancer.

According to the latest NZ Health Survey (MOH, 2013), the prevalence of COPD in NZ adults (aged 45+) is approximately 5.5%. There is a minor difference in the prevalence in males (4.8%) and females (6%) but there is no significant difference between Māori and non-Māori. The incidence and mortality rates of COPD increases with age for both males and females (MOH, 2013). The mortality rates due to a chronic respiratory disorder, mostly COPD and asthma, have remained relatively stable since 2001 for each

region of NZ and for each Central Region DHB. COPD accounts for about 70% of respiratory deaths in the Central Region, of which approximately 91% are people aged over 65 years and the remainder for adults aged 45 - 64 years (MOH, 2013).

With regard to adult deaths due to respiratory disorders in NZ, 90% involve individuals aged 65+ years, 9% are aged 45 - 64 years, and 1% are aged 25 – 44 years. According to the results in the latest NZ Health Survey (MOH, 2013), there are no significant differences from the national rate in any region (or any Central Region DHB) for any age groups, for COPD, asthma and respiratory disease in general.

Table 5

Mortality Rates per Non-Communicable Disease in the Central Region

NON-COMMUNICABLE DISEASE	MORTALITY RATE
Cardiovascular disease	33%
Cancer	19%
Chronic respiratory disease	6%
Stroke	5%
Diabetes mellitus	3%

Note. Adapted from Mortality Collection (CCDHB, 2008)

A considerable proportion of the non-communicable disease death rates are avoidable as noted in Table 6 on page 18. In NZ, the term “amenable mortality” is a subset of avoidable mortality that applies to deaths from conditions that are preventable with proper and timely healthcare provision (MOH, 2014). Avoidable mortality also includes deaths due to traffic accidents of any kind and applies to deaths occurring to those under 75 years of age. Avoidability strategies include population-based health promotion programmes, prophylactic, or therapeutic interventions in primary healthcare services, and injury prevention campaigns (MOH, 2014).

Table 6

Most Common Causes of Avoidable Mortality for All Ages in the Central Region.

CONDITION	TOTAL	% POPULATION DEATHS
Cancer	443	7.6%
Cardiovascular disorder	376	6.4%
Injuries (self + other inflicted)	187	3.1%
Respiratory disorder	109	2.0%
Cerebrovascular disorder	98	1.7%
Diabetes mellitus	78	1.3%
Total top 6	1289	22.0%

Note. Adapted from Ministry of Health, 2014.

Chapter 3. Traumatic Injury

The present chapter aims to shed some light on the incidence and impact of traumatic injuries in NZ. The relevance of geography and ethnicity in trauma outcomes will also be considered along with the management and economic impact of the problem on individuals with dementia, their caregivers, and society. Particular attention will be paid to trauma risk factors and disabilities in residential facilities.

3.1. Introduction

The impact of traumatic injuries on human health worldwide has led the WHO, with support from the Center for Disease Control and Prevention (CDC), to develop programmes to support countries in planning and setting up traumatic injury care strategies to strengthen their capacity to care for patients, a collaboration which is already leading to improvements across the globe (CDC, 2014). Global health changes do not happen overnight. Determination, inter-institutional collaboration, and exchange of data among those working in the field can help lead to more widespread and systematic efforts to strengthen trauma care services. In so doing, the lives of many people with traumatic injuries may be saved, many falls-related disabilities averted, and people with disabilities may receive better services and be integrated back into active life more fully (CDC, 2014).

In June 2012, the NZ MOH and the ACC established and jointly funded the Major Trauma National Clinical Network (MTNCN) to provide clinical leadership and oversight to ensure there is a planned and consistent approach to major trauma service provision in NZ. The development and implementation of a national major trauma

database, the NZ Major Trauma Registry, has been the key objective of the MTNCN (Health Quality & Safety Commission NZ (HQSC) (2012).

The PNH – a part of the MCDHB - is currently implementing protocols of falls risk assessment for patients deemed to be at risk. This is an initiative derived from the data collection requirements of the DHBs (MOH, 2013). Under the MOH's Operating Policy Framework, health providers have to update a Health Needs Assessment (HNA) for their local population at least every three years to inform their strategic plans. The methodology used to develop the HNA report includes meetings with the relevant stakeholders, research and data collection, developing data cubes, undertaking analysis and report writing, hiring epidemiologists, and engaging clinical experts to review relevant subject areas. Data on hospital admissions in HNA reports are sourced from the National Minimum Dataset (NMDS) (2012) and are used as a reference for health status (MOH, 2013).

3.2. Traumatic Injury in New Zealand

The national average major traumatic injury rate is 6.5/1000 and there is a greater than 1.5-fold variation by DHB (MOH, 2013). There is no significant difference in major injury rates by ethnic group. There are two peaks in incidence: one in those aged 0–24 and another in those aged 65 and over. The major injury rate in those aged 65 and over is double that of those aged 25–64. Approximately 250 people a year die in hospital following first admission with a primary diagnosis of major injury (MOH, 2014). The mean in-hospital mortality rate nationally for major trauma admissions over three years is 10/1000. No DHB has a mortality rate significantly greater than the national mean (MOH, 2014).

Older people (65 years and over) are significantly more likely to die following admission for major injury, with a mean rate of 35/1000 (MOH, 2014). Major trauma represents about 8% of all patients admitted with injuries, meaning broad conclusions regarding all trauma care cannot be drawn from these data (HQSC, 2012). Over the years reported on, there have been 149 deaths in the trauma registries, from a total of 1482 major trauma admissions. There is no significant variation in mortality rate between the DHBs, with a mean rate of 97.2/1000 (MOH, 2014). Mortality is significantly higher in the 65 years and older age group compared with all other age groups (mean rate 243/1000). There are no significant differences in mortality rates by ethnicity or gender, although males represented 70% of admissions for major trauma and, by count, twice as many males die (MOH, 2014; NMDS, 2012; Statistics NZ, 2006).

In NZ almost 90% of all major trauma is blunt, approximately 6% penetrating, and 5% burns, all of which are capable of causing severe external and internal organ damage (MOH, 2013; NMDS, 2012). Injures may be isolated or affect multiple regions including the head, neck, face, chest, abdomen, pelvis, extremities and spine. Knowledge of the common mechanisms and patterns of injury are necessary to predict, treat, and prevent traumatic injury. The severity and body region injured is often dependent on the means of injury and are prognostic of patient survival and recovery. Blunt injuries are caused by acceleration, deceleration, compression or shearing forces resulting in the body being crushed or impacted with another object, or the shifting of internal organs (NMDS, 2012).

Among the general population, the majority of blunt trauma, 57%, is related to traffic accidents, followed by falls (22%). In the older population groups (65+), the most common cause of traumatic injury is falls. Blunt trauma is most often of non-intentional causes, with less than 5% related to assaults. More than half of the patients who suffer blunt thoracic trauma have considerable injuries to the head, with more than one-third having significant injuries to the area, and upper and lower extremities (NMDS, 2012). The national and regional data regarding traumatic injury risk factors, body area, and health outcomes will be compared and discussed with those of the results of this study. Finally, serious abdominal injuries occur in about 15% of patients, so that the total number of patients requiring abdominal surgery in blunt trauma is relatively small. Trauma care in NZ has a multidisciplinary provision, from the composition of trauma teams to the strong reliance on subspecialised surgical care (Curtis, Caldwell, Del Prado, & Munroe, 2012).

3.3. Trauma and Geography in New Zealand

New Zealand has many communities in remote and rural regions. This means that there are large distances between homes and medical facilities which may have varying levels of healthcare available (NMDS, 2012). Trauma centres are often situated in the metropolitan areas and, therefore, many trauma patients are assessed, stabilised, admitted, or treated in non-trauma centres. For the major trauma patient, distance from comprehensive healthcare can have life-threatening consequences and a person suffering a serious traumatic injury in rural NZ is twice as likely to die than if the same accident had occurred in a city setting (NMDS, 2012). It is for this reason that education for pre-hospital and rural personnel is so important.

The time taken for the injured individual to reach definitive medical care should be as short as possible (HQSC, 2012). Getting the patient to the right hospital at the right time is a vital principle of effective trauma care, where too far may often mean too late. Geography in NZ delays arrival to hospital in some regions but improvements in trauma systems, from pre-hospital triage to early trauma management, are being implemented so that patients can get treatment in time (HQSC, 2012). The national data show that an average of 32% of major trauma cases arrive in the hospital less than one hour after the injury. About 80% arrive within three hours and the majority (88%) do so within six hours (HQSC, 2012). As will be shown in the Results section on page 81, acute and emergency admissions to the PNH are mixed in terms of geographical origin. Most patients are referred to the Hospital from Region 4, which includes a large rural population from Taranaki, Rangitikei, Horowhenua, and Wairarapa as previously shown in Table 1 on page 6.

3.4. Trauma Morbidity

For every trauma patient who dies from their injuries, there are nearly six who survive to hospital discharge. Almost half of the admissions with severe injuries, that is, those with an Injury Severity Score (ISS) > 15, are admitted to intensive care for an average of seven days (NMDS, 2012). The ISS is an anatomical scoring system that provides an overall score for patients with multiple injuries (Palmer et al., 2013). Each injury is assigned an Abbreviated Injury Scale (AIS) score (see Table 7 on page 24) and is allocated to one of six body regions: head, face, chest, abdomen, and upper and lower extremities (including pelvis). In the AIS, injuries are ranked on a scale of one to six, with one being minor, five severe and six an unsurvivable injury. Only the highest AIS

score in each body region is used for scoring purposes (NMDS, 2012; Palmer et al., 2013).

The three most severely injured body regions have their score squared and added together to produce the ISS score. The ISS score ranges from 0 to 75. If an injury is assigned an AIS of 6 (unsurvivable injury), the ISS score is automatically assigned to 75. The ISS score is virtually the only anatomical scoring system in use and correlates linearly with mortality, morbidity, and hospital stay (NMDS, 2012; Palmer et al., 2013).

Table 7

AIS Score and Degree of Injury Classification

AIS Score	Degree of injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Unsurvivable

Note. Adapted from Trauma.org, 2014

The average length of hospital stay for seriously injured individuals is around 20 days (Statistics NZ, 2013). Not all morbidities resulting from traumatic injury are severe or extremely disabling. Some result in significant dysfunction, pain, and other sequelae, while many minor injuries heal, leaving little or no residual dysfunction. Many times, when full recovery is incomplete, injury results in a degree of ongoing dysfunction or the onset of secondary conditions (such as osteoarthritis in injured joints). Amongst the people with a reported disability, over 15% of the cases implied a traumatic injury as the underlying cause (Statistics NZ, 2013). Moreover, patients with a history of a major trauma are known to have developed mental health problems as a consequence (Australian Bureau of Statistics, 2012). What remain to be determined are the type and the prevalence of the injuries.

3.4.1. Trauma risk factors.

Whether accidental or not, a considerable number of physical injuries can be prevented by identifying their causes and removing them (HQSC, 2012). Understanding some of the risk factors for injury may have a predictive value in anticipating patterns of traumatic injury in certain populations and/or informing and evaluating injury prevention strategies. There are multiple trauma risk factors, the most common of which are age, gender, substance use, occupation, environment, and road safety issues (HQSC, 2012). With a growing awareness of the incidence and cost of trauma to society, there has been an increasing emphasis on prevention strategies and the establishment of legislative changes to improve road and workplace safety (HQSC, 2012).

New Zealand has been world leader in introducing legislative changes to improve road and workplace safety. Continuous improvements in trauma-related morbidity and mortality strategies have demonstrated the important contribution of legislation in addressing trauma as a public safety (Ministry of Business, Innovation and Employment, 2012). The perception of trauma as a preventable occurrence rather than a random event is fundamental to the success of injury prevention programmes (Statistics NZ, 2013). One such preventable trauma factor in older people is polypharmacy. Polypharmacy or multi-medication is the intake of many medicines (prescribed or over-the-counter), usually over five of them (DiPiro & Schwinghammer, 2011; Norris et al., 2011). Many senior citizens take five or more medicines, mostly to treat non-communicable diseases like cardiovascular disorders (like ischaemic heart disease), hypertension, hyperlipaemia, diabetes, chronic respiratory disorders, and psychiatric problems (mostly anxiety and depression) (Norris et al., 2011). Every medication has potential adverse side-effects, and as a new drug is added to a treatment routine, the risk

of side effects and interactions increases exponentially (HQSC, 2012). Older people (traditionally defined as those aged 65 years and over), especially those with frailty or multiple comorbid conditions, are more susceptible to medication-related traumatic injury morbidity and mortality. The frequency of adverse drug events increases with the number of medicines taken: from 13% with two medicines and 58% with five medicines to 82% when seven or more medicines are taken (HQSC, 2012).

In older people, certain classes of medicines carry a substantially higher risk of adverse effects. Two examples are antipsychotics and benzodiazepines, of which common adverse effects include impaired functional ability, agitation, confusion, blurred vision, urinary retention, constipation, postural hypotension and falls. These adverse effects increase if both classes are given together (DiPiro & Schwinghammer, 2011; Norris et al., 2011).

Another way to consider polypharmacy is to investigate whether older people are receiving combinations of medicines known to result in interactions with other medications or with food (Aronson, 2004; DiPiro & Schwinghammer, 2011; Norris et al., 2011). One example is the combination of antiplatelet aggregation drugs with anticoagulant agents, which is associated with an increased risk of bleeding complications (DiPiro & Schwinghammer, 2011). Nationally, approximately one in four people aged 65–75 received five or more long-term prescription drugs in 2011 and over one in two people aged 85 and over receive five more long-term medicines. Although the number of people receiving 11 or more long-term medicines is lower, at around 1 in 20 people, the rate increases sharply with age, with, people aged 85 and over were 2.5

times more likely to receive 11 or more medicines than those aged 65–75 (Aronson, 2004; Norris et al., 2011).

An example of drug-food interaction includes medications prescribed to treat common comorbidities, such as depression, in patients with dementia like mono-amine oxidase inhibitors (MAOIs) (Braunwald et al., 2008; DiPiro & Schwinghammer, 2011). These antidepressants act by blocking an enzyme called mono-amine oxidase (MAO). The enzyme breaks down a substance called tyramine. Tyramine is a catecholamine-releasing agent. Blocking the MAO increases the concentration of tyramine in blood and, therefore, of catecholamines. If MAOIs are consumed together with a tyramine-rich diet containing smoked, cured, or fermented foods like most cheeses, yogurts, and wines, catecholamines can rapidly reach high levels and cause a life-threatening hypertensive crisis (Braunwald et al., 2008). Vegemite® and Marmite® are also among the dangerous drug-food combinations of MAOIs. Fortunately, MAOIs are currently seldom routinely prescribed and are not considered a first-line treatment for depression due to their multiple and serious side effects, drug-drug and drug-food and drinks interactions (Braunwald et al., 2008; DiPiro & Schwinghammer, 2011).

3.5. Trauma Clinical Management Implications

There is evidence suggesting insufficient standardisation – diagnosing and classification – of trauma leading to errors in trauma management, a worrisome and costly issue (Celso et al., 2006; Fitzgerald et al., 2006). Errors in trauma management notably contribute to the data regarding preventable or potentially preventable morbidity and mortality. Indeed, most preventable errors occur not because of ignorance or lack of resources, but because the correct therapeutic and diagnostic measures are not

performed at the right time, to the right degree, or in the right order. Implementing and maintaining principles of standardising trauma care is vital to optimising patient recovery and emergency nurses are essential to this process (Celso et al., 2006; Fitzgerald et al., 2006).

The Early Management of Severe Trauma guidelines were introduced to NZ adapted under license from the Advanced Trauma Life Support guidelines by the Royal College of Surgeons in 1988 (Royal College of Surgeons, 2014). This has ensured a standardised approach to managing trauma for all emergency healthcare clinicians. In addition, the establishment of organised trauma systems has improved patient survival. The aim of a trauma system is to facilitate treatment of the injured patient at the right hospital, resulting in better care for all trauma patients. Particular emphasis is placed on the development of a trauma system that involves pre-hospital care, acute care in the hospital setting, recovery and rehabilitation, in both hospital and home settings, although this has not been evaluated in all of the NZ regional health systems (Royal College of Surgeons, 2014). The verification of trauma centres conducted by the Royal Australasian College of Surgeons (RACS) demonstrated significant improvements in patient care in the last decade, enhancement of institutional policies, and commitment to care of the injured patient (RACS, 2012).

An organised multidisciplinary team is essential to optimise patient outcomes and the successful development of injury management services. A good trauma service is one that coordinates a larger multi-disciplinary team tailored to the needs of each patient, and typically consists of various medical and nursing specialties, allied health, and rehabilitation clinicians. The recent establishment of core trauma nursing strategies in

major trauma centres, like the PNH, ensures that trauma patients and their families are provided with comprehensive physical and emotional care (RACS, 2012).

3.6. The Cost of Traumatic Injury

The cost of traumatic injuries with an AIS score ≥2 includes the cost of hospital and nursing home care, health specialists services, rehabilitation, community-based services, medications, complementary diagnostic services (imaging and laboratory), adaptations made to the house, and insurance processing (ACC, 2012). It should be noted that these costs do not account for the long-term effects of traumatic injuries such as physical and/or mental disability, dependence on others, lost time from work, household, and family duties, and reduced quality of life.

The Australasian Trauma Society claims that traumatic injury is a leading cause of productive life loss (RACS, 2012). In NZ, injury to self and others, whether intentional or not, is the leading cause of death for ages 1- 34 years, and the second leading cause of hospitalisation. Traumatic injuries account for more potential years of life lost than cancer and heart disease combined (RACS, 2012). That fact adds to the relevance of the current work. In childhood, injuries accounts for approximately 60% of all deaths; and by adolescence and young adulthood, injuries (including suicide) accounts for approximately 80% of deaths. In 2008 the social and economic costs of injury were estimated to be at least $7 billion per year. Higher costs are associated with severity of injury and length of stay (HQSC, 2012; CDC, 2013).

Human cost as a result of traumatic injury is considerable, ranging from extensive recovery periods to long-standing disabilities and loss of life (RACS, 2012). For those who do not survive traumatic injury, there is a net loss of their contribution to family and society. If ACC compensation would ever be available for individuals with incipient trauma-related dementia, they might remain productive members of society for longer. On average, each fatal injury before the age of 75 results in the loss of 32 years of potential life, compared with nine years for cancer and five for cardiovascular disease. Injuries were responsible for 7% of the total burden of disease and injury in Australia and NZ in 2003 (RACS, 2012). The subsequent grief and loss experienced by families and significant others has an impact on the mental health both of patients and caregivers, and leads to secondary healthcare costs, loss of productivity, and subsequent macro-economic burden (Curtis, Ramsden, & Lord, 2011; Leonard & Curtis, 2014).

For traumatic injury survivors, recovery periods and long-term disabilities result in a reduced economic contribution and/or long-term economic liability imposed on health and social systems. Cognitive changes as a result of traumatic brain injury have been recognised as a significant cause of psychological impairment in response to traumatic injury (Bisson, Shepherd, Joy, Probert, & Newcombe, 2004). Yet, the psychological consequences of other serious traumas are currently neglected in burden-of-injury calculations. While there is little evidence for the efficacy of early debriefing on post-traumatic injury, there is some evidence that early therapy may lessen the psychological impact of injury and help prevent progression (Bisson et al., 2004).

Among the population of NZ, traumatic injury is the fourth leading cause of death and the leading cause of potential years of life lost (Statistics NZ, 2007). For the purpose of

having some perspective, when injury fatality rates in the USA were compared with those in NZ using mortality censuses and national hospital discharge data, it was found that, overall, unintentional injuries were 1.57 times more likely to be fatal in NZ but intentional assault injuries were 1.14 times more likely to be fatal in the USA. Firearms were involved in 50% of assaults in the USA versus 8% of NZ assaults (Spicer, Miller, Langley, & Stephenson, 2005). Cutting/piercing injuries are 1.85 and firearm injuries and traffic-related injuries are 1.43 times more to be likely fatal in NZ. Natural/environmental injuries were significantly more likely to be fatal in the USA. Possible reasons for the observed results include differences in region of provenance and proportion of population in rural areas, trauma system differences, road design and vehicle types, seatbelt use, accessibility to weapons, legislation, policies, and environmental factors (Spicer et al., 2005).

Almost 80% of falls-related deaths - and their associated costs - in NZ are due to traumatic injuries to the head and neck and to the lower extremities, mostly to the femur (ACC, 2012). This is in keeping with the findings of the Results chapter in this work (see pages 81 to 87). Injuries to internal organs are responsible for 30% of falls-related deaths and account for 30% of costs. Fractures are both the most common and most costly non-fatal injuries. Just over one-third of non-fatal injuries are fractures, but these account for 60% of total non-fatal costs.

Hospitalisations account for nearly two-thirds of the costs of non-fatal fall injuries and emergency department treatment account for 20% of it (Stevens, Corso, Finkelstein, & Miller, 2006). As previously mentioned, hip fractures are the most frequent, serious, and

costly of all fall-related fractures. Their related hospitalisation costs account for 45% of the direct medical costs (Roudsari, Ebel, Corso, Molinari, & Koepsell, 2005).

3.7. Traumatic Injury Disability in New Zealand

Nearly all adults living in residential care facilities report having one or another type of disability (99%). Most of them have multiple disabilities (94%) and high need for support (82%) (Statistics NZ, 2006). Table 8 on page 33 illustrates the main causes of disability in the overall adult population of NZ. In 2006, an estimated 660,300 New Zealanders reported a disability, representing just over 14% of the total population (Statistics NZ, 2006). The percentage of people with disability increases with age to 45% for adults aged 65 years and over.

Among the general adult population, approximately 332,600 females and 327,700 males have a disability in NZ, with more males under the age of 65 having a disability than females in the same age group. The difference is mostly due to occupational hazards and more men being involved in high risk activities (Statistics NZ, 2006). However, disability has worse outcomes in women aged 65 years and over, accounting for 57% of the disabled people in this age group. This is partly due to the greater number of women in this age group, where the rate of disability is very high, and partly because of the higher prevalence of osteoporosis in elderly women. Males have a slightly higher rate of disability in the age groups under 65 years, while females have a higher rate in the age group 65 years and over (Statistics NZ, 2006).

Accidental injury is the most common cause of disability for individuals aged 15 to 45 years, representing just over 30% of people with disability in NZ, closely followed by non-specified or non-reported causes (29%). Accidents or injuries are also the most common cause of disability for adults aged 45 to 65 years (34% of people with disability), followed by disease or illness (32%) (Statistics NZ, 2013). Age-related conditions are, however, the most common cause of disability for adults aged 65 years and over, affecting more than half of the adults with disability. Disease or illness is the second most common cause for this age group (47 % of adults with disability).

Table 8

Causes of Disability[a] *in the Adult Population of New Zealand*

	Number of adults	Disabled adults	Total adults
Disease	211,100	37%	7%
Presence at birth	57,900	10%	2%
Natural ageing	153,200	27%	5%
Injury	166,300	29%	5%
Other causes	142,100	25%	5%
Not specified	53,500	9%	2%

Note. Adapted from Statistics NZ, 2013

[a] People may have more than one cause of disability

Forty percent of adults with disability have a single disability and 60% report having multiple disabilities (Statistics NZ, 2013). The percentage of people with multiple disabilities increases with age. Fifty percent of people aged 15 to 45 years have multiple disabilities compared with 53% of adults aged 45 to 64 years old and 73 % of adults aged 65 years and over. Sixty-three percent of females and 54% of males with disability report having multiple disabilities (Statistics NZ, 2013).

3.7.1. Disability in residential facilities.

Nearly all adults living in residential care facilities report having a disability (99.7%), compared with 17.4% of adults living in households (Statistics NZ, 2013). Only 5% of disabled adults living in residential facilities are aged under 65 years. Most adults with disability living in residential facilities have high support needs (82% of disabled) and more than two types of disability (94%) (Statistics NZ, 2013). In contrast only 12% of adults with disability living in households have high support needs and 58% have more than two disabilities.

Two-thirds of adults with disability in residential facilities live in rest homes and one third reside in continuing care hospitals. Ninety-seven percent of adults living in residential facilities have physical disabilities. Other types of disabilities (70%) and sensory disabilities (60%) are also common in residential facilities. Frequent causes of disability for adults living in residential facilities are disease or illness (70% of disabled adults) and natural ageing (56% of disabled adults). In comparison, 35% of disabled adults living in households report disease or illness as a cause of disability and 25% report natural ageing as a cause. Accidents or injuries are a more common cause of disability for adults living in households (30%) than adults living in residential facilities (20%) (Statistics NZ, 2013).

3.7.2. Traumatic injury disability and ethnicity.

In 2006 there were an estimated 96,600 Māori with disability in NZ. Nearly all Māori with disability (99%) report living in households and less than 1% live in residential facilities (Statistics NZ, 2006). The number of people with disability by ethnic group is

shown in Table 9 below. The total disability rate for Māori (17%) is higher than the disability rate for Pacific peoples (11%) but lower than the disability rate for European (18%). Māori and Pacific peoples have a different age-structure to Europeans, with a higher proportion of people aged less than 45 years. Eighty percent of Māori are aged less than 45 years, compared with 54% of the European population (Statistics NZ, 2006). In other words, Māori and Pacific peoples constitute a younger population with higher health needs and higher morbid-mortality rates.

Table 9

Number of People with Disability by Ethnicity and Age in New Zealand

ETHNICITY	AGE GROUP (years)				TOTAL
	0-14	15-44	45-64	65+	
European	42,500	80,100	142,600	183,700	448,900
Māori	28,200	33,000	24,300	11,000	96,500
Pasifika	6,100	8,100	6,600	4,000	24,800
Asian	4,300	3,300	4,600	5,000	17,200
Other	8,900	17,000	30,400	16,500	72,800
Total	90,000	141,500	208,500	220,200	660,200

Note. From Statistics NZ, 2013

Māori people have a higher disability rate than other ethnic groups in every age group. The higher proportion of young people in the Māori population means that the majority of Māori with disability (63%) are aged under 45 years. Nearly one-third of children with disability (31%) and nearly one-quarter of adults aged 15 to 44 years with disability (23%) are Māori (Statistics NZ, 2013). Two-thirds of Māori adults with disability (12% of all Māori aged 15 years and over) have physical disabilities. Forty-five percent of Māori adults with disability had other disability types such as difficulty speaking, learning, remembering or doing everyday activities.

Other common disability types in the Māori population group include sensory (hearing and/or seeing) disabilities (37% of disabled) and psychiatric or psychological disabilities (26% of disabled).

The most common causes of disability for Māori adults are diseases or illnesses (34% of Māori adults with disability) followed by accidents or injuries (32%). The most common types of accidents or injuries occurred in the workplace or at home, or involved traffic accidents. Thirty-eight percent of the Māori adults with disability have a single disability and 62% have multiple disabilities (Statistics NZ, 2013). These data indicate that there are ongoing ethnic health inequalities in NZ that need to be pointed out and further addressed by specialised researchers and policy-makers.

Chapter 4. Falls

The present chapter focuses on falls, the most common cause of traumatic injury in the elderly and, specifically, in the dementia population. Two types of falls risk factors will be considered: intrinsic and extrinsic. The former are inherent to the individual like muscle weakness, sensory deterioration, or medical conditions, and the latter are extraneous to the person such as substance adverse effects and the natural and built environment. The section will conclude with a reference to falls in rest and nursing homes and their impact on the morbi-mortality of residents.

4.1. Introduction

The WHO (2012) considers falls a major public health problem globally. An estimated 424,000 fatal falls occur each year, making it the second leading cause of unintentional injury death after road traffic injuries. Over 80% of fall-related fatalities occur in low- and middle-income countries, with regions of the Western Pacific and South East Asia accounting for more than two thirds of these deaths. In all regions of the world, death rates are highest among adults over the age of 60 years. Approximately 37 million falls are severe enough to require medical attention each year worldwide. Such falls are responsible for over 17 million disability-adjusted life years (DALYs) lost. The largest morbidity occurs in people aged 65 years or older, young adults aged 15–29 years and children aged 15 years or younger (WHO, 2012). While nearly 40% of the total DALYs lost due to falls worldwide occurs in children, this measurement may not accurately reflect the impact of fall-related disabilities for older individuals who have fewer life

years to lose. In addition, those individuals who fall and suffer a disability, particularly older people, are at a higher risk for subsequent long-term care and institutionalisation.

The financial costs from fall-related injuries are substantial for both the tax-payer and the administration (ACC, 2012; WHO, 2012). To offer some international perspective, for people aged 65 years or older, the average health system cost per fall injury in Finland and Australia are US$ 3,611 and US$ 1,049 respectively (WHO, 2012). The age, gender, and general health status of the individual affect the type, severity, consequences, and prognosis of injury.

Of all the well established falls risk factors (see Table 10 on page 39), age is the most relevant one (WHO, 2012). Older people have the highest risk of death or serious injury arising from a fall and the risk increases with age. For example, in the USA, 20–30% of older people who fall suffer moderate to severe injuries such as bruises, hip fractures, or head trauma. This risk level is in part due to physical, sensory, and cognitive changes associated with ageing, and in part due to built environments that are not adapted for an aging population (WHO, 2012).

Across all age groups in developed countries, both genders are at risk of falls (WHO, 2012). Older women and younger children are especially prone to falls and increased injury severity. Worldwide, males consistently sustain higher death rates and DALYs lost. Possible explanations of the greater burden seen among males may include higher levels of risk-taking behaviours and hazards within occupations (WHO, 2012).

Table 10

Falls Risk Factors

RISK FACTORS FOR FALLS
Age (younger and older age groups)
Gender (males > females)
Occupational / recreational hazards
Alcohol and/or substance use (including side effects of medication)
Socioeconomic factors (poverty, overcrowded / cluttered / inadequate housing)
Sedentarism
Poor cognition (mostly dementia)
Poor sensory acuity (visual, auditory, propioceptive)
Living in an institution (like a rest or nursing home)

Note. Adapted from WHO, 2012.

According to the United Nations Organization (UNO) (2012) fall prevention strategies should be comprehensive and multidimensional. They should prioritise research and public health initiatives to further define the burden, explore variable risk factors and utilise effective prevention strategies. They should support policies that create safer environments and reduce risk factors. They should also promote engineering to remove the potential obstacles for falls, the training of health care providers on evidence-based prevention strategies; and the education of individuals and communities to build risk awareness (UNO, 2012).

Effective fall prevention programmes aim to reduce the number of people who fall, the rate of falls and the severity of injury should a fall occur. For older individuals, fall prevention programmes can include a number of components to identify and modify known risks, such as those shown in Table 11 on page 40.

Table 11

Strategic Recommendations to Reduce Falls Risk by the WHO

W.H.O. FALLS RISK PREVENTION STRATEGIES
Falls risk screening at home and at primary and secondary healthcare service settings
Screening within living environments for risks for falls
Clinical interventions to identify risk factors, such as medication review and modification, treatment of low blood pressure, Vitamin D and calcium supplementation, treatment of correctable visual impairment
Home assessment and environmental modification for those with known risk factors or a history of falling
Support device prescription to address physical and sensory impairments; use of hip protectors for those at risk of a hip fracture due to a fall

Note. Adapted from WHO, 2012

Within the WHO Global Burden of Disease database, falls-related deaths and non-fatal injuries exclude falls due to assault and self-harm, falls from animals, burning buildings, transport vehicles, and falls into fire, water or machinery (WHO, 2009). The disability-adjusted life years (DALYs) extend the concept of potential years of life lost due to premature death to include equivalent years of "healthy" life lost from being in a state of poor health or disability (WHO, 2009).

4.2. Falls Risk Factors

A predisposition to falling is the result of the cumulative effect of multiple factors (WHO, 2012). The likelihood of injury as a result of a fall depends on the intrinsic characteristics and the circumstances of the fall. A history of falls may be a marker of frailty, poor mobility, or an acute or chronic condition, but it contributes little to the aetiology of these accidents. Investigating the different coexisting risk factors involved is crucial to establish preventive strategies and, thus, aim to prevent the recurrence of such breakdowns and their negative consequences. Falls risk factors vary depending on the individual and their context (WHO, 2012).

People experience the aging process in their own particular way depending on factors like socioeconomic status, lifestyle, and personality (Studenski, Duncan, Chandler, Samsa, & Prescott, 1996). The appearance of age-related conditions, though, influence the way people respond to and cope with a fall. Thus, the same risk factors acquire different relevance if they appear in an institutionalised elderly person or in one being cared for by a relative at their homes (Ambrose, Paul, & Hausdorff, 2013; Berry & Kiel, 2013; Bird, Pittaway, Cuisick, Rattray, & Ahuja, 2013).

Studenski et al. (1996) classified the elderly into several subgroups according to their functional capacity and, therefore, their risk of falling. Among the low risk group were those who were either totally immobile and those who kept good mobility and stability. In the high risk group were those who were mobile but had some degree of instability and those who were frail, had mental and functional disability associated medical comorbidities, and who were on multiple medication. These individuals often fell at home while performing activities of daily living (ADL). It was estimated that high risk individuals suffered recurrent falls almost five times more frequently than the low risk ones (Studenski et al., 1996).

Another high risk group was made of vigorous elderly, often males, functionally very active, with high level of autonomy who suffered a fall while performing potentially hazardous leisure outdoor activities as when they were younger (Studenski et al., 1996). Falls in the elderly with good health and successful aging tend to be much more violent compared to those suffered by the frail elderly (Ambrose et al., 2013; Davuluri & Dharmarajan, 2013).

Among the risk factors for falls are those dependent on the individual, due to aging or comorbidities (intrinsic factors) and those which relate to the environment around that individual (extrinsic factors) (Arnold, Busch, Schachter, Harrison, & Olszynski, 2005; Tzeng, & Yin, 2010; Yamashita, Noe, & Bailer, 2012). A fall is usually the result of a combination of both factors, but the most relevant ones for the risk of falls are those intrinsic to the individual influenced by coexisting underlying diseases (Martin, 2011).

Drugs are also particularly relevant risk factors because of their side effects and their interactions with underlying disorders and other drugs being taken. Among the medications involved in increased risk for falls the most important ones are psychotropic drugs - those that have an effect on the CNS - followed by drugs that affect the cardiovascular system, drugs with extrapyramidal effects, and ototoxic drugs (Kojima, 2012; Kraus et al., 2005; Ziere et al., 2006). Environment falls risk factors intervene in the equation when the individual engages in some form of physical effort that challenges coordination or stability (Lord, Menz, & Sherrington, 2006).

4.2.1. Intrinsic factors.

Intrinsic factors influencing the risk of falling are those relating to the individual. In the elderly population, these factors involve the age-related physiological wear and tear and / or degeneration of the different organ-systems, particularly balance, and the effects of acute or chronic conditions (WHO, 2012).

4.2.1.a. *Age-related changes*.

In this section chronic and acute disorders together with visual impairment will be discussed with special consideration given to how they hamper the individual's ability to remain stable on their feet.

4.2.1.a.1. *Instability.*

During the aging process the reflex mechanisms maintaining balance and the ability to quickly and effectively respond to their loss gradually deteriorate. Decreased responsiveness and / or speed of the reflex arc responsible for maintaining balance, muscle atrophy (primary and / or secondary) and poor mechanical joint hinder the implementation of a fast response to loss of balance (Sirven & Malamout, 2008). The physiological process of maintaining balance depends on a very complex reflex arc consisting of receptors and afferent pathways (visual system, peripheral sensorineural system, vestibular apparatus), motor nuclei, and efferent pathways (vestibular nuclei of the brainstem, cerebellar nuclei, cerebral cortex), together with peripheral effectors (the musculoskeletal system) (Sirven & Malamout, 2008).

The above mentioned conditions explain why the elderly often present postural instability and gait changes with a pattern of gait characterized by shorter and insecure steps, decreased hip range movement, toe separation increases in order to improve the individual's support base, and a motor reflex response to postural changes that is slow and inconsistent. Put differently, the elderly present more difficulty maintaining mechanical stability around their centre of gravity, which is disturbed by movement, and they have more difficulty generating reflex postural corrective manoeuvres. The appearance of certain diseases that cause alterations in gait as stroke, Parkinson's

disease, sensory, etc. increase instability and, therefore, the risk of falling (Gunter et al., 2003; Muir, Berg, Chesworth, Klar, & Speechley, 2010; Verfaillie, Nichols, Turkel, & Hovell, 1997).

4.2.1.a.2. *Sensory deterioration.*

In the older age groups it is typical the appearance of pathologies in the vestibular and vision systems such as cataracts, reduced visual acuity, reduced tolerance to light and dark adaptation, and hearing loss. Those conditions can cause an alteration in the ability to orientate with respect to objects in the environment. The loss of sensitivity appears to be more important than visual acuity in the origin of the falls, and the consequences of not seeing an object are often less traumatic than not being able to land a foot on a step with strength and precision (Lord, 2006; Nagamatsu, Liu-Ambrose, Carolan, & Handy, 2009).

4.2.1.a.3. *Modifications of the locomotor system.*

As people age biomechanical alterations of the joints appear due to degenerative or previous traumatic pathologies. This increases the likelihood of falls (Menz, Morris, & Lord, 2006). The deterioration of the articular mechanoreceptors produced by aging can lead to postural changes. At the level of the spine, the tone of the paravertebral muscles weakens, the thoracic spine kyphosis increases, and there is a higher occurrence of osteoporotic vertebral microfractures. The lumbar lordosis increases contributing to the centre of gravity being displaced forward. The elderly often wear inappropriate footwear and walk too fast for their ability, continuously trying to overcome their imbalance, as if they were in pursuit of their centre of gravity (Jessup, 2007). In the hip, articular cartilage degeneration ensues together with a reduced ability to mobilise the

joint. There is also progressive bowing of the lower limbs due to knee osteoarthritis and joint instability with decreased joint mobility. Those conditions make it increasingly difficult to go up or down the stairs. Loss of bone density is a determining factor in the production of fractures. Ultimately, the neck of the femur lengthens, the neck shaft angle develops an inwards angulation, and there is redistribution of the bone mass. All these conditions contribute to increase fracture risk or severity (Berry & Kiel, 2013; Menz et al., 2006; Shumway-Cook, Ciol, Gruber, & Robinson, 2005).

4.2.1.a.4. *Fear of falling.*

Many elderly people who have suffered a serious fall develop a fear of having another one and tend to make preventative changes like restricting their ambulation, which may result in isolation and depression over the loss of self-confidence (Vellas, Wayne, Romero, Baumgartner, & Garry, 1997). The research group noted that elders who had suffered a fall had coexisting problems related to mobility and cognitive functions. They also found that the increasing loss of confidence in their abilities means that these individuals often also avoid performing basic and instrumental activities of daily living. Regarding their caregivers, after the first fall, they usually react by being overprotective of their elderly relative, which often means an additional restriction of mobility. In this setting, relatives who cannot access or afford in-home care services tend to make the decision to institutionalise their relative (Dunn, Furner, & Miles, 1993).

Fear of falling is common in the elderly regardless of whether they have already had one fall or not. According to various studies there is a correlation between the fear of a further fall with restricted mobility and decreased functional capacity (Ayoubi et al., 2013; Resnick et al., 2014; Vellas et al., 1997). Elderly people with fear of falling seek

for points of support and for anchors they can grab in case of need. They sometimes adopt a triple flexion position (hip flexion, knee flexion and ankle dorsiflexion) with the feet wider apart to increase their support base (Ayoubi et al., 2013).

The main risk factors to develop a fear of falls are: age over 75 years, female, decreased mobility, impaired gait and balance, history of chronic vertigo, and having had a fall with a prolonged period (over 15 minutes) on the ground (Andresen et al., 2006; Ayoubi et al., 2013). Approximately 10% of the older people who fall report remaining on the floor for what they considered a long time. The average stay on the ground or floor was 12 minutes for people who did not suffer serious injuries and 19 minutes for those with serious injuries (Andresen et al., 2006; Ayoubi et al., 2013). Risk factors correlated with remaining on the ground after a fall were: age greater than 80 years, decreased muscle strength in lower limbs, arthritis, balance problems, the need to use canes and dependence to perform activities of daily living (ADL). About 50% of older people who fall need help to get up (Andresen et al., 2006; Ayoubi et al., 2013). The loss of self-confidence to perform basic ADL is a very limiting consequence of falls and is an important element of the post-fall fear syndrome. The syndrome refers to a change of behaviour and of attitude of the senior individual who has suffered a fall and includes restriction of mobility, fear of falling again, further loss of self-confidence and social isolation (Ayoubi et al., 2013; Resnick et al., 2014; Vellas et al., 1997).

4.2.1.b. *Falls risk conditions.*

Falls in the elderly population have a multifactorial origin (WHO, 2012). The higher the number of risk factors, the greater the likelihood of a fall, a relationship that is not

additive but multiplicative, meaning that each cause or risk factor potentiates the effect of another (Andresen et al., 2006; Yamashita el al., 2012).

4.2.1.b.1. *Chronic disorders.*

As people get older, the combined development of one or more chronic conditions that are falls risk factors increase in frequency. Apart from dementia, the neurological disorders involved in this increased risk are Parkinson's disease, delirium, cerebrovascular disease; sensory nerve pathology such as vertigo; cardiovascular diseases such as orthostatic hypotension, arrhythmias, heart failure and syncope; and musculoskeletal conditions such as osteoarthritis, foot deformities, osteoporosis, and pathological fractures (Carlson, 1997).

Some studies claim that mental disorders contribute both to the risk of a first fall and to that of successive ones. Dementia can increase the number of falls by impairing visuospatial perception and orientation. (Sibley, Voth, Munce, Straus, & Jaglal, 2014; Wood, 2013). Vertigo (established dizziness) is a hard to evaluate symptom as different people describe it very differently and because it has multiple aetiologies (James, 2013).

Syncope (sudden loss of consciousness with spontaneous recovery) is a rare but serious cause of falls caused by a rapid decrease in cerebral blood flow. Most often syncope is the result of a cardiovascular condition but in many cases a clear cause cannot be established (Anne-Kenny & O'Shea, 2002; Rubenstein & Josephson, 2002). Musculoskeletal pathology is a falls risk factor in the elderly because it tends to cause pain, joint instability, a decreased range of motion, and the appearance of inadequate joint positions. Advanced degenerative joint disease results in mechanical incongruity of the affected articulation, bone erosion, and the presence of osteophytes. The result is

an unsteady gait. The use of inadequate footwear that does not properly hold the foot or have slippery soles adds to the instability and to the proclivity to fall (Lloyd et al., 2009; Portegijs et al., 2006).

4.2.1.b.2. *Acute disorders.*

Infections, dehydration, anaemia, and fever are known risk factors for falls because of the physical challenge they pose, especially in an already frail elderly individual (Angalakuditi, Coley, Kirisci, Saul, & Painter, 2007; Batchelor, Mackintosh, Said, & Hill, 2012). Because of the effect of acute disease on mental state, they may result in different degrees of confusion. A fall in an elderly person may also be a presenting sign of a major disease such as myocardial infarction, stroke, or pneumonia. Acute disorders compound on chronic ones and significantly worsen instability and cognition in the elderly (Angalakuditi et al., 2007; Batchelor et al., 2012).

4.2.1.b.3. *Visual impairment.*

After the age of 65 there is a steady decrease in visual acuity, contrast sensitivity, tolerance for glare and field of vision, and there is a tendency to develop persistent miosis. Depth perception worsens from the age of 75. Another important issue, though easy to correct, is visual impairment due to refractive errors in old prescription glasses. Cataracts (opacities of the crystal lense) and glaucoma (high fluid pressure in the eye) on one side and macular degeneration (loss of vision in the centre of the visual field) on the other are, respectively, common causes of reversible and irreversible blindness in the elderly that influence the risk of falls (American Geriatrics Society, 2003).

4.2.2. Extrinsic factors.

Some falls are independent of the person's age or health status and relate to external conditions like substance consumption, medications, and environmental hazards.

4.2.2.a. *Iatrogenic factors.*

Iatrogenic causes of falls refer to those unintentionally caused by medical intervention and commonly means the effects of prescription medication (Bennett et al., 2014; DiPiro & Schwinghammer, 2011). Drugs are particularly important risk factors in falls because of their individual side effects; their interactions with other drugs, food, or drinks; and their influence on preexisting pathologies (Bennett et al., 2014). Medications can be a primary cause of falls or a secondary one by worsening underlying diseases. As aging occurs, pharmacokinetic changes modify the half-life of drugs and pharmacodynamic alterations can generate unexpected responses in this population group (Bennett et al., 2014; DiPiro & Schwinghammer, 2011).

Drugs that have an effect on the CNS are the most commonly involved in falls risk, like psychotropics, antihypertensives, those with extrapyramidal effects, and ototoxic drugs (Bennett et al., 2014). It should be keep in mind that is not uncommon in the elderly population to have poor treatment compliance due to memory lapses that may lead to a repeated administration of the drug (overdosing), confusion between different drugs, and self-medication (usually over the counter drugs or herbal remedies) (Bennett et al., 2014).

In a context of age-related impairment to metabolise and/or excrete drugs, polypharmacy, and comorbidities the adverse effects of drugs are more common in the

elderly and contribute to an increased risk of falling (Bennett et al., 2014). Because of the high prevalence of chronic diseases in the elderly, this population group is the one that consumes the largest amounts of medications. A risks - benefits evaluation of the age-related pharmacokinetic and pharmacodynamic alterations caused by each prescribed drug in the organism of older people with multiple pathologies is done by physicians before prescribing medications for their geriatric patients (Bennett et al., 2014).

Practically 80% of people aged 65+ are on some kind of medication, with two thirds of them taking at least two drugs (Bennett et al., 2014; DiPiro & Schwinghammer, 2011). Increased consumption is proportional to age. The number of drugs older people take daily ranges from five to twelve (Bennett et al., 2014). When two drugs are administered at the same time, the possibility of interaction is 6%, but the risk increases to 50% when five drugs are consumed, and it is 100% with eight drugs. The adverse effects of drugs in the elderly is very high and they can be particularly hazardous when psychoactive drugs are involved (Bennett et al., 2014).The consumption of three or more drugs is associated with an increased rate in falls in the elderly. Specific groups of drugs have a higher falls risk potential, especially those that may produce orthostatic hypotension, arrhythmias, confusion, drowsiness and tremors (Baranzini et al., 2009; Bennett, et al., 2014; DiPiro & Schwinghammer, 2011; Ziere et al., 2006).

4.2.2.b. *Alcohol.*

Alcohol may be a cause of unreported or hidden instability and falls. The characteristic ataxia of alcohol abuse increases when visual disturbances or proprioceptive alterations coexist (Tait, French, Burns, Byles, & Anstey, 2013). Confusion and reduced ability to

assess intrinsic and extrinsic falls risks add to the damaging effects of alcohol regardless of age.

4.2.2.c. *Lifestyle factors.*

Lifestyle factors related to falls risk refer to the individual's level of activity and the built - more than the natural - environment in which the person lives.

4.2.2.c.1. *Activity level.*

Most falls occur while doing activities of daily living (WHO, 2012). In the elderly population, a small percentage take place when performing common activities like reaching for something or getting in or out of the bathtub (WHO, 2012). A potentially dangerous activity is going down the stairs (more than going up) and this is how about 10% of falls occur. The risk is lower in adequate-for-age physically active seniors or in those who go up and down stairs regularly (Yu, Hwang, Hu, Chen, & Lin, 2013).

4.2.2.c.2. *The environment*.

A considerable proportion of falls happen because of inadequate built environments and most often occur at home (Hedman, Fonad, & Sandmark, 2013; WHO, 2012). Compared to nursing homes, it is difficult to find a home correctly adapted to the needs of elderly people (Hedman et al., 2013). Typical falls risk hazards at home are: cluttering, uneven floors, lack of adequate lighting, stairs without railings or landings, very high steps, inaccessible shelves, bathrooms with very low sinks or toilets, no grip handles in showers and toilets, too-high beds (this occurs more frequently in hospitals), ill-fitting shoes with slippery soles, and high, narrow heels. Once out of the usual

residence, further falls risks include uneven footpaths, poorly maintained pavement, highly polished and slippery (wet) floors, and lack of ramps (Hedman et al., 2013; Oliver & Masud, 2004).

4.3. Falls in Rest and Nursing Homes

Falls among the institutionalised elderly occur frequently and repeatedly (Oliver & Masud, 2004). The results are older adults being either disabled or dying each year from avoidable falls-related injuries. Those who survive frequently sustain further injuries, commonly more falls that result in permanent disability and reduced quality of life. About 5% of adults 65 and older live in nursing homes. Between 50% and 75% of nursing home residents fall each year. That is twice the rate of falls for older adults living in the community. Moreover, nursing home residents account for about 20% of deaths from falls in this age group (Oliver & Masud, 2004).

Each year, a typical nursing home with 100 beds reports 100 to 200 falls (Hedman et al., 2013). In the elderly, many falls are unwitnessed and many others go unreported (WHO, 2012). It is common for rest home residents to fall more than once. The average is between two and three falls per person per year. About 35% of fall injuries occur among residents who cannot walk independently because of a pre-existing disability (Hedman et al., 2013; Oliver & Masud, 2004). Not all rest home falls lead to serious injury. Although up to 20% of rest/nursing home falls cause serious injuries, only 6% cause fractures.

The most common causes of falls in residential and nursing facilities are shown in Table 12 on page 53. Falls result in disability, functional decline, and reduced quality of life, and fear of falling can cause further loss of function, depression, feelings of

helplessness, and social isolation for which psychotherapy is the treatment of choice (Nicholson, 2005).

Table 12

Most Common Causes of Falls in Institutionalised Older People

CAUSE	% OF FALLS
Gait problems (imbalance, muscle weakness)	24%
Environmental hazards (challenging terrain / home design, wet floors, poor lighting, inadequate bed height, defective wheelchairs)	16-27%
Substance use (sedatives, polypharmacy, alcohol)	19%
Socioeconomic factors (poorly fitting shoes, no access to walking aids, limited access to healthcare services)	6%

Note. Adapted from Alzheimer's NZ, 2005

Repeated falls are often an indicator of other health problems (WHO, 2012). People in nursing homes are generally frailer than older adults living in the community (Hedman et al., 2013; Oliver & Masud, 2004). They are usually older, have more chronic conditions, and have more difficulty walking. They also tend to have more advanced cognitive problems (mostly dementia), have difficulty with activities of daily living, and need help getting around or taking care of themselves. Although often better than in patients' private homes, fall prevention in nursing homes presents multiple challenges like individualisation of medical and psychological treatment, rehabilitation, and environmental changes (Oliver & Masud, 2004).

Falls prevention interventions (see Table 13 on page 54) can be implemented at institutional, staff, or patient levels. The most effective interventions address multiple factors and use a multidisciplinary team (Alzheimer's NZ, 2005; Hedman et al., 2013; Nicholson, 2005; Oliver & Masud, 2004).

Table 13

Falls Prevention Strategies in Institutions for the Elderly

STRATEGY
Policies and education about fall risk factors and prevention strategies in institutions and in the community
Preventative falls risk assessment in institutions and the community
Post-fall assessment to identify and address risk factors and treat the underlying compounding conditions
Structural changes in residential home like grab bars, raised toilet seats, lower beds, and ramps instead of steps
Implementing exercise programs to improve balance, strength, walking ability, and physical functioning
Teaching cognitively sound residents behavioural strategies to avoid potentially hazardous situation
Ensure adequate nutrition and hydration and treatment compliance
Provide mobility aids
Limit the use of unnecessary physical restraints

Note. Adapted from Alzheimer's NZ, 2005.

4.4. Falls and Death

Falls are the most commonly reported external cause of hospital admission for both males and females (WHO, 2012). Although fall rates are higher in the older age groups as noted in the literature reviewed in the current study, a full understanding of the contributing factors is limited by the fact that a large number of falls is considered to go unreported (WHO, 2012; Stevens & Rudd, 2014). In about 80% of deaths related to falls, the cause of the fall was unknown or not documented. Of the factors contributing to falls-related deaths ground-level tripping, slipping, or stumbling were the most common followed by falling from a building or structure, stairs or steps and on or from ladders (WHO, 2012). Falls reporting, early treatment, and prevention strategies are essential for injury prevention programme development and are essential to reduce falls-related death rates (Chisholm & Harruff, 2010; Korhonen, Kannus, Niemi, Palvanen, & Parkkari, 2013; Stevens & Rudd, 2014).

Chapter 5. Dementia

The following section deals with the definition, classification, incidence, management, and health and socio-economic impact of dementia. It will also address the intricate relationship between traumatic injury and dementia, or how a major traumatic event, regardless of its location, can directly or indirectly cause organic or functional brain damage leading to progressive cognitive impairment. The pros and cons of trauma-induced dementia being eventually considered for ACC will also be weighed up.

5.1. Definition of Dementia

Dementia is defined by the WHO (2012) as a syndrome – usually of a chronic or progressive nature – in which there is deterioration in cognitive function (i.e., the ability to process thought) beyond what might be expected from normal aging. It affects memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgement. Consciousness is not affected. The impairment of cognitive function is commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behaviour, or motivation (WHO, 2012). Dementia is thought to be caused by a variety of conditions that primarily or secondarily affect the brain (Braunwald et al., 2008). Regardless of the cause, dementia implies a gradual loss of brain function due to cerebral structural changes that hampers the individual's ability to cope and function at a personal, family, work and community levels (Alzheimer's NZ, 2015). About 50,000 New Zealanders are reported to suffer from dementia. Dementia is the most prevalent neurodegenerative condition among individuals of over 75 years of age (Braunwald et al., 2008; Sadock & Sadock, 2014). This means that the number of cases

in New Zealand is likely to be on the rise due to the increasing number of elderly in the population (Alzheimer's NZ, 2015; Statistics NZ, 2007).

5.2. Types of Dementia

Dementias can be classified according to various factors such as aetiology, prevalence, age of onset, brain location, and genetic factors (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.1. Classification by aetiology.

Regarding aetiology, dementias are divided into primary or idiopathic, i.e., those without a known cause such as Alzheimer's and Pick's disease, and secondary, where the dementia is the consequence of another condition like hydrocephalus or severe vitamin deficiency (Braunwald et al., 2008). Primary dementias are progressive and irreversible. Secondary dementias are potentially reversible and reflect the brain damage aftermath of other conditions. Such conditions include multiple sclerosis, infectious diseases, metabolic disorders, poisoning, substance abuse (including alcohol) and - in rare cases- brain tumours (Braunwald et al., 2008).

5.2.2. Classification by prevalence.

Alzheimer's disease is the most common type of dementia followed - in this order - by vascular dementia, dementia with Lewy bodies, fronto-temporal lobar degeneration and alcohol- related dementia (Alzheimer's Society, 2014).

5.2.2.a. *Alzheimer's disease*.

Alzheimer's disease (AD) is the most common cause of dementia in developed countries and represents about 65% of all the dementia cases in the world and in NZ (Alzheimer's NZ, 2015; Alzheimer's Society, 2014). The onset of symptoms usually begins after the age of 65 although in some patients it may occur before the age of 40, in which case it is usually associated with hereditary forms of the disease, which can happen in 25% of the cases (Braunwald et al., 2008). Initial symptoms are often mistaken for age-related concerns, or manifestations of stress. The disease process appears to be associated with abnormal protein deposits outside and inside the neurons, called plaques and tangles respectively. At first, the picture is limited to occasional failings of the memory, but afterwards a disturbance of recent memory (ability to store and retrieve new information after a period of time) and learning ability ensue, and with the passage of time there is loss of remote memory (remembering distant events). There is no cure for the disease, which worsens as it progresses to complete mental and bodily dysfunction resulting in death. No treatment currently exists that can stop or reverse its progression and therapy is merely symptomatic (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.2. b. *Vascular dementia*.

This condition is the second most common cause of dementia with up to 20% of the diagnosed cases (Alzheimer's NZ, 2015). Vascular dementia happens as the result of a stroke that interrupts blood flow to part of the brain causing the death of the affected neurons. When multiple strokes are involved the condition is called multi-infarct dementia. This is one of the few dementias that can be prevented by avoiding or controlling risk factors such as hypertension, hypercholesterolemia - which can cause

atherosclerosis - or diabetes (Braunwald et al., 2008). Although this type of dementia typically results from the accumulation of multiple white matter or cortical infarcts, cerebral haemorrhages have also been variably included in the pathogenesis. People with vascular dementia alternate moments of lucidity with moments of confusion and their personality typically remains unchanged unlike AD (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.2.c. *Dementia with Lewy bodies*.

Lewy body dementias include dementia with Lewy bodies (DLB) and Parkinson's disease with dementia (Braunwald et al., 2008). DLB is the third leading cause of dementia (<5%) in older people. The condition is named after Frederick Lewy, a neurologist who discovered the presence of tiny intracellular corpuscles in the brain cells of some patients with dementia (Alzheimer's Society, 2014). As the name implies, the typical feature of DLB is the presence of "bodies" or intracellular clumps of a protein called α- synuclein. These clumps are known as "Lewy bodies". The anatomical distribution of these clumps influences the types of symptoms: cognitive, motor, and psychiatric (Braunwald et al., 2008; Sadock & Sadock, 2014).

DLB is associated with low levels of neurotransmitters, mostly dopamine and acetylcholine, which explain the motor disability in these individuals. A controversial study by Ballard, Shaw, Lowery, McKeith, and Kenny (1999) suggested that having had five or more falls in three months could be sufficient diagnostic evidence to differentiate between DLB and AD. Another typical difference between the two disorders is the presence of early hallucinations and/or delusions in the former (Braunwald et al., 2008).

5.2.2.d. *Fronto-temporal dementia (Pick's disease).*

This is a degenerative disorder believed to affect about 2% of the population with dementia and is characterised by the presence of abnormal substances known as Pick bodies in the interior of neurons located in the frontal and temporal lobes of the brain (Braunwald et al., 2008). It usually affects middle-aged individuals, that is, those aged between 45 and 65. It is often a type of dementia that progresses slowly and its main clinical feature is a remarkable early change in the individual's personality (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.3. Classification by age of onset.

The traditional classification which distinguished between senile and presenile dementias or juveniles depending on the age of onset, is hardly used nowadays. It only serves to differentiate subtypes like the typical senile Alzheimer's dementia from sporadic juvenile or family Alzheimer´s disorder (Alzheimer's NZ, 2015). Presenile or early onset dementia has a clear inherited component and begins before age 65. It has a more rapid course and does not exceed 1% of cases in most statistics. Senile or late, dementia appears after age 65, is mostly sporadic and progresses slowly. This type accounts for over 98 % of cases (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.4. Classification by brain location.

Based on location, dementia can be classified as cortical or subcortical (Braunwald et al., 2008). Cortical dementias involve the bodies of neurons that specialise in memory, language, perception, thought, and attention, and are currently not curable. Examples of cortical dementias are Alzheimer's disease (AD), dementia with Lewy bodies (DLB),

fronto-temporal lobar degeneration (FTLD), and dementia pugilistica or chronic traumatic encephalopathy (CTE) (Braunwald et al., 2008). Subcortical dementias typically involve the neuronal bodies in the grey nuclei located deep in the brain and white matter tracts and are potentially curable. Among the most common subcortical dementias are metabolic dementias and Parkinson's disease dementia. Vascular dementia, depending on location, may be cortical or subcortical.

The boundaries between different forms of dementia are indistinct and mixed forms often coexist (Alzheimer's Society, 2014). Cortical dementias are a result of degenerative changes in the cerebral cortex. The cerebral cortex, or grey matter, is made of the cellular bodies of neurons. Cortical dementia is clinically evidenced by amnesia, aphasia, apraxia and agnosia, as well as difficulties in working memory. Subcortical dementias affect the basal ganglia, the thalamus, and the projections of these structures to the frontal lobe. They are characterised by more striking alterations in the level of patient supervision and care required, as well as additional difficulties in information processing and retrieval, psychomotor retardation, capacity for abstraction, impaired ability to develop strategies, and affection and personality disorders such as depression and apathy (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.2.5. Classification by genetic factors.

This classification has been better studied in Alzheimer's disease (AD) because it is the most common cause of dementia. Presenile, inherited, familial or early-onset AD has an autosomal dominant pattern, which means it is directly passed from parent to child (Braunwald et al., 2008). The suspected mutated genes involved are APP, PSEN1, or PSEN2. Because each gene synthesises one protein, a defective gene will produce a

defective protein. The defective protein in AD, known as beta-amyloid, does not serve any purpose and it is deposited in the brain forming plaques, which are characteristic of the condition. The buildup of this ineffective protein has toxic effects that result in the death of nerve cells and to the progressive signs and symptoms of AD.

The inheritance pattern of senile, sporadic, or late-onset AD is uncertain and its aetiology is considered to be multifactorial. Individuals who inherit one copy of one of the over 100 genes thought to be involved, have an increased chance of developing the disease but may not develop it (Braunwald et al., 2008; Sadock & Sadock, 2014).

5.3. Signs and Symptoms of Dementia

Dementia affects each person in a different way, depending on the impact of the disease and the individual's personality (Alzheimer's Society, 2014). The signs and symptoms of dementia also vary depending on the person's general health status, age of onset, and the disease stage (Braunwald et al., 2008; Sadock & Sadock, 2014). With regard to the latter, dementia can be classified as mild, moderate, and severe (Sadock & Sadock, 2014):

5.3.1. Mild dementia.

In this stage, incipient signs of dementia are often overlooked because the onset is so gradual. Common symptoms include: forgetfulness, losing track of time, and getting lost in familiar places.

5.3.2. Moderate dementia.

As dementia progresses, the signs and symptoms become more distinct and restricting. These include: behavioural changes, wandering, speech repetition, getting lost at home, increasing communication difficulties, needing assistance with activities of daily living and personal care.

5.3.3. Severe dementia.

In the late, or advanced, stage of dementia the person becomes completely inactive and dependent. Symptoms include: inability to recognise relatives and friends, time and place disorientation, abnormal behaviour (like aggression).

5.4. Incidence of Dementia

Worldwide, over 35 million people have dementia and almost 60% of them live in low- and middle-income countries (Alzheimer's Society, 2014). Every year, there are 7.7 million newly diagnosed cases. The estimated proportion of the general population aged 60 and over with dementia at a given time is between two to eight per 100 people. The total number of people with dementia is projected to double every 20 years, to 65.7 million in 2030. Prevalence rates for dementia in developed countries have been well established and are: 40-64 years: one in 1400, 65-69 years: one in 100, 70-79 years: one in 25, 80+ years: one in six (Alzheimer's Society, 2014). Currently, dementia affects just over 1% of the New Zealand population and the numbers are on the rise due to an increasingly ageing population (Alzheimer's NZ, 2015).

5.5. Treatment of Dementia

A pre-requisite for the treatment of dementia is having a diagnosis of dementia. The PNH, in keeping with WHO (2013) recommendations, uses the ICD-10 classification to label medical and mental disorders. Additionally, a considerable number of medical files of dementia admissions in the PNH often contained DSM diagnoses, in keeping with the American Psychological Association best practice guidelines (APA, 2002). The treatment of dementia aims to deal with the causes (curative treatment) and / or the symptoms (palliative treatment) of the condition (Alzheimer's NZ, 2015).

Currently there is no curative treatment for primary dementia, and interventions focus on managing treatable medical and psychological comorbidities (Alzheimer's Society, 2014; RANZCP, 2013). Medical treatment is the approach of choice of GPs, neurologists, and psychiatrists for aetiological and symptomatic management, and was the most common form of treatment mentioned in the database available for this study, which focuses on acute admissions of patients with dementia.

Emergency pharmacological treatment is necessary where the behavioural symptoms of dementia may pose a risk to the patient and to others, and medication is often required as an immediate action (RANZCP, 2013). The available medications most often prescribed to and used by people with dementia will be discussed next followed by the psychological approaches to dementia.

5.5.1. Medical treatment.

The most commonly diagnosed dementias in NZ are cortical and, at present, there is no curative medical treatment for them (Alzheimer's NZ, 2015). A diagnosis of dementia (cortical or not) does not exclude its comorbidity with other causes of dementia, usually subcortical dementias, which are potentially curable (Braunwald et al., 2008). Among the curable dementias are those due to hypothyroidism and severe depression. Dementia due to lack of vitamin B12 and / or folic acid is a relatively rare entity that should be suspected in certain contexts of malnutrition, alcoholism, and gastrointestinal diseases. Space-occupying conditions of the central nervous system (CNS) like normal pressure hydrocephalus, chronic subdural haematoma, or frontoparietal meningioma are also potentially reversible causes of dementia (Braunwald et al., 2008).

Not only is pharmacological treatment unavailable at present for the most prevalent types of dementia (Alzheimer's Society, 2014), it is unfortunately not even possible to say that the course of the condition can be altered with the current available medications like acetylcholinesterase inhibitors (Eagger & Harvey, 1995; Hughes et al., 2000). The low levels of acetylcholine detected in the affected areas of the brain in most cases of Alzheimer's disease led clinicians to the conclusion that increasing the concentration of the neurotransmitter in the brain would result in restoration of cognition (Eagger & Harvey, 1995; Hughes et al., 2000). One way to elevate the concentration of acetylcholine is to reduce the rate at which it is broken down by deactivating the enzyme responsible for it – acetylcholine esterase - at the level of the synaptic cleft. Acetylcholinesterase inhibitors like tacrine, donepezil, galantamine, and rivastigmine were used in the 70's with more adverse effects than benefits (Eagger & Harvey, 1995; Hughes et al., 2000).

The treatment of dementia based on other pathophysiological hypotheses, like those considered in the current study, includes substances that may modify the course of the disease by acting on cell metabolism, like antioxidants. The intermediate products of oxidative metabolism are highly reactive with proteins and lipids, with the potential to alter cell membranes and tissues. Selegine® is a levomethamphetamine derivative with potentially antioxidant effects that has shown molecular and clinical benefits in patients with neurodegenerative diseases such as Alzheimer's and Parkinson's. Using Selegine® and vitamin E appeared to act synergistically in patients with Alzheimer's (Crichton, Bryan, & Murphy, 2013; Desideri et al., 2010). Ginkgo Biloba is a substance that can be purchased over the counter and has antioxidant and antiplatelet actions (Crichton, Bryan, & Murphy, 2013; Gonenc, Hacisevk, Erdemoglu, Dagkiran, & Torun, 2013).

The use of nonsteroidal anti-inflammatory drugs is based on epidemiological and neuropathological studies suggesting a protective effect of this group of drugs in patients with risk of dementia (Breitner et al., 2009). However, the relatively high incidence of gastrointestinal bleeding due to chronic use limits their use. Propentofylline (HWA 285) is a new xanthine derivative that increases extracellular levels of adenosine, a molecule that interferes with the inflammatory process of glial cells in Alzheimer's disease and vascular dementia. Current trials have shown preliminary results that suggest that this medication not only brings about symptomatic improvement but it could also mitigate the progression of the underlying disease (Marcusson et al., 1997).

Finally, certain types of hormones have been thought to influence the growth of neurons in the CNS and reduce the production of beta-amyloid protein: estrogens. Some studies have suggested that hormone replacement therapy around menopause may reduce the

risk of Alzheimer's disease (Craig & Murphy, 2010; Sano, 2000), but there are discrepancies between studies. Additionally, the risk-benefit ratio does not warrant a widespread implementation of hormonal therapy in females with dementia. That said, the preliminary data from a comprehensive study showed a risk reduction of 40-50% in the treated group, regardless of APO-E genotype, ethnic group, or other demographic factors (Sano, 2000).

Although there is little in the way of curative treatment for idiopathic dementia, much can be offered to support and improve the lives of people with dementia and their caregivers. The principal goals for dementia care are an early diagnosis, improving physical and mental health, diagnosing and treating comorbid conditions, and providing information services and long-term support to caregivers (Alzheimer's Society, 2014; Alzheimer's NZ, 2015). Prevention, when possible, is the best available strategy for people with risk of dementia. The alternative comes in the form of palliative and psychological treatment.

5.5.2. Psychological treatment.

The Royal Australia and NZ College of Psychiatrists (RANZCP) (2013) clinical practice guidelines strongly recommend a non-pharmacological approach to be used alone or in conjunction with medical treatment for many mental health disorders including dementia.

The RANZCP (2013) psychosocial therapy recommendations refer to a person-centred approach in an adapted built environment free of restraints, in keeping with those previously mentioned in Table 13 on page 54.

A multiaxial DSM-V diagnosis and formulation of the case is the starting point of psychosocial treatment. Formulation is a diagram like that depicted in Figure 3 outlining the presenting problem and related predisposing, precipitating, perpetuating, and protecting factors, an approach known as the 5P's model (Merrick, 2013). It begins with the so-called ABC evaluation to characterise precipitating events and resultant behaviours. The evaluation involves the identification of antecedents, behaviours and consequences of the disorder.

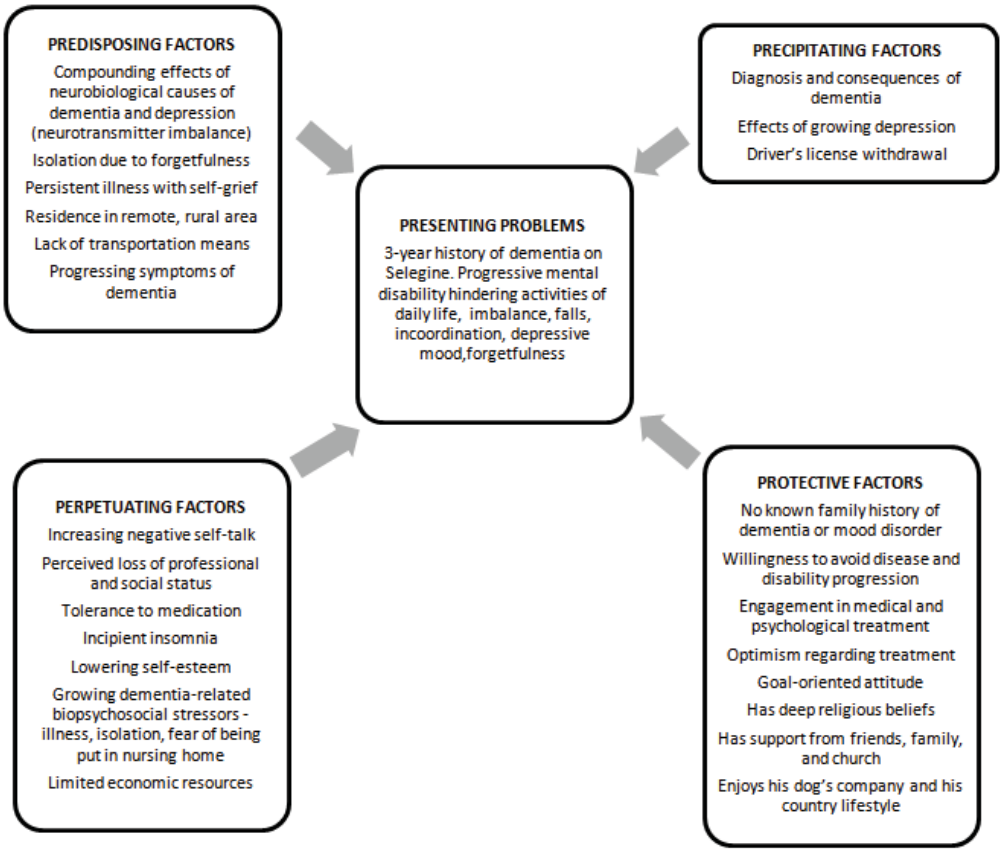

Figure 3. Formulation of a Sample Case of Dementia for Psychological Therapy. *Note*. Modified from Westerberg, 2013.

In the case of dementia, three forms of psychotherapy are recommended, namely validation, reminiscence, and orientation therapies (RANZCP, 2013). Validation therapy involves acknowledging the current feelings of the individual.

For example, a man with dementia may be agitated because he cannot find his (deceased) wife. Rather than reply that she has passed away, a more appropriate response is to acknowledge how much he must miss his wife and to then talk about her and them as a couple. Reminiscence therapy aims to stimulate memory in the context of the patient's life history through talking about some momentous past personal events such as marriage, children, work, a song or singer of their time, or major local and/ or international event. Orientation therapy is about helping the person situate him or herself in time, place and personal relationships and involves the use of clocks, personal mementos, and, ideally, regular visits from relatives and friends (RANZCP, 2013).

5.6. Prevention of Dementia

Dementia is one of the major causes of disability and dependency among older people worldwide (WHO, 2009; Alzheimer's Society, 2014). The burden of the disorder is overwhelming not only for the patient but also for their caregivers - often a relative. The impact of dementia on caregivers is physical, psychological, social and - very importantly - economic (WHO, 2009). Research identifying modifiable risk factors of dementia is scarce because of the complexity of the disorder in terms of comorbidities with other types of dementia and with medical and psychological conditions. Prevention focuses on reducing risks identified by research and includes diabetes, hypertension, obesity, smoking and sedentarism (Robertson, Brown, & Whalley, 2014; Solomon, 2014).

5.5.2. Social and economic impact of dementia.

Dementia has significant socio-economic implications in terms of medical costs, social costs, and the costs of informal care. In 2010, the total global societal cost of dementia was estimated to be US$ 604 billion (Alzheimer's Society, 2014). This corresponds to 1.0% of the worldwide gross domestic product. In year 2011 slightly under 50,000 New Zealanders had dementia, just about 1% of the NZ population, an increase of approximately 18% in three years since 2008 (Alzheimer's NZ, 2015). Females have a higher prevalence of dementia (60%) possibly due to the fact that they tend to live longer than men. It is estimated that by year 2050 over 2.6% of the population will have dementia, three times the current figures. The data just mentioned are mere approximations to reality as the Alzheimer's Disease International revealed that in NZ only 60% of cases are diagnosed / documented. This means that about 40% of people with dementia in NZ have not been -and may never be - diagnosed (Prince, Bryce, & Ferri, 2011).

The financial cost of dementia in 2011 was estimated to be close to one billion NZ dollars and it included not just the healthcare services provided to the patient but the financial burden on caregivers, who have to fully or partially withdraw from the jobs to care for their relative with dementia) (Alzheimer's NZ, 2015). Because of the strain dementia puts on patients, caregivers, society and healthcare systems, the WHO (2009) recognises dementia as a public health priority. The 2012 WHO report entitled "Dementia: A public health priority" aims to raise awareness about dementia.and advocates proactive public and private efforts to improve care and support for people with dementia and for their caregivers (Alzheimer's Society, 2014).

5.8. Traumatic Injury and Dementia

Traumatic injury to the brain (TBI) is the leading cause of long-term disability worldwide (WHO, 2012). A very recent NZ population-based incidence study in urban (Hamilton) and rural (Waikato District) area hospitals showed that the total incidence of TBI per 100,000 person-years was 790 cases (95%, CI 749 - 832). Over 38% of the TBI cases were due to falls, 20% were a consequence of traffic accidents, and 17% were the result of assaults (Feigin et al., 2013). The authors noted that the incidence of mild TBI was much higher than what statistics showed. Similarly, in the USA, Shively, Scher, Perl, and Diaz-Arrastia (2012) reviewed epidemiological data that linked severe, moderate and repeated mild TBI with early dementia, emphasising that the long term effects of repeated mild TBI was as exceedingly common as it was unreported and as damaging as a single episode of a higher grade TBI.

Dementias of the Alzheimer's and Parkinson's types have frequently been reported to develop in patients with TBI. Additionally, TBI may adversely affect multiple organ systems and accelerate neurological degeneration (Hesdorffer, Rauch, & Tamminga, 2009). The mechanisms believed to influence the incidence of dementia include accumulation of abnormal protein deposits and chronic inflammation. This means that it may be an alteration in the brain chemicals that may lead to the development of dementia, not just a genetic predisposition or mutations (Jawaid, Rademakers, Kass, Kalkonde, & Schulz, 2009) and that preventing or inhibiting the effects of chemical imbalance may prevent certain dementias in patients with a history of traumatic injury.

Whereas the relationship between TBI and dementia has been exposed in research studies over the years (Shively et al., 2012), the same cannot be said about major bodily

trauma and dementia. This is the justification and key specific goal of the current research.

The direct and indirect consequences of head injury alone have been implicated as a possible risk factor for Alzheimer's disease in a number of case-control studies (Dikmen, Corrigan, & Levin, 2009; Fleminger, Oliver, & Lovestone, 2003; Hesdorffer et al., 2009). Factors that hinder the understanding of the bodily responses to traumatic injury relate to the variables influencing the general mechanisms (type, intensity, duration, frequency) and individual responses (biology, demographics, and general medical condition) of the organism to trauma (Carlson, 1997).

Legal and economic implications are other relevant variables to consider. The assessment of post-traumatic body damage claims should be closely related to psychological damage assessment because they may both be the result of the action of an external noxa on the person, possibly leading to mental disorder. But for a mental disorder to be considered under the Accident Compensation Corporation (ACC) (2014) scheme and benefit from subsidised treatment, rehabilitation, and community or home support, the mental disorder should be a sequel of trauma. Preexisting or degenerative conditions are excluded from ACC, as are any kind of illness, psychological conditions related to ageing, all non-job related injuries that come on gradually, and certainly being a caregiver (ACC, 2014).

It may be challenging to make a clinically and economically sustainable case to get compensation for a condition like dementia which is likely to end up affecting the majority of individuals in a country like NZ that has a growingly ageing population

(Statistics NZ, 2007). Further research, however, could provide the solid evidence required that a traumatic event (major or repeated), regardless of its location, could directly or indirectly cause organic or functional brain damage leading to progressive cognitive impairment, that is, dementia, and that it should, therefore, be covered by ACC.

Chapter 6. Methods

This chapter describes how the researcher went about collecting and analysing the data of this retrospective, hospital-database, case-control study. Operational definitions (inclusion criteria) and exclusion criteria will be explained together with the ethics protocols followed and the statistical software and type of data analyses used.

6.1. Database

The PNH is a public hospital funded by the MOH. It is one of the major trauma centres in NZ and one of the largest teaching hospitals in the country. It is associated with the Otago and the Auckland medical schools. Its database consists of the medical claims data and registration files of a population of about 500,000 individuals (MCDHB, 2014). Honorary clinicians under supervision – as in the case of the Author - may be granted different clearances to access the database for research purposes.

Emergency admissions comprise around 65% of hospital admissions in NZ. Most of them are cardiovascular and respiratory emergencies, and a bit over 30% of the acute admissions are due to traumatic injuries (NZ Health Technology Assessment [NZHTA], 1998). The data in this study were obtained from the MCDHB patient database in the PNH, which has an administrative and not a clinical or research function. This remark is important because it means that the database is not designed to be searched for key words. Only database administrators can search for some ICD-10 codes as not all of them are recorded. For the purpose of this study, the database provided consisted of de-identified files.

The registry accessed was made up of files with information on patient demographics (age, gender, ethnicity, and area of residence), cause of admission, diagnoses, emergency department and hospital admissions, assessments / treatments, disposition types and discharge diagnoses, and patient outcome. Details on the PNH database generation, monitoring, and maintenance are published online by the MCDHB on their website under the "All Publications" tag (MCDHB, 2015).

6.2. Research Ethics

This study complies with the Massey University and the MCDHB Ethics protocols. A Massey University Ethics notification was approved on April 15, 2013 for the current research. The study was deemed to be low risk after peer review and as such it has been recorded in the Annual Report of the Massey University Human Ethics Committees.

Once Massey's Low Risk Notification was obtained, the researcher approached the MCDHB Chief Medical Officer, Dr Kenneth Clark, who a few weeks afterwards confirmed granting of approval for the researcher to hold Honorary Staff status with the MCDHB. It was effective from 20 May 2013 until 6 February 2015. The researcher's PNH staff supervisors were Dr Richard Fong and Prof Dr Anna Ranta. Dr Fong was the Author's database safety supervisor. He is the clinical advisor of the MCDHB Health Information and Data Quality Department. Dr Ranta, was the medical supervisor. She is a consultant neurologist and senior lecturer at the University of Otago.

The Hospital Ethics notification meant compliance of the author with the MCDHB pre-employment health screening requirements, emergency procedures, knowledge of

location of safety equipment and materials, identification and minimisation of hazards to which the researcher may be exposed to or create.

Because the MCDHB is constantly undertaking hazard identification and risk assessment, the researcher was informed that she may be included in this process during her time at the PNH. Additionally, copies of the MCDHB's "Code of Conduct Policy & Standards of Integrity" and the "Conduct and Disciplinary Procedures Policy" were given to the researcher for her knowledge and to ensure compliance with them.

The MCDHB's Vision motto is "Quality Living - Healthy Lives". As an Honorary Staff Member of MCDHB the researcher was encouraged to take every opportunity to assist the organisation to achieve that goal and she hopes that the current work be considered an honest effort in that direction.

6.3. Study Sample

To calculate the optimal sample size, Raosoft^{R} software was used. For a margin of error of 1.25%, a confidence level of 95%, a response distribution of 50% and a population sample of 390,550 (107 acute admissions per day in 10 years) (MCDHB, 2015), the sample size suggested was 6,052. The actual sample size used in this study was 6,943 records. The PNH acute admissions database considered ran from 1 April 2003 to 1 April 2013.

The Hospital uses the WHO (2013) International Classification of Diseases (ICD-10) which is the world's standard tool to capture mortality and morbidity data. In this study,

the ICD-10 codes served as operational definitions and inclusion criteria for both cases and controls.

Each disease category in the ICD-10 has a title indicating its composition and a unique alphanumeric code for identification purposes. A further aid to disease taxonomy in the ICD-10 includes the dagger ("†") and asterisk ("*") symbol system, which provides classification by aetiology and manifestation respectively (WHO, 2004). Simply put, an asterisk means that there is another associated coded condition that must be included as well, and the dagger means that there is a link to a causative condition of which the code must be included too.

The acute admissions database accessed included 5,598 files with a diagnosis of dementia, known in this study as “cases”. After exclusions (age over 25 years, not unconscious, no history of alcoholism) 4,541 cases remained of which the first 2,295 files sighted with a diagnosis relating to “traumatic injury” were selected. Cases were categorised as per the ICD-10 diagnostic codes as shown in Table 14 on page 77.

In the case of dementia, two ICD-10 codes are relevant: F and G. Most of the “F” codes are subsidiary codes and have to be indicated with an asterisk (*). However, the most important codes for cases are the “G” codes and they have to be used with the “dagger” (“†”) symbol. Similarly, controls were categorised using G codes and M codes, the latter referring to diseases of the musculoskeletal system and connective tissue.

Table 14

ICD-10 Codes for Dementia Cases

F CODES	DESCRIPTION	G CODES
F00*	Dementia in Alzheimer disease:	(G30.- †)
	Alzheimer disease with early onset (<65yrs)	G30.0
	Alzheimer disease with late onset (>65yrs)	G30.1
	Other Alzheimer disease	G30.8
	Alzheimer disease, unspecified	G30.9
F00.0*	Dementia in Alzheimer disease with early onset	(G30.0†)
F00.1*	Dementia in Alzheimer disease with late onset	(G30.1†)
F00.2*	Dementia in Alzheimer disease, atypical or mixed type	(G30.8†)
F00.9*	Dementia in Alzheimer disease, unspecified	(G30.9†)
F01	Vascular dementia	-
F01.0	Vascular dementia of acute onset	-
F01.1	Multi-infarct dementia	-
F01.2	Subcortical vascular dementia	-
F01.3	Mixed cortical and subcortical vascular dementia	-
F01.8	Other vascular dementia	-
F01.9	Vascular dementia, unspecified	-
F02*	Dementia in other diseases classified elsewhere	-
F02.0*	Dementia in Pick disease	(G31.0†)
F02.3*	Dementia in Parkinson disease	(G20†)
F02.8*	Dementia in other specified diseases classified elsewhere:	
	Dementia (in):	
	epilepsy.	(G40.- †)
	dementia with Lewy bodies.	(G31.83)
	frontotemporal dementia.	(G31.09)
	dementia in other diseases classified elsewhere	
	dementia without behavioural disturbance	
	dementia with behavioural disturbance	
F03	Unspecified dementia	
	Other degenerative diseases of nervous system	G31
	Circumscribed brain atrophy	G31.0
	Senile degeneration of brain, not elsewhere classified	G31.1
	Other specified degenerative diseases of nervous system	G31.8
	Degenerative disease of nervous system, unspecified	G31.9

Note. Adapted from WHO, 2013.

In order to select an adequate control group, the researcher opted for a neurological condition that, per se, did not involve cognitive impairment. The condition chosen was "peripheral neuropathy" and 2,451 files were obtained. The first 2,402 files with that diagnosis and no evidence of the exclusions were chosen as "controls". Controls were also categorised as per ICD-10 codes as shown in Table 15 on page 78. Cases and controls selection relied on the PNH emergency admissions as a measure of incidence, because for this type of study most individuals with a serious injury are going to end up

in hospital in the acute stage of their condition. Exposure status was determined by the medical records. It should be noted that cases and controls selection was done on the basis of admissions and that some patients had multiple admissions. Trimming the records list to one file containing all the variables relevant for this study per admission took additional meticulous work.

Table 15.

ICD-10 Codes for Controls

G CODES	DESCRIPTION	M CODES	DESCRIPTION
G50	Trigeminal neuralgia, G50.0, G30.0†, G50.1, G30.1†, G50.8, G30.8†, G50.9, G30.9†)	M50.0	Cervical disc disorder with myelopathy
G52	Disorders of other cranial nerves, G52.0, G52.1, G52.2, G52.3, G52.7, G52.8, G52.9	M50.1	Cervical disc disorder with radiculopathy
G54	Nerve root and plexus disorders	M51.0	Other disc disorders with myelopathy
G55*	Nerve root and plexus compression in diseases classified elsewhere,: G55.0, G55.1* (M50 – M51), G55.2* (M53-M54), G55.3*(M45–M46†, M48-†, M53–M54†), G55.8*	M51.1	Other disc disorders with radiculopathy (G55.1*)
G56	Mononeuropathies of upper limb: G56.0, G56.1, G56.2, G56.3, G56.4, G56.8, G56.9	M54.1	Radiculopathy
G57	Mononeuropathies of lower limb: G57.0, G57.1, G57.2, G57.3, G57.4, G57.5, G57.6, G57.7, G57.8	M54.2	Cervicalgia
G58	Other mononeuropathies: G58.0, G58.1, G58.10, G58.11, G58.12, G58.19, G58.7	M54.3	Sciatica (M51.1)
G59*	Mononeuropathy in diseases classified elsewhere	M54.4	Lumbago with sciatica
		M54.5	Low back pain
		M54.6	Pain in thoracic spine

Note. Adapted from WHO, 2013.

6.4. Data Analysis

The results of this retrospective, hospital-based, case-control research design were analysed on MedCalc^{R} using different types of statistics. In order to evaluate the differences in outcomes between dementia patients with and without trauma, descriptive statistics were used and calculated on the following data variables: age, injuries, and

hospital discharge types using the *t* test. Differences in history of trauma, injury mechanism, and types of discharge from hospital between cases and controls were compared using Pearson's χ^2 statistics given that the data are categorical and that the aim is to determine the percentage of discrepancy between the variables.

The *p* value selected in the case of history of trauma was $p < 0.0001$ with one degree of freedom and for the rest of the variables a standard $p < 0.05$ with three degrees of freedom. To assess the morbi-mortality of dementia, χ^2 tests were used to compare the differences between patients with and without TI in terms of demographic characteristics (patient's area of residence: Northern, Central, Eastern and Southern) and selected comorbidities (stroke, diabetes, hyperlipidaemia, hypertension, coronary heart disease (CAD), heart failure (HF) and atrial fibrillation (AF) at baseline.

Patients without TI were designated as the reference group, and the unadjusted and adjusted hazard ratio (HR) for the analyses were obtained by evaluating the association between TI and dementia during the 10-year period, after adjusting for demographic characteristics and selected comorbidities. The 10-year dementia-free rates were subsequently estimated by the Kaplane-Meier method using the log rank test to examine differences in dementia-free rates between cohorts. Stratified Cox proportional hazard regressions (stratified by sex, age, and year of admission) were performed to compare 10-year dementia-free rates between the two cohorts, after adjusting for geographic region and the selected comorbidities.

The relationship between TI and dementia was also explored in the different age groups. HR values along with a 95% confidence interval (CI) were computed with a

significance level of 0.05. The statistical analyses were conducted using Raosoft® and MedCalc® (Version 14.12.0) softwares.

Differences between ethnic groups were not included in the final statistical analysis because of concerns with the operational definition as discussed in the Ethnicity section of Chapter 1 on page 11. Ethnic-related health inequalities, mostly between individuals of European vs. Māori descent have been, however, highlighted throughout this study.

Chapter 7. Results

This chapter focuses on the main objectives that drove the researcher to conduct this study in the first place. The main goal was to bring about awareness regarding the relationship between a history of major traumatic injury (TI) and subsequent development of dementia. Specific objective number one was to determine whether dementia may be associated with an increased risk and incidence of trauma in the past and whether such an association may be explained by the injuries or by medical comorbidities. The second specific objective was to identify any possible differences in the mechanisms of injury and forms of discharge from hospital for individuals with and without dementia.

7.1. Specific Goal #1

A key objective of this study was to determine whether dementia was associated with a higher incidence of trauma in the past and whether such an association might be explained by the injuries or by medical comorbidities. From the PNH dementia database 2,295 TI cases and 2,246 non-TI controls were selected for comparative analysis. No significant differences in age or sex were observed between the groups. Demographic characteristics and comorbidities of the TI and non-TI patients are shown in Tables 16, 17, and 18 on page 82. The mean age of the patients in the database was 70.21 years (*SD* 18.30 years) and 55.30% of them were men. After matching for age and sex, TI patients had a higher prevalence of stroke ($p < 0.0001$), diabetes ($p<0.0001$), hyperlipidaemia ($p < 0.001$), hypertension ($p < 0.0001$), coronary artery disease ($p < 0.0001$) and heart failure ($p < 0.0001$) than those without TI. There were no significant

differences in the distribution of other comorbidities, such as atrial fibrillation, between the groups.

Table 16

Comparison of Demographic Characteristics (Gender and Age Groups) Between Traumatic Injury and Non-Traumatic Injury Patients

DEMENTIA	TI (n=2,295)		no TI (n=2,246)		*p* value
	n	%	n	%	
Males	1354	58.99	1158	51.56	1.0000
Females	941	41.11	1088	48.44	1.0000
Age					
25-34	138	6.01	144	6.41	1.0000
35-54	184	8.02	184	8.20	1.0000
55-64	205	8.92	202	9.00	1.0000
65-74	873	38.07	848	37.75	1.0000
75+	895	39.00	868	38.67	1.0000

Table 17

Comparison of Demographic Characteristics (Region of Residence) Between Traumatic Injury and Non-Traumatic Injury Patients.

DEMENTIA	TI (n=2,295)		no TI (n=2,246)		*p* value
	n	%	n	%	
Region 4	885	38.56	1077	47.89	<0.0001
Region 3	581	25.31	507	22.61	<0.0001
Region 2	717	31.24	562	25.04	<0.0001
Region 1	75	3.26	68	3.05	<0.0001

Table 18

Comparison of Comorbidities Between Traumatic Injury and Non-Traumatic Injury Patients with Dementia

DEMENTIA	TI (n=2,295)		no TI (n=2,246)		*p* Value
	n	%	n	%	
Comorbidities					
Stroke	24	5.33	5	2.44	<0.0001
Diabetes	17	3.98	7	3.14	<0.0001
High cholesterol	19	4.29	9	4.00	<0.001
Hypertension	36	8.12	15	6.86	<0.0001
Coronary disease	20	4.49	8	3.60	<0.0001
Heart Failure	6	1.45	2	1.13	<0.0001
Atrial fibrillation	1	0.33	0.642	0.29	<0.001

The crude and adjusted HRs for dementia during the 10-year period between the cohorts are shown in Table 19 on page 84. In the study population of 390,550 patients in the acute admissions database, 4,541 (or 1.16 %) were cases with dementia and 2,402 (0.61%) were non-dementia controls. Stratified Cox proportional hazard regressions (stratified by sex, age group, and year of admission) showed that the HR for dementia in traumatic injury patients within the 10-year period was 2.06 (95% CI 1.93 to 2.20; $p<0.001$) in comparison with the non-TI patients.

The HR value was analysed after adjusting the data for stroke, diabetes, hyperlipidaemia, hypertension, CAD, HF, and AF. The HR value thus obtained was 1.790 (95% CI 1.678 to 1.910; $p < 0.001$). Figure 4 below shows the dementia-free curves using the Kaplane-Meier method.

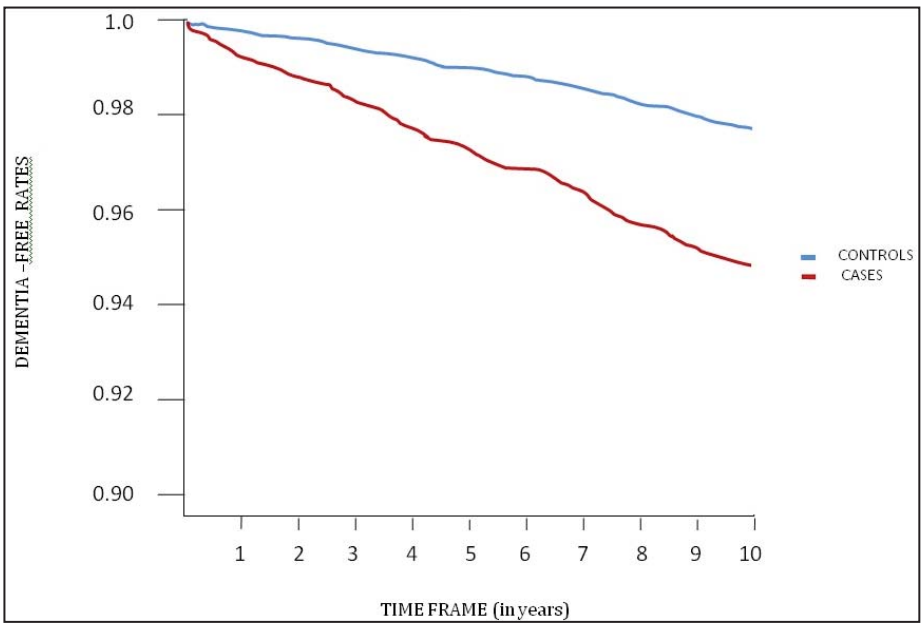

Figure 4. Dementia-Free Rates for Cases (in Red) and Controls (in Blue) for the 10-Year Study Period

Table 19

Ten-Year Dementia Risk Estimates for Cases and Controls with a 95% CI HR

Dementia occurrence	Total (n=6,943)		Patients with traumatic injury (n=3,129)		Patients without traumatic injury (n=3,314)	
	N	%	n	%	n	%
Yes	4,541	65.40	2,295	73.35	2,246	67.77
No	2,402	34.60	834	26.65	1,563	47.16
Crude HR			2.06* (1.93 - 2.20)		1.00	
Adjusted HR			1.68* (1.57 - 1.80)		1.00	

Note. *$p < 0.001$. HR was calculated using the stratified Cox proportional regression method stratified by sex, age group, and admission year. Adjustments were made for selected comorbidities (stroke, diabetes, hypertension, CAD, HF, and AF).

Stratified Cox proportional hazard regressions (HR) yielded the following results for the 10-year period (see Table 20 on page 85): in the age group 35-54 years, the HR of TI patients was 2.03 (95% CI 1.61 to 2.56; $p < 0.001$); and in the age group 65-74 years, the HR of TI patients was 1.43 (95% CI 1.27 to 1.60; $p < 0.001$). The HR for TI patients older than 75 years was 3.69 (95% CI 3.33 to 4.10; $p < 0.001$). Furthermore, the adjusted HR for dementia during the 10-year period in TI patients aged 35-54 years was 1.74 (95% CI 1.38 to 2.21; $p < 0.001$); the corresponding value in TI patients aged 65-74 years was 1.27 (95% CI 1.13 to 1.42; $p < 0.001$) whereas in patients older than 75 years, the HR value for TI patients was 2.60 (95% CI 2.33 to 2.91; $p < 0.001$). No significant differences between groups were observed for the 25-34 year olds (HR 1.05, 95% CI 0.86-1.30, $p < 0.001$) or for the age group of 55-64 years (HR 1.06, 95% CI 0.87 to 1.29, $p < 0.001$).

Table 20

Crude and Adjusted HR with a 95% CI for Dementia Among Sampled Patients of Different Age Groups Between 1 April 2003 and 1 April 2013

DEMENTIA GROUP	Total n = 4,541		Trauma patients n = 2,295		Non-trauma patients n = 2,246	
Age group	n	%	n	%	n	%
25-34	7	0.14	4	0.17	3	0.13
Crude HR			1.05 (0.86-1.30)		1.00	
Adjusted HR			1.01 (0.81-1.20)		1.00	
35-54	7	0.14	5	0.22	2	0.13
Crude HR			2.03 (1.61-2.56)		1.00	
Adjusted HR			1.74 (1.38-2.21)		1.00	
55-64	13	0.29	6	0.26	7	0.29
Crude HR			1.06 (0.87-1.29)		1.00	
Adjusted HR			1.00 (0.81-1.21)		1.00	
65-74	32	0.69	18	0.81	14	0.66
Crude HR			1.43 (1.27-1.60)		1.00	
Adjusted HR			1.27 (1.13-1.42)		1.00	
75+	34	0.75	25	1.33	9	0.43
Crude HR			3.69 (3.33-4.10)		1.00	
Adjusted HR			2.60 (2.33-2.91)		1.00	

7.2. Specific Goal #2

Another objective of the current research was to identify the mechanisms of injury and types of discharge from hospital for trauma patients with and without dementia. To compare data to those of the patients of the same age range without a diagnosis of dementia and to identify the injury patterns seen in the patients who were admitted with dementia. Between 1 April 2003 and 1 April 2013 there were approximately 390,550 acute admissions in the PNH, an average of 107 patients a day (MCDHB, 2015). Among the acute admissions, 1.43% (or n = 5,598) had a preexisting diagnosis of dementia and an average age of 82.1 years (range 40-102). After the exclusions, 4,541 patients were considered in this study. The 2,451 controls or non-dementia admissions (0.63% of the total admissions) during the same time frame had an average age of 59.3 years (range 29-98). The number of eligible controls was 2,402. Most patients with dementia and a history of trauma reported their injuries to be due to a fall (1,928 of 2,295); 176 due to a traffic accident; 135 due to a laceration, 37 due to blunt weapon injury, and 19 due to leisure activity injuries. The most common injuries for dementia and non-dementia patients included head-neck soft tissue injuries, femur fractures, concussions, and intracranial haemorrhage (see Table 21 on page 86). Under one third of the patients without dementia (697 of 2,402) referred a fall as the cause of injury. In

the latter group, 816 of the injuries were due to traffic accidents. Patients with dementia were significantly older than those without dementia (82.1 versus 59.3; $p < 0.0001$). Patients with dementia were also significantly more likely ($p < 0.05$) to be discharged to a residential institution or a specialised care facility, like a nursing home, and significantly less likely ($p < 0.05$) to be discharged to their home (see Table 22 on page 87). Falls and traffic accidents were the two most common injuries in both groups. Patients with dementia were significantly more likely to be injured by a fall in comparison to those without dementia (84.0% vs. 29.1%, respectively), who were more likely to experience a traffic accident (34.0% versus 7.7%, respectively, $p < 0.0001$).

Table 21

Injury Types Among Trauma Patients With and Without Dementia.

INJURY TYPE	Traumatic Injury with dementia (n=2,295)		Traumatic injury without dementia (n=834)	
	n	%	N	%
Head and Neck STI[a]	867	37.8	327	39.2
Femur Fracture	826	36.0	317	38.0
Concussion	521	22.7	242	29.0
Intracranial Haemorrhage	482	21.0	187	22.4
Upper Leg STI	369	16.1	124	14.9
Forearm STI	209	9.1	71	8.5
Trunk STI	167	7.3	75	9.0
Cervical spine fracture	161	7.0	137	16.4
Lower leg STI	160	7.0	96	11.5
Facial fracture	144	6.3	77	9.2
Rib fracture	142	6.2	60	7.2
Upper arm fracture	137	6.0	52	6.2
Hand STI	135	5.9	26	3.1
Lower Leg fracture	133	5.8	53	6.3
Skull fracture	103	4.5	46	5.5
Lumbar spine fracture	87	3.8	18	2.1
Lower Leg STI	82	3.6	33	4.0
Pelvis fracture	80	3.5	17	2.0
Upper arm fracture	80	3.5	21	2.5
Thoracic spine fracture	71	3.1	20	2.4
Forearm fracture	57	2.5	23	2.7
Intra-thoracic injury	55	2.4	39	4.6
Hand fracture	48	2.1	28	3.3
Intra-abdominal injury	39	1.7	20	2.4
Foot fracture	23	1.0	13	1.6
Foot STI	16	0.7	10	1.2

Note. [a] Soft tissue injury (STI) means: lacerations, contusions, abrasions, haematomas, and avulsions. STI excludes fracture to the body area. Percentages refer to the proportion of trauma patients with dementia per diagnostic category. Total percentages exceed 100% due to multiple diagnoses per patient.

Table 22

Discharge Types (in Parentheses) and Codes (in Capitals) for Dementia and Controls.

DISCHARGE TYPE	% DEMENTIA	% CONTROLS	*p* VALUE*
DD, ED (morgue)	8.69	1.80	>0.001
DR, ER, DS, DI (usual residence)	22.03	70.05	<0.001
DW, DP (rehabilitation services)	11.89	14.97	>0.001
DT (to another health institution)	28.32	2.03	<0.001
EI, ET, SNU (nursing)	26.92	7.42	<0.001
DF (home health)	1.05	2.69	>0.001
DC (community care)	0.70	0.89	>0.001
DA (home - other NES)	0.40	0.15	>0.001

Note. * Significant differences on *t* test for the differences between percentages are those with a *p* value <0.001

The results show that there is a significant difference in trauma occurrence between the dementia group (33%, n = 2,295) and the no dementia group (12%, n = 834), χ^2 (1, n=6,943), $p < 0.001$. In other words, this study shows that individuals with dementia are more likely to have a history of trauma than those without dementia and that they are almost three times more likely than the non-dementia patients not to be discharged to their usual residence if they lived independently.

Chapter 8. Discussion

Based on the data in the previous chapter, this section aims to illustrate how the key findings in this study are explained and related. The how and why traumatic injury and dementia constitute major health and socio-economic problems contributing to long-term disability will be evaluated. This chapter also addresses the implications for healthcare service delivery and for medico-legal compensation of the Results section.

8.1. Discussion about Traumatic Injury and Dementia Findings

The main finding of this study was that TI is a risk factor for the development of dementia. The scanty but growing literature on the topic of traumatic injury in general - and not only to the brain - and dementia, warrants an explanation of the rationale for the hypothesis in this study: that the consequences of traumatic injury, especially in the elderly, regardless of its location, may play a role in the development - or maybe progression - of dementia. Those consequences, as will be discussed next, include toxic protein deposits, high inflammatory mediators levels, and apoptosis (self-induced nerve cell death).

The way the body responds to traumatic injury is both locally – at the site of injury – and systemically – adjusting the different organ-systems to protect them and to improve wound healing (Braunwald et al., 2008; Winterborn & Cook, 2003). The effectiveness of the healing process - including healing time - depends on variables like age, sex and severity of the trauma. Common symptoms of severe trauma include confusion, fever, tachycardia, and generalised oedema. The less vital organ-systems like the skin, the gastrointestinal system, and the urinary system respond to major trauma by shutting

themselves down in order to maintain homeostasis in the most vital organs, namely, the heart and brain (Price & Wilson, 2002; Winterborn & Cook, 2003). Additionally, after severe injury an inflammatory response ensues to protect against further damage and to start the healing process. During the process of inflammation, a number of substances known as inflammatory markers appear in the blood and in the fluid around the cells to promote healing. During the acute (early) phase of inflammation markers like C-reactive protein (CRP), interleukins, and α-tumoral necrosis factor (α-TNF) are elevated. As time passes, chronic (late) markers of inflammation take over. One of those is cortisol (Johansson et al., 2011). If the trauma is severe or sustained, the defence mechanism that the inflammatory response is supposed to be may turn into a damaging process.

Whether acute or chronic, the inflammatory response may persist for an extended period of time and potentially result in life threatening conditions like multi-organ dysfunction syndrome or systemic inflammatory response syndrome (Marino, 1998). Chronic inflammation has also been aetiologically related to certain cancers but its association with degenerative neurological disorders is not as clear (Chaturvedi et al., 2011). Although the relationship between inflammation and cognitive impairment is not completely understood, the neurologic, genetic, and epidemiologic evidence supports the link (Weaver et al., 2002; Yaffe et al., 2003).

The most prevalent form of dementia, Alzheimer's disease (AD), is characterized not only by protein deposits but, according to recent research, also by chronic inflammation in certain regions of the brain related to AD, namely the hippocampus, the medial temporal lobe, and the parietal lobe (Dziedzic, 2006; Giunta et al., 2008; Wilson, Finch,

& Cohen, 2002). The fact that some studies have shown that non-steroidal anti-inflammatory drugs may delay the progression of AD (Dziedzic, 2006) support the inflammatory theory for dementia. In that line, several population-based studies have found a link between acute inflammatory markers and cognitive impairment. One study found that baseline c-reactive protein (CRP) predicts the risk of developing dementia over 25 years (Schmidt et al., 2002). Another representative study found that high levels of α1-antichymotrypsin, interleukin-6 (IL6), and CRP increased the risk of dementia after seven years of follow-up (Engelhart et al., 2004).

Whereas an association between inflammatory response and dementia has been shown in a small number of studies, the same cannot consistently be said for the non-dementia population, as supported by the results of the study at hand. However, Schram et al. (2007) found evidence of cognitive decline in individuals without dementia who had high levels of CRP and / or IL6. Similar results were obtained by Weaver et al. (2002) concerning the link between IL6 and cognitive decline, but other researchers could not find equivalent results. For example, in 2006, Weuve and his study group concluded that they could not find a link between CRP and cognitive function in their study with individuals without dementia. However, Jordanova et al. (2007) found that persistently increased levels of IL6 partly predicted subsequent cognitive decline. On the other hand, two separate 2008 cross-sectional studies, one by Alley et al. and another by Baune et al., failed to show any significant relationship between an elevated IL6 and cognition. Some longitudinal studies seem to contradict the lack of association between inflammatory markers and cognitive function in the non-dementia population. A consistently high CRP on baseline predicted cognitive impairment in a 12-year follow up study by Komulainen et al. (2007) in healthy elderly women. High serum levels of

IL-6 and/or CRP and/or plasma levels of α-TNF were shown to be prospectively linked with cognitive impairment in well-functioning elderly individuals by Yaffe et al. (2003, 2004). With regard to the relationship between chronic inflammatory markers, like cortisol, and dementia, the interest and the number of studies is notably scarce. In a recent cross-sectional study, Johansson et al. (2011) noted that patients with dementia had higher 24-hour urine cortisol levels than the controls. Likewise, increased plasma cortisol levels were shown to be associated with a faster progression of preexisting dementia in a study by Csernansky et al. (2006). On the other hand, a large prospective population-based cohort study known as "The Rotterdam Study" conducted by Schrijvers et al. (2011) concluded that no relationship could be demonstrated between serum levels of cortisol and a higher risk of developing dementia. The general conclusion of the studies on cortisol and dementia is that higher levels of cortisol can be observed in persons with cognitive decline and dementia but that it is not known whether those higher levels are a cause or a consequence of the dementia process or of comorbidities.

Traumatic injury not only sets inflammatory and immunological responses in motion, metabolic changes also ensue concurrently, with an increased production of glucose (or gluco-neo-genesis) which involves the consumption of fat or lipolysis. Following gluconeogenesis, the body undergoes a process called anabolism which aims to restore the spent storage of glucose (the main source of energy in the body) and protein. In this state the body consumes very high amounts of energy, much more than in the resting state, for the purpose of synthesising substances to heal the injured tissues (Price et al., 2002; Winterborn & Cook, 2003). And just like during any other building process, a lot of debris is produced. In the context of major trauma, the injured area - and often the

patient - are immobilised for a considerable time. The blood flow slows down leading to generalised dysfunction of the cellular excretion material elimination process and to the local deposition of metabolic by-products. The normal homeostasis is altered, including the extracellular pH levels, essential for proper metabolic and immune function. The cells find themselves in a toxic environment from which they will have to try to absorb the necessary nutrients to synthesise their own structures and substances (Price et al., 2002; Winterborn & Cook, 2003). Particularly important is the synthesis of proteins in the pathophysiology of dementia. If defective proteins are produced, they may accumulate and have insufficient or adverse effects on their target organs. Simply put, during major trauma many organs shut down and the brain receives less blood than usual, at a lower rate, and with more potentially damaging substances in it.

Beta-amyloid peptide deposition is known to occur after TBI, similar to the deposition seen in Alzheimer's patients. Repeated mild head trauma in both animals and humans accelerates amyloid beta peptide accumulation and cognitive impairment. Supporting the study at hand, Yaffe et al. (2003) found that inflammation is associated with an increased risk of cognitive deterioration in the elderly. Exactly how disturbances of production and aggregation of the proteins involved in the pathophysiology of dementia, especially of Alzheimer's disease (AD), is not yet well understood. Certain cytokines (inflammatory signallers) and an increased microglial activation rate are strongly thought to have a role in the pathology of AD (Chaturvedi et al., 2011).

Inflammation is a general marker of tissue damage in any disease, and may be either secondary to cellular injury - in the case of AD - or just be a marker of the immunological response (Greig et al., 2004). At the cellular level, direct or indirect neuronal damage initiates a chain of events known as axonal degeneration and neuronal

regeneration. Neurons, Schwann cells, oligodendrocytes, macrophages, and microglia are involved in these responses. Unlike in the peripheral nervous system, in the CNS injured axons usually cannot regenerate (Ross & Pawlina, 2006). The removal of debris after injury is essential to the neuronal regeneration progress as excessive debris hampers or stops the process. The first sign of injury, which takes place 8 to 24 hours after the axon has been damaged, is axonal swelling followed by axonal disintegration (see Figure 5), which means the breakdown of the axonal cytoskeleton (Ross & Pawlina, 2006). This is explained by the fact that when support cells, namely oligodendrocytes, lose contact with axons, they respond by initiating apoptosis - programmed cell death.

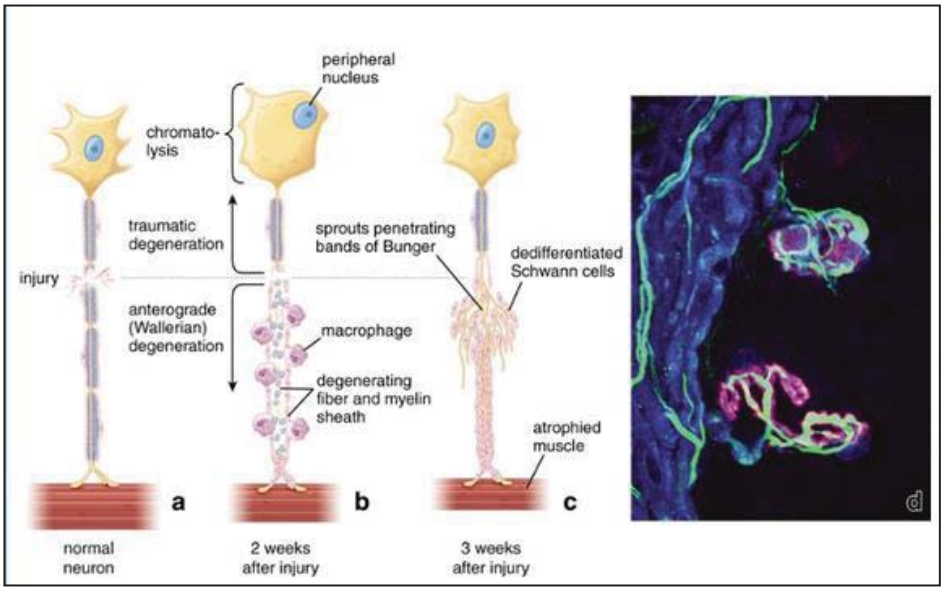

Figure 5. Response of a Nerve Fibre to Injury.
Note. From Ross and Pawlina, 2006

Tissue macrophages, the cells in charge of debris removal, become activated after nerve injury. They migrate to the site of nerve injury, proliferate, and then engulf myelin debris. An inefficient clearance of debris together with reactive gliosis (scar formation

resulting in a plaque) are major factors in the failure of nerve regeneration in the central nervous system (CNS) (Ross & Pawlina, 2006). Moreover, nerve injury can lead to changes in gene expression. The changes in the cell body are proportional to the amount of axon destroyed by the injury and extensive loss of axoplasm can result in neuronal death. Gliosis is a consistent feature of many disorders of the CNS, including stroke, neurotoxic damage, genetic diseases, inflammatory demyelination, and neurodegenerative disorders (Ross & Pawlina, 2006). Prevention or inhibition of glial scarring could, theoretically, improve neural regeneration.

Having said all of the above, none of the mentioned studies were done in the general trauma population, excepting some which involved the specific TBI population. They were all conducted with populations where inflammation is known to have a role in the patients' underlying condition like the metabolic syndrome and the cancer populations. As in the studies in the literature mentioned, the cases in the database of this study had comorbid conditions like cancer and cardiovascular, gastrointestinal, respiratory and urinary tract disorders. In comparison with patients without TI, patients with TI were more likely to have stroke, diabetes, hyperlipidaemia, hypertension, coronary artery disease and/or heart failure. Because patients with TI receive more medical care after injury, it makes sense to assume that comorbidities are more likely to be identified and treated sooner in this group of individuals than in non-TI patients. Whether those conditions and not the trauma itself are responsible for the findings remain to be determined by further future studies.

8.2. Discussion about Traumatic Injury Types and Dementia Outcomes

The current research found not only that there was a link between traumatic injury and dementia, but that the outcomes were worse for people with dementia than for the non-cognitively impaired. It also showed that traumatic injuries in individuals with dementia may present a combination of problems for the patients, caregivers, and healthcare providers. Dementia is an established risk factor for falls (WHO, 2012). What the current study showed was that falls (the most common form of traumatic injury in the elderly population) had the potential to result in dementia. Needless to say, people with dementia are not precluded from suffering other forms of traumatic injuries including traffic accidents and penetrating trauma. The morbidity and mortality of traumatic falls has been extensively documented in the literature (Chisholm & Harruff, 2010; Gryfe, Amies, & Ashley, 1977; Scott & Gallagher, 1999; Stevens & Rudd, 2014).

With regard to NZ, as the elderly population throughout the MidCentral Region increases, the number of patients with dementia who may require hospital admission for trauma may similarly increase, as the older population is at an increased risk of falls and the associated health outcomes (Statistics NZ, 2007; Alzheimer's NZ, 2015).

In the present study, the most commonly injured areas were the head and neck and the femur. The majority of injuries in the dementia group were reported to have been sustained in falls. Over 35% of the dementia patients had a femur fracture and slightly over 20% of the dementia patients had an intracranial haemorrhage (ICH). Hip (upper femur) fractures in the elderly, especially in patients with dementia, had a considerable morbidity and mortality with about 20% of dementia patients with a hip fracture typically dying in the first two years after injury. After a hip fracture dementia patients

were unable to regain their pre-injury ambulatory status as also noted by Shumway-Cook et al. (2005). In addition to this, these individuals were also at a high risk of sustaining a fracture in the contralateral hip.

Intracranial haemorrhages are another serious consequence of traumatic injury in people with dementia. They often result in worsening of their preexisting mental status, weakness, sensory deficits, higher rates of lower respiratory tract infections and cardiovascular instability and are predictive factors of morbidity and mortality (Sirven & Malamut, 2008). Moreover, because cardiovascular-related conditions increase with age, many elderly patients are on antithrombotic therapy, which increases the risk of intracranial haemorrhage with even minor trauma (Braunwald et al., 2008). That said, and given the high risk of falls in dementia patients, the decision to prescribe antithrombotic drugs involves a difficult risk-benefit evaluation.

In this study, patients with dementia admitted due to traumatic injury were more likely to be discharged to a nursing facility or a specialised care centre compared to those without dementia (55% vs. 9%). Only 22% of patients with dementia were discharged to their usual residence following trauma compared to almost 73% of patients without dementia. Seitz et al. (2014) found that patients with severe dementia who were hospitalised for hip fracture had a significantly poorer prognosis than those without cognitive impairment. There is controversy as to whether patients with dementia should be referred to rehabilitation services because success is largely dependent on the pre-morbid level of function. Individuals with dementia progressively lose the ability to walk, which predicts a less favourable outcome of rehabilitation after hip or lower extremity fracture (Bellelli & Trabucchi, 2007; Giusti, Barone, & Pioli, 2007).

In the current study 12% of the patients with dementia were discharged to a rehabilitation facility; they were most commonly discharged to a nursing home (27%) or to a specialist centre (28%). Those without dementia were most commonly discharged to their usual residence (70%) or to rehabilitation services (15%). Discharge dispositions are a transition from the hospital to the best recovery facility for the patient (Shepperd et al., 2013). When and where a hospitalised patient should be discharged implies evaluating a number of factors from medical, to psychosocial, logistic, and economic considerations. The goal is to ensure the best patient health results and avoid readmissions (Shepperd et al., 2013). How often that goal is met would warrant further research. The differences found between the discharge dispositions of dementia and non-dementia patients may reflect a higher need for specialised care in the former.

Ambulatory problems in patients with dementia are a concern for caregivers and an important reason for putting them in a specialised healthcare facility like a residence or a nursing home (Dunn, Furner, & Miles, 1993). Dementia-related mobility impairment has a multifactorial aetiology that includes the neurodegenerative process itself, a previous history of falls and / or joint surgery, muscular weakness and/or atrophy, and pain of any origin (Stevens & Rudd, 2014; Verfaillie et al., 1997). In the late stages of dementia, behavioural problems like aggression can make any form of physical therapy difficult or even unadvisable (Rooney, 2014; Vincent & Dodds, 2012).

The PNH discharge services include Disability Support and Personal Health (MCDHB, 2009). Disability Support Services (DSS) are available for selected older people and include the following: assessment, treatment and rehabilitation services (AT&R), needs assessment and service co-ordination (NASC), home support services (personal care

and household management), carer support and respite care, environmental support (equipment, home, and vehicle modifications), residential care (rest home facilities, dementia unit or continuing care hospital). AT&R are specialist services dedicated to improving and maintaining the health of people so they can live independently (MOH, 2014).

AT&R services aim to reduce the number of people with complex health problems to stay longer in hospitals or long-term residential care. In most cases patients are elderly individuals who have experienced problems such as a stroke, heart failure, or Parkinson's disease (MOH, 2014). Community-based support services include home support, carer support and respite care. In 2007, the home support services using Client Claims Processing System (CCPS) data, noted that 12,577 people aged 65+ in the Central Region received help at their home for household management and personal care. Carer support is a subsidy funded by the MOH (2014) to assist unpaid, full-time carers of a disabled person to take a break from caring for that person.

Respite services provide a higher level of relief care than that offered by Carer Support, e.g., more days/short term breaks, are community based, and available to disabled people and to carers and family whose primary role involves the care and support of a disabled family member. They reduce the need for acute admissions or long-term residential care. In the Central Region DHBs carer support increases with age, with the rates of access highest in the 85+ group. About 600 people aged 65+ received carer support in 2007 in the Central Region Residential care. Residential care for older people includes accommodation/care for older people with the provision of meals and/or laundry a minimum requirement (this includes facilities providing medical or nursing

care for older people). The definition includes rest homes, continuing care hospitals, dementia units and rest home serviced apartments and excludes independent self-care flats or houses within a retirement village (MOH, 2014).

Additionally, the MCDHB Falls Action Group meets regularly, on what they have called April Falls Day, to develop, plan and implement protocols to reduce the risk of falls in the community (MCDHB, 2011). On admission, patients are assessed for their risk of falling. This strategy aims to assist healthcare providers to determine the level of support and type of assistance that each patient requires. The group is concerned with not just the incidence of falls in the region and in health services institutions, but with the short and long-term impact of falls on patients and their caregivers. The group aims to raise awareness about how people who have suffered a fall, especially the elderly, lose confidence in their ability to walk and often tend to stay longer in hospital. Group members are at stations set up around PNH, including the main foyer of the hospital, providing information for patients and staff about falls prevention. Representatives from ACC, Active Rehab, and St John may take part in the project.

In their effort to prevent falls in the PNH, the Falls Action Group has successfully been using a device called Invisibeam, invisible precision infrared beams that set off a warning system when a patient at risk of falling leaves their bed or chair. Additionally, the PNH in collaboration with ACC is distributing Vitamin D supplements to residential care facilities in the region. The rationale behind it are the known beneficial effects of the recommended daily dose of Vitamin D, 50-100nmol/L, on bone and muscle strength of institutionalised elderly people (Australian and NZ Bone and Mineral Society, 2005), with the hope that this strategy will help reduce the morbid-mortality of falls in the elderly community during their stay in a rest or nursing home (MCDHB, 2011).

Data from the 2006 census of population and dwellings for the Central Region suggests that approximately 6% of people aged 65+ lived in residential care in 2006 which is slightly lower than the proportion in 2001 (6.6%). Of these 7.5% were females and 4.1% males in 2006. The number of people in residential care increases substantially with age: 1.6% of people aged 65-74 were in residential care compared to 22.1% in the 75+ age group (NMDS, 2012). Whether people live in their own homes or in a residential facility, a diagnosis of dementia has an additional impediment for those who suffer from it.

In NZ, GPs have a legal obligation to advise the NZTA if they suspect that a person might have dementia – even if mild- and could, therefore, be unfit to drive (NZ Traffic Authority [NZTA], 2009). The assessment process is initially carried out by a nurse, social worker, or occupational therapist at home, and if results are inconclusive, the GP will refer the patient to the mental health services for more comprehensive assessment by a clinical psychologist or a psychiatrist (NZTA, 2009). Insurance companies in NZ require that any condition likely to affect a driver's ability must be disclosed, or the company has the right to turn down a claim. Recommendations from the American Academy of Neurology and the American Association for Geriatric Psychiatry agree with NZTA policies that patients, even with mild dementia, should be discouraged from driving (NZTA, 2009). Surrendering their driver’s license often has a negative psychological impact on individuals with early dementia and it is advisable that it be addressed as soon as possible (Alzheimer’s NZ, 2015).

8.3. Benefits of the Study

The results of this study support the hypothesis that there is an association between traumatic injury (regardless of location) severe enough to require acute admission in hospital and the development of dementia. These findings may induce other researchers to want to find out more about how the proposed pathophysiological mechanisms - a general and sustained alteration of homeostasis - are linked to the development of dementia. This knowledge could bring about preventative strategies that could reduce the incidence and / or impact of the condition in a context of an ageing population.

Another point of relevance of this research was the fact that it is a pioneer work in NZ, as - to the best of the Author's knowledge - there are no studies regarding the relationship between TI and dementia. Particularly relevant was that the topic had not been addressed in NZ, a country where trauma from traffic accidents, falls, assaults, and contact sports are high relative to the population (Statistics NZ, 2007, 2014). With regard to the psychological impact of trauma on dementia, the researcher aimed to advocate for the intervention of neuropsychologists in the palliative management of patients with dementia and of elderly patients suffering from the mental health consequences of traumatic injury: post-fall syndrome and lack of self-confidence.

A further positive aspect of the study was that common research threats to internal validity like insufficient power and selection bias were minimised by the comprehensive coverage of the NZ healthcare system which allowed for a large sample size to be available for the purpose of this study.

This work also aimed to bring to light the benefits for patients, their caregivers, and the society in general of having universal healthcare coverage. Hospital discharge types reflected those benefits in that the study sample patients requiring further or special assistance were referred to publicly funded specialised facilities.

Finally, the present multidisciplinary research was a cooperation between two key Central Region institutions: Massey University and the MidCentral DHB. Inter-institutional collaboration, which is promoted by both organisations, is beneficial for knowledge sharing and is particularly relevant for investigation regarding human health.

8.4. Limitations of the Study

This study had some limitations. The PNH admissions database has an administrative-fiscal and not a clinical purpose. Administrative databases have been claimed to miss about 15% of cases and to be inaccurate across clinical categories (Woodworth, Baird, Garces-Ambrossi, Tonascia, & Tamargo, 2009). This pitfall was countered in this study by the PNH's strict quality assurance protocols (McHugh, 2001), and strong data and coding validation criteria. Although the diagnostic protocols used by the Hospital are of the highest quality standards, clinical coders can only make restricted decisions in coding hospital admissions, and dementia cases could potentially have been missed or left undocumented as a coded diagnosis. Coders assign ICD-10 codes to a patient, but ICD codes fail to consider clinical data collection tools or interpretation of patient history, meaning that additional clinical information may get lost in translation. The limitation of failing to code a condition was compounded by the consistent underreporting of falls in the elderly population.

A further limitation was that the demographic and health data gathered by the DHB's and by Statistics NZ in their health publications are self-reported and not always up to date, meaning that there is a potential threat of incomplete or incorrect information being contained in those reports. Because the study was retrospective and non-experimental, makes it difficult to draw long-term conclusions like whether referring patients with dementia to rehabilitation services had any beneficial effects on their health outcomes. Finally, the results in this study may not be generalisable to other settings including other regions and other hospitals in NZ with a different demographic, geographic, and /or healthcare service structure.

The researcher was aware of the many unanswered questions regarding TI and dementia, like the existence of individual variability based on genetic make-up, lifetime health status, available care, and the use of substances and medications - especially if unreported. Although it was not possible to control for all the possible confounds, every effort was made to identify and control for the most relevant ones.

8.5. Conclusions

The findings in this work strongly suggest that the relationship between dementia and traumatic injury may not be unidirectional but a positive feedback loop. In other words, dementia increases the risk of having a traumatic injury (usually a fall) and traumatic injury may increase the risk of developing dementia. Whereas dementia has consistently been shown to be a risk factor for traumatic injury, the opposite remained to be considered, not to mention researched, by the scientific community. Although the literature reviewed indicates that the organic repercussions of traumatic injury could be a risk factor for dementia, further research is warranted to assess what type, location,

frequency, or degree of trauma would be required for dementia to ensue if at all. Further studies could also help to evaluate how a better understanding of the pathophysiology and the management of traumatic injury could affect the evolution of dementia.

The study showed that following a traumatic injury serious enough to require acute hospital admission less than 50% of patients with dementia who were previously living at home went back there at the time of discharge from hospital. Those patients – the cases in this study – were additionally much less likely than the controls to be discharged to rehabilitation services.

This research also noted that the prevalence of comorbidities and the associated multimedication worsen the prognosis of trauma patients with dementia. This is especially relevant in the case of antithrombotic medication for cardiovascular disorders due to the increased risk they pose to the development and management of intracranial haemorrhages in falls-prone individuals. The impact of polypharmacy on falls risk in patients with dementia warrants further investigation for risk-benefit evaluation.

To this day, there is no curative medical treatment for most dementias. Psychological, preventative, and safety approaches are the current available options to ensure the best possible health outcomes in patients with dementia, particularly among those who have a history of major traumatic injury. A causative relationship is unlikely to be proven between traumatic injury and dementia so that the latter, if suspected to be the result of trauma, may be covered by ACC. That said, and although the combined diagnoses and consequences of dementia and traumatic injury have a bleak prognosis, the fact that the NZ healthcare system has comprehensive healthcare and support services for patients and caregivers is reassuring. It is a privilege worth preserving.

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