The Impact of Chronotype on Obesity-Related Outcomes: Diet, Behaviour and Metabolic Health

A thesis presented in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Nutritional Sciences at Massey University, Auckland, New Zealand

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List of abbreviations

AG	Android to Gynoid fat percentage ratio
BF%	Body fat percentage
BMI	Body mass index
EAT-26	Eating Attitudes Test-26
ET	Evening type
FM: FFM	Fat mass to fat free mass
HC	Hip circumference
HDL-cholesterol	High-density lipoprotein cholesterol
HOMA-IR	Homeostatic model assessment – insulin resistance
IT	Intermediate Type
JBI	Joanna Briggs Institute
MCTQ	The Munich Chronotype Questionnaire
MSF	Mid-sleep on free days
MSFsc	Mid-sleep midsleep corrected for sleep duration on free days
MSW	Midsleep on weekdays
MT	Morning type
NC	Neck circumference
NZ	New Zealand
PA	Physical activity
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO	International Prospective Register of Systematic Reviews
RCT	Randomised controlled trail
SCN	Suprachiasmatic Nuclei
TFEQ	Three Factor Eating Questionnaire
WC	Waist Circumference

Abstract

Background: Describing the effect of the diet on obesity risk as simply excessive energy intake, does not factor in the *patterning* (spacing, skipping and timing), or *format* (food combinations and nutrient content) of meals consumed or the specific *eating behaviours* that contribute to weight gain. In addition to this, chronotype, may also play a role in the complex aetiology of obesity. Being a very late (=evening type, ET) or early chronotype (=morning type, MT) not only determine preferred sleep- and wake-times but may also influence mealtimes, nutritional composition of meals and eating behaviour. This may impact the circadian timing system and in the long-term, result in weight gain, obesity, and poor metabolic function.

Aims: To explore the diet in-depth (eating patterns and eating behaviours) as it relates to different chronotypes and metabolic health markers of New Zealand (NZ) European and Pacific women with different body fat profiles and varied metabolic disease risk factors.

Methods: This research formed part of the PRedictors linking Obesity and gut Microbiome (PROMIsE) cross-sectional study that was conducted at the Massey University in Auckland, NZ. Healthy women, between the ages of 18 and 45 years were recruited based on healthy BMI (18.5 -24.9 kg/m^2) and obese BMI (≥30 kg/m^2) within NZ European (n = 162) and Pacific (n = 142) ethnic groups. Chronotype, was assessed using the Munich Chronotype Questionnaire. Fasting venous blood samples were collected to assess metabolic biomarkers (hormones, lipid profile and glucose homeostasis). Anthropometrical measurements included, body mass index (BMI), and whole-body total fat percentage (BF%), android- and gynoid- fat mass were assessed using dual-energy x-ray absorptiometry. Five-day food records were used to assess dietary intake. The Three Factor Eating Questionnaire and the Eating Attitude Test -26 was used to assess eating behaviour and - attitudes.

Results: Half of participants were intermediate type (IT; n = 155, 54%) followed by ET (n = 97, 34%) and MT (n = 35, 12%), with most Pacific women being ET (n = 83, 86%) and most NZ Europeans being IT (n = 115, 65%). Due to low sample size, the MT and IT were combined as MT-IT, for some analyses. The MT-IT women had lower BMI, BF% and android to gynoid fat percentage (AG) ratio, lower concentrations of triglycerides, insulin, leptin, LDL-cholesterol, HbA1c and higher HDL-cholesterol and ghrelin in comparison with ET.

Total daily energy and macronutrient intakes were similar across the chronotype groups. Women classified as MT-IT vs ET had higher intakes of energy, protein, carbohydrate, and fat in the morning

(by 10:00). Conversely at night (after 20:00) the ET had a higher energy, protein, carbohydrate, and fat intake.

The ET in the high BF% as well as high AG ratio group predicted lower energy, protein and carbohydrate intakes in the morning, and predicted higher energy, carbohydrate and fat intake at night compared with MT-IT.

Women with an earlier chronotype (MT-IT) followed dietary patterns that consisted of high micronutrients, protein, fat and fibre, namely, the *Healthy* food pattern, *Animal Products* food pattern and the *High Protein-Fat-Fibre* nutrient pattern. The ET women followed the *High Carbohydrate* nutrient pattern high in poor quality carbohydrates.

Eating behaviour was associated with chronotype. The ET had higher scores for unfavourable eating behaviours such as lower *restraint* scores (conscious restriction of food intake to control body weight), while having higher *hunger* scores in comparison with the MT-IT. In the high BMI group, ET predicted lower *restraint*, *rigid control*, but higher perceived *hunger*, *internal locus for hunger*, *habitual disinhibition* (loss of control of food intake) and *bulimia & food preoccupation*, compared with MT-IT.

Conclusion

This PhD thesis found that ET when compared to MT were more likely to have a higher body composition and an unhealthy metabolic biomarker profile. This chronotype-body composition relationship may be linked to the ET's irregular eating patterns and unhealthy eating behaviours that may contribute to the circadian misalignment. Although the chronotype groups had similar nutrient intakes, clear differences were noticeable regarding the types and combinations of foods consumed together as well as the distribution of food intake throughout the 24-hour day. The ET distributed their nutrient intake towards the night, the circadian phase of the day that is metabolically more suited to fasting in comparison with the MT-IT that had a higher nutrient intake during the morning, the feeding phase of the day. This PhD thesis showed that consuming a higher amount of nutrients in the morning and during the early part of the day, seemed to be protective against developing obesity.

These findings give further insight into the differences in eating patterns and eating behaviours between non-obese and obese individuals, placing an individual's chronotype at the center.

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1 Chapter 1: Introduction

1.1 Background to the problem

Obesity is a multifactorial and complex disease with an ever-increasing prevalence. Obesity is characterised by lipid accumulation in the adipose tissues over time, as well as low-grade systemic inflammation, all of which result in a deterioration of glucose and lipid metabolism (Heymsfield & Wadden 2017). Globally, 60% of adults are overweight of which 25% are obese (OECD 2019). This is a major health concern as it increases the risk for non-communicable diseases (NCD) such as, type two diabetes mellitus, hypertension, fatty liver disease, cardiovascular diseases, and certain types of cancers. Obesity and accompanying NCD, decreases both the quality and expectancy of life (Blüher 2019).

The aetiology of obesity is not fully understood despite decades of research dedicated to the investigation of the underlying mechanisms and the development of successful interventions and treatments (Schwartz et al. 2017). The aetiology can be simplistically described as the result of sustained positive energy balance or in other words, prolonged excessive energy intake that does not match with energy expenditure. However, intervention strategies targeting the reduction of excessive energy intake and increasing physical activity had largely been unsuccessful in promoting long-term weight reduction (Taubes et al. 2013). This unsuccessful long-term weight loss in obese individuals may be explained by our evolutionary physiology, where the human body is inclined to preserve body fat to ensure survival (Schwartz et al. 2017). The mechanisms involved in the prevention of weight loss and the promotion of further weight gain are complex and involved hormonal, metabolic and neurochemical adaptations (Blüher 2019). Additionally, describing the effect of the diet on obesity risk as simply excessive energy intake, does not factor in the contribution of macronutrient intakes or the contribution of eating behaviours. It is suggested that eating behaviours affect energy intake (Provencher et al. 2003) by influencing the types and amounts of foods eaten, timing of food intake and where food intake occurs. Eating behaviours encompass a range of concepts relating to dieting, food choices and motives, feeding practises and eating-related problems such as eating disorders, disturbed eating attitudes and obesity (LaCaille 2013). The distribution of macronutrient intakes across the day is also suggested to influence energy balance by altering the total energy intake and expenditure throughout the day (Schwartz et al. 2017). Furthermore, individuals don't eat singular nutrients, they consume a variety of foods typically grouped together at different eating occasions (as meals or as snacks) across the 24-h day which represents an eating pattern (Kant 2018). The term eating pattern is used to describe overall dietary intake in terms of *patterning* (meal spacing, skipping and timing) and *format* (food combinations and nutrient content) (Leech et al. 2015). Therefore, the nutritional composition of meals in terms of energy density, macro- and micronutrients and at different time points are important variables to consider when exploring the impact of the diet on energy balance and weight status. Also, differences in eating patterns

are related to different macro- and micronutrient intakes as well as diet quality (Leech et al. 2015). Further, using a food-based approach to assess dietary diversity and eating patterns is often better able to capture the subtle interactions of nutrients or dietary compounds with that of disease outcomes or risk factors (Tucker 2010).

Obesity aetiology is complex with a large range of biological (e.g., genetics), environmental (e.g., food environment) and socio-economic (e.g., poverty) factors with interplaying relationships (Blüher 2019). One of these biological factors involved in the complexity of obesity aetiology are circadian rhythms of internal biological clocks in the brain and body (Sato-Mito et al 2011, Bass 2012, Kanerva et al. 2012, Maukonen et al. 2016, Jiang & Turek 2017, Manoogian & Panda 2017, Maukonen et al. 2017, Muñoz et al. 2017, Blüher 2019, Maukonen et al. 2019). Research studies have shown the important role of the biological circadian rhythms, demonstrating the obesity-promoting effects of dietary intake during the dark cycle of the circadian phase (Garaulet et al. 2013, Ruiz-Lozano et al. 2016). Most organisms, including humans have developed an internal time keeping system. That is, biological clocks that generate circadian (approximate 24-hour) rhythms of metabolism, behaviours and gene expression that needs to be synchronised with the solar light-dark cycle of the earth's rotation (Ko & Takahashi 2006, Bass 2010, Garaulet & Madrid 2010, Huang et al. 2011). Humans are physiologically suited to spend about two thirds of their 24-hour day awake, being active and eating and storing energy. They usually spend one third asleep, being in a fasting state at nighttime (Buxton et al. 1997). During the day, ingested food provides energy to support metabolic processes and during the night, stored energy is mobilised to maintain homeostasis (Damiola et al. 2000, Cipolla-Neto et al. 2014). Since the internal circadian clocks produces a rhythm with a period length of approximately 24-hours, an external zeitgeber ('time cue') is required to entrain the circadian clocks with the external environment (Aschoff 1965, Moore 1973, Roenneberg et al. 2003), thereby ensuring that physiology and behaviour is in sync with the solar day (Ko & Takahashi 2006). This ability to synchronise circadian rhythms by means of external zeitgebers is vital for survival and important in the feeding-fasting cycle, ensuring that food is ingested, and metabolised at the expected times. Without this ability, the circadian rhythms will gradually become desynchronised from the environment (Reppert & Weaver 2002). Environmental light is the primary zeitgeber for the central circadian clock, however other external cues such as food intake, including the timing and composition of the food intake is also capable of setting the rhythms in the peripheral clocks as well as the clock-controlled genes in the body tissues and organs (Bass 2012, Youngstedt et al. 2016, Jiang & Turek 2017). These clock genes in turn influence the timing of digestion, nutrient uptake and metabolism, metabolite and hormonal regulation, food intake, behaviour and appetite (Bass 2012, Jiang & Turek 2017). Both timing and composition of food intake (particularly macronutrients) are therefore further important zeitgebers for the circadian timing system (Manoogian & Panda 2017). Energy requirements and the oxidation of macronutrients vary across the 24-hour light-dark cycle (Rácz et al. 2018), where insulin sensitivity and glucose tolerance are optimal during

daytime, coupled with increased energy expenditure, lipogenesis and adiponectin synthesis, making it the ideal phase of the 24-hour day for acquiring and storing energy through dietary intake (Cipolla-Neto et al. 2014). Conversely, upregulation of gluconeogenesis, glycogenolysis and lipolysis is prominent at night (Cipolla-Neto et al. 2014), with resulting higher levels of circulating fatty acids, triglycerides and cholesterol concentrations (Takahashi et al. 1968, van Moorsel et al. 2016), making this the ideal phase for fasting. Consequently, the timing of energy intake has different effects on energy utilisation and as a result affect weight loss effectiveness, body composition and metabolic health outcomes (Garaulet et al. 2013, Jakubowicz et al. 2017).

However, in today's 24/7 society, the internal circadian body clocks are challenged by various factors such as work obligations, social demands and chronotype, all of which alter the normal behavioural patterns, where feeding and activity is no longer restricted to daylight hours and fasting and sleep is no longer prioritised to night hours, as for example night shift workers (Roenneberg et al. 2012).

The term chronotype is used to describe an individual's sleep-wake timing (Ehret 1974, Roenneberg et al. 2013) that influences the timing of their diurnal preferences for eating and the modulation of physiological and cognitive functions (Horne & Östberg 1976, Roenneberg et al. 2003, Roenneberg et al. 2007, Wirz-Justice 2007). Chronotype can be subjectively assessed by using validated questionnaires, which can allow for example calculation of mid-sleep time based on weighted sleep timing on work and free days. The Munich Chronotype Questionnaire (MCTQ) is used to assess an individual's chronotype (Roenneberg et al. 2007). Using these questionnaires, humans can be classified as either early chronotypes (= morning types; MT), intermediate (IT) or late chronotypes (=evening types; ET) (Adan et al 2012). The MT prefer an early bedtime and early morning rise time, as they perform daily physical and cognitive activities preferably during the early part of the day. On the other hand, ET, prefer a later bedtime and a later morning rise time, as they perform daily physical and cognitive activities optimally later in the day (Adan et al 2012). The majority of individual chronotypes lie in a range between extreme MT and ET (Covassin et al. 2016). The extreme chronotypes are more likely to experience misalignment of their internal clocks as a result of the mismatch between their preferred circadian timing and social demands such as having to rise early for work or work late at night (e.g., night shift work). This mismatch has been termed 'social jetlag' (Wittmann et al. 2006). The ET tend to display unhealthier dietary behaviours such as delayed meal timing and breakfast skipping, than MT (Baron et al. 2011, Sato-Mito et al 2011, Kanerva et al. 2012, Reutrakul et al. 2014, Muñoz et al. 2017). The delay in food intake in ET is likely be due to their later wake-up time, which leads to redistribution of the energy and macronutrient intake of the skipped meals to later in the day, thereby still consuming the same amount of food (Meule et al. 2012, Dashti et al. 2015). As a result, the food is consumed at a presumably suboptimal physiological time of the day in relation to the metabolic rhythms (Qin 2003). Suboptimal timing of meals results in a mismatch of the internal clock rhythms to the

external environment, which ultimately contributes to the risk of developing obesity as well as adverse metabolic health outcomes (Garaulet et al. 2013, Jakubowicz et al. 2017, Collins et al. 2018). Therefore, properly timed meals in alignment with the circadian timing of other biological clocks are important to ensure energy balance, metabolism, and health (Garaulet et al. 2013, Jakubowicz et al. 2017, Collado et al. 2018, Collins et al. 2018). The timing and composition of food intake relative to daily activities serve as a powerful zeitgeber (Salgado-Delgado et al. 2010 , Nelson & Chbeir 2018, Challet 2019). Chronotype therefore drives not only preferences for sleep and wake times, but also the timing of food intake (fasting or eating), which may explain the higher obesity prevalence in ET.

Describing the obesity aetiology solely as excessive energy intake mismatched with energy expenditure, may be too simplistic. Of the vast range of biological, environmental, and socio-economic factors causing obesity, the timing of food intake, energy density and composition of meals and eating behaviour may all influence each other and further be modulated by the circadian regulation of metabolic functions. Thus, interactions between the timing of food intake and the composition of food / meals, are all potentially involved as obesity risk factors (schematically shown in **Figure 1.1**). This may further contribute to explaining the complex aetiology of obesity and other metabolic diseases and may be a potential intervention target area for treating and preventing obesity. In turn, these factors may also exert effects on other internal clocks.

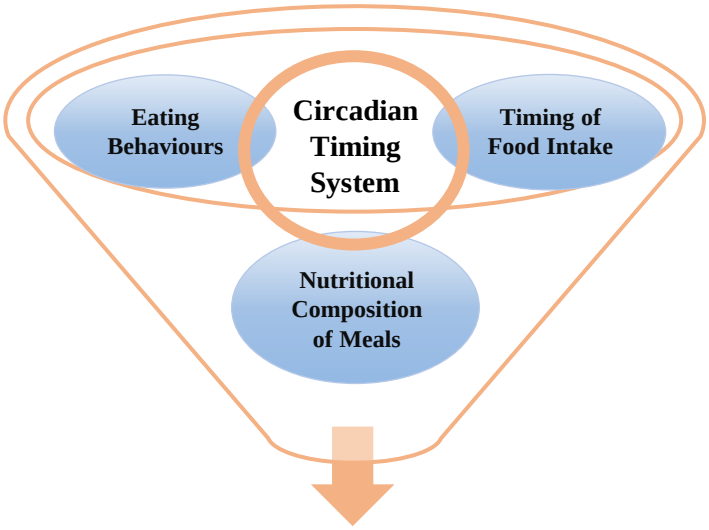

Figure 1.1 *- Possible interplaying factors in obesity & metabolic disease outcome*

1.2 Problem statement & research questions

This PhD research forms part of the PRedictors linking Obesity and the MIcrobiomE (PROMIsE) study (funded by the Health Research Council New Zealand (NZ)) which was conducted as a large cross-sectional prospective clinical study, led by Prof. B. Breier, College of Health, Massey University and

data collection was performed between July 2016 - September 2017. The PROMIsE study aimed to (1) categorise the gut microbiome in the NZ Europeans and Pacific population groups, and (2) explore roles for taste perception, diet, sleep and physical activity in modifying the gut microbiome and its impact on obesity (Kindleysides et al. 2019). The study included healthy NZ European and Pacific women aged between 18 and 45 years from the Auckland region, specifically recruited as to have different body composition profiles in each ethnicity (normal and obese profiles) (Kindleysides et al. 2019). The two ethnic groups were chosen to enable the study to compare the ethnic group with the lowest prevalence of obesity (NZ Europeans) with the Pacific women with the highest obesity prevalence in NZ. The PROMIsE study is of special interest for the reasons that the overweight and obesity prevalence in NZ is increasing with an accompanying rise in chronic lifestyle disease prevalence, which places a huge strain on the health care system. Overweight and obesity is strongly related to ethnicity and socioeconomic inequalities (Ministry of Health 2019). Younger women of childbearing-age (15 – 49 years) are especially vulnerable to weight gain (Adamson et al. 2007, Gordon-Larsen et al. 2010, Dutton et al. 2016) because of greater food insecurity (Nord et al. 2009), sole parenting (McPherson 2006), and work/family schedules (Olson 2005) to name a few contributing factors. Additionally, if these women also become overweight or obese, their long-term health outcomes are more adverse than healthy weight women (Marchi et al. 2015, Ministry of Health 2019). Therefore, implementing behavioural interventions to prevent weight gain or correct overweight and obesity in this vulnerable group, have the potential to sustainably improve health and well-being of women in addition to the future generation in New Zealand (Hutchesson et al. 2013). Currently, recommendations for achieving and maintaining a healthy body weight as well as optimal metabolic health outcomes are based on the statement that the fundamental cause of obesity is excessive energy intake that is not balanced with energy expenditure. Although existing dietary and lifestyle recommendations aimed at reducing energy intake and increasing physical activity are beneficial, thus far, they have been largely unsuccessful in achieving long-term weight loss (Blüher et al. 2019, Ministry of Health 2022). Hence, understanding the underlying and possible overlapping mechanisms that contribute to obesity and the related comorbidities in this group of NZ European and Pacific women are key, before tailored interventions can be designed or initiated.

Preliminary data analysis from the PROMIsE study data set (unpublished food record analysis) has identified that there is no statistically significant difference in the total daily energy, protein, and fat intakes ($P > 0.05$) between lean and overweight/obese women of both ethnic groups (Renall 2020). This is consistent with research from other studies in both humans and animals, which found that the timing of energy and macronutrient intake, rather than the total intakes is a crucial factor with regards to weight gain and adverse metabolic health outcomes (Arble et al. 2009, Garaulet et al. 2013, Garaulet et al. 2014, Ruiz-Lozano et al. 2016).

The main research questions that arise from these problems are:

(1) Is chronotype associated with body composition?

(2) Are there differences in eating patterns (nutritional composition and timing of meals) between different chronotypes?

(3) Are chronotype-specific eating patterns associated with body composition?

(4) Is chronotype associated with metabolic health biomarkers?

(5) Are there differences in eating behaviours between different chronotypes?

(6) Are chronotype specific eating behaviours associated with body composition?

1.3 Research aim

The aim of this PhD research is therefore to explore the diet in-depth (eating patterns and eating behaviours) as it relates to different chronotypes and metabolic health markers of NZ Europeans and Pacific women with different body fat profiles (lean and obese) and varied metabolic disease risk factors. **Figure 1.2** illustrates the three identified research areas as well as the related variables that will be explored.

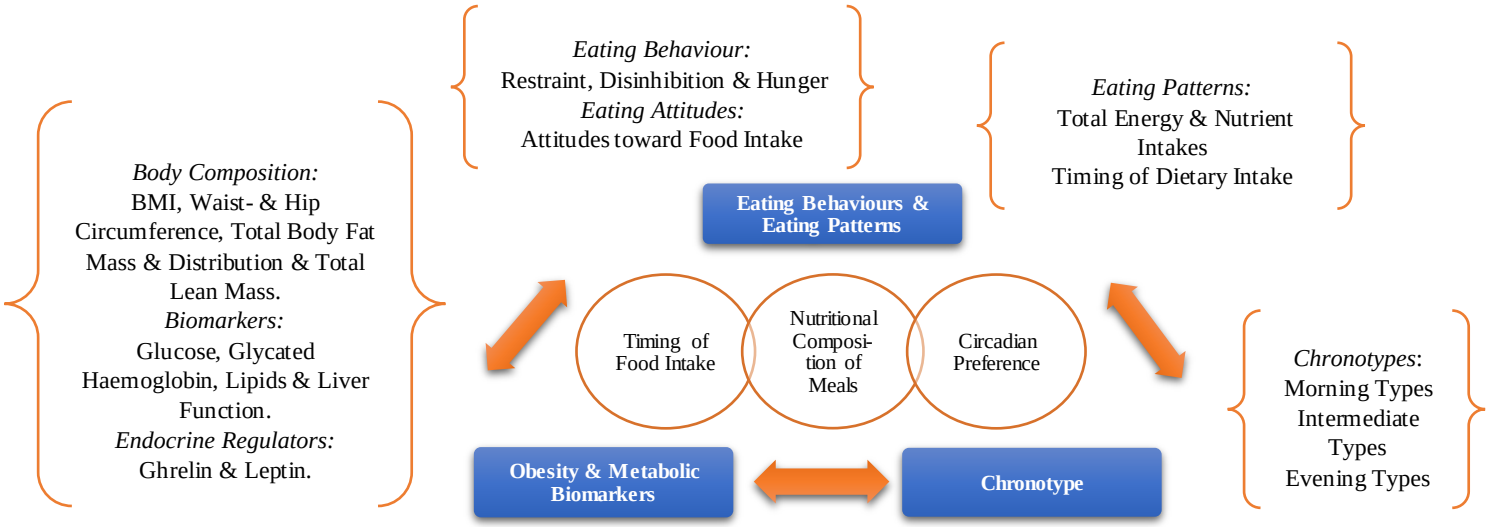

Figure 1.2 *- Schematic overview of the key research variables*

1.4 Research objectives

(1) Systematic Review: To conduct a systematic review of the literature (i) to assess published

evidence whether chronotype is associated with body composition, and (ii) if this has an effect on dietary intake, eating behaviours and metabolic biomarkers in healthy adults. This systematic review provided background knowledge, identified the research gaps and guided the statistical analysis of the PROMIsE study data.

Based on the data of the PROMISE study:

(2) To investigate if chronotype is associated with body composition in healthy young women (NZ European, Pacific women).

(3) To describe the diet of these women of in terms of eating patterns (nutritional composition and timing of meals) and to investigate whether chronotype and body composition are associated with the eating patterns.

(4) To investigate whether chronotype is associated with metabolic biomarkers.

(5) To identify dietary patterns (food and nutrient patterns) and investigate if the patterns are associated with chronotype and body composition.

(6) To investigate the associations between chronotype, eating behaviours (restraint, hunger, and disinhibition), eating attitudes (dieting, oral control, and bulimia & food preoccupation), and body composition.

1.5 Research outcomes and contributions

With this PhD research we focused on diet in terms of patterning (spacing, skipping and timing of meals), the format of the meal (food combinations and nutrient content), and the eating behaviours, within different chronotypes and associations with body composition measures (body mass index, body fat percentage, android and gynoid fat distribution), appetite hormones (leptin and ghrelin) and metabolic biomarkers (plasma glucose, insulin, glycated haemoglobin, and lipid profile).

The first step was a systematic review, and the second step was a comprehensive data analysis by using the available PROMIsE data set.

This research project had the potential to identify chronotype as an important factor to consider when recommending tailored dietary changes for overweight and obese individuals.

1.6 Structure of thesis & research activities

Figure 1.3 gives an overview of the completed research activities of the PhD research.

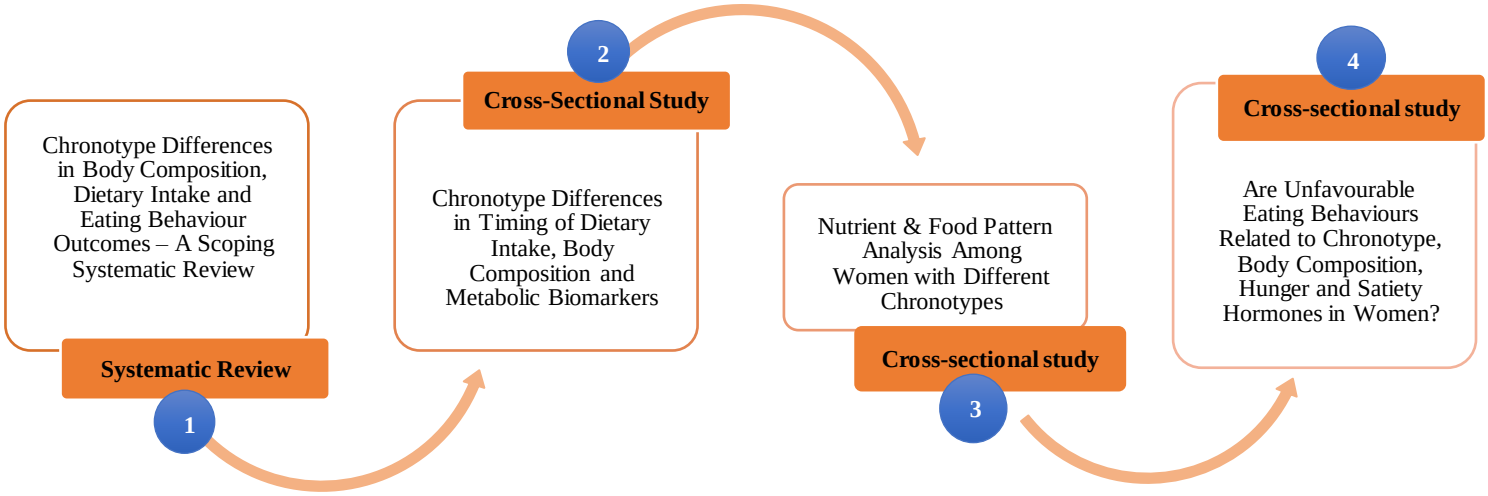

***Figure 1.3** - Overview of Planned Research Activities*

1.7 Research Team

Role of Team Member	Name
Main supervisor: Expert in dietary assessment strategies and was co-investigator of the PROMIsE study. The study leader gave guidance on the planning of the dissertation, participated in the systematic review, gave support with the dietary, eating behaviour, body composition and metabolic health data analysis, gave support with the statistical analysis, interpretation and writing up of all the data, and reviewing the dissertation.	Prof R. Kruger, Massey University
Co-supervisor: Expert in chronobiology and sleep physiology. The co-supervisor supported with the planning of the dissertation, and supported with the systematic review, the chronotype data analysis, statistical analysis, interpretation and writing up of the data, and reviewing the dissertation.	A/Prof Miriam Münch, Massey University (now: University of Basel, Switzerland
Co-supervisor: Expertise in dietary intake assessment and metabolic health. The co-supervisor supported with the statistical	Dr Marilize Richter

Role of Team Member	Name
analysis, interpretation and writing up of the results, and reviewing the dissertation.	
Co-supervisor: Expert in metabolic health and principal investigator of the PROMIsE study. The co-supervisor supported in an advisory role with the planning of the dissertation.	Prof B. Breier
Advisor: Expertise in statistics and advised on the statistical analysis plan and supported with the statistical analysis	Dr Marine Corbin
Advisor: Expertise in statistics and advised on the statistical analysis plan and supported with the statistical analysis	Prof Jeroen Douwes
MSc Dietetics student: The student assisted with dietary data processing.	Amy Richter, Dietetics student, Massey University

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2 Chapter 2: Literature review

Obesity is a global problem of epidemic proportions with 60% of adults who are overweight and 25% who are obese (OECD 2019). Obesity is characterised as the abnormal and excessive accumulation of white adiposity tissue over a period of time (World Health Organization 2021). This accumulation of adipose tissue results in low-grade systemic inflammation and deterioration of glucose and lipid metabolism (Heymsfield & Wadden 2017). Obesity is well known to be related to a number of non-communicable diseases (NCDs) including cardiovascular and metabolic diseases as well as certain types of cancers. This is of great concern as obesity and accompanying NCDs, decreases both the quality and expectancy of life (Blüher 2019). Energy homeostasis is the foundation of the body weight regulation process. Energy homeostasis is described as the balance between energy intake (from food and drinks) and energy expenditure (energy utilised to maintain essential body functions and physical activity) (Park 2019). When energy intake exceeds energy expenditure, the unused energy is stored as adipose tissue. On the other hand, when expenditure exceeds energy intake, the stored energy is metabolised to meet the energy needs (Park 2019). Excess body fat can disrupt the balance of energy homeostasis. Various factors can be drivers of an unbalanced energy homeostasis and are specific to each individual (**Figure 2.1**). These factors include environmental factors such as the food environment, shift work schedules, stress, social constraints and eating culture. Biological factors are also involved and may include factors such as sleep, circadian biology and epigenetics (Blüher 2019). Unhealthy dietary patterns have been suggested as the main driver of overweight and obesity (San-Cristobal et al. 2015). For example, the "Unhealthy" or "Western dietary pattern" characterised by the overconsumption of energy dense foods, low in micronutrients, which is associated with a higher body composition and an elevated risk for chronic lifestyle diseases (Newby et al. 2003, Murtaugh et al. 2007, Paradis et al. 2009). In comparison, healthy dietary patterns like the "Prudent dietary pattern" high in whole grains, fruits, vegetables, legumes, and fish, has been linked with more healthy body composition and consequently a lower risk for disease development (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015) Dietary intake in combination with the various other environmental and biological factors form part of the multifactorial aetiology of obesity (Blüher 2019, Park 2019).

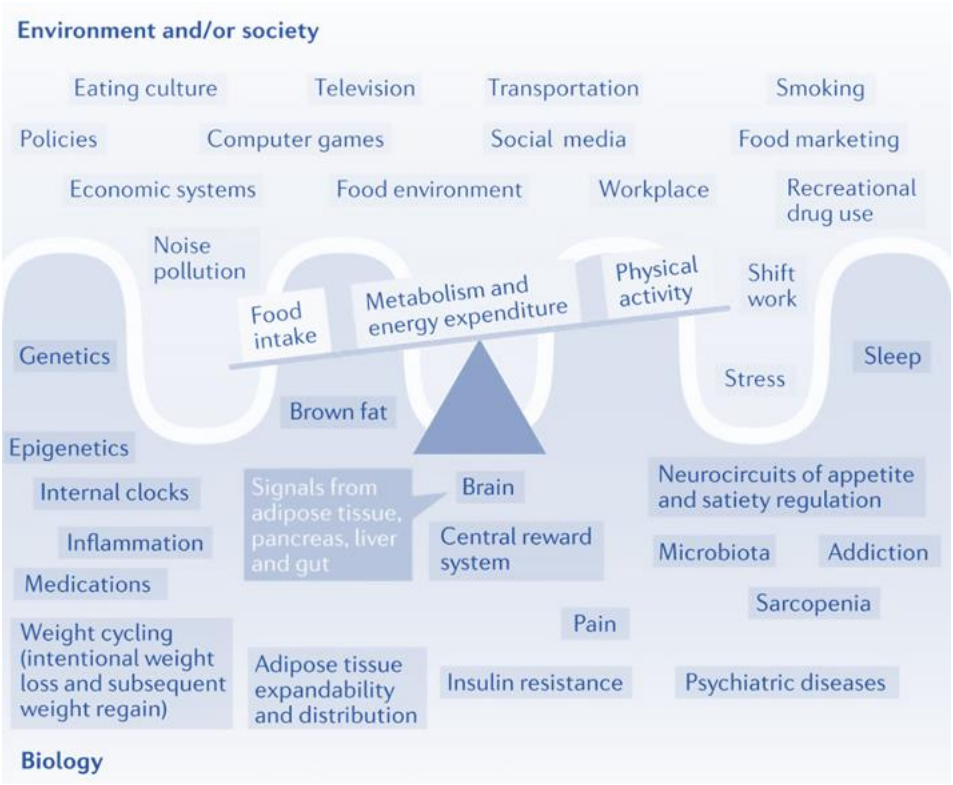


Figure 2.1 *- Summarised biological, environmental, and social factors involved in the aetiology of obesity (Blüher 2019). Reproduced with permission.*

Lifestyle changes such as increasing physical activity (PA) and adopting a healthy eating pattern that is lower in calories is key to maintaining good health and a normal body weight (Bray et al. 2018). However, these interventions have largely been unsuccessful in promoting long-term weight reduction in overweight and obese persons. Therefore, research focus has shifted towards understanding the underlying mechanisms of obesity development (Taubes et al. 2013). One of these emerging research fields is circadian biology. There is increasing evidence that circadian biology is an important factor that may shed additional light on the drivers of obesity-related factors. One of the circadian factors is the timing of food intake. Food intake at the incorrect time of the day has been linked with obesity and poor metabolic health outcomes (Garaulet et al. 2013, Ruiz-Lozano et al. 2016).

2.1 Obesity context in the New Zealand Pacific & New Zealand European population

Globally the prevalence of obesity has tripled since 1975 (World Health Organization 2021). A high obesity prevalence is also seen in the New Zealand (NZ) population and is a major public health concern. NZ has the fourth highest obesity rate amongst the Organisation for Economic Co-operation and Development (OECD) countries and rates are continuously increasing (Ministry of Health 2020). Results from the 2020/2021 NZ Health Survey have shown that 66.4% of the population is currently overweight or obese, and 31.2 % obese. This is equivalent to one in every three adults that are affected

(Ministry of Health 2020). There is a strong relationship between obesity, ethnicity, and socioeconomic inequalities. Māori and Pacific peoples are more likely to be overweight or obese in comparison with the general NZ population. Currently, 63.4% of Pacific peoples are affected in contrast with 47.9% Māori, 29.3% European/Other and 15.9% of Asian adults (Ministry of Health 2020). Socio-economic background is also a factor, that is, adults living in the most socio-economically deprived areas are more likely to be overweight or obese than those not living in deprived areas (Ministry of Health 2019). Food insecurity, defined as "the limited or uncertain availability of nutritionally adequate and safe foods or limited or uncertain ability to acquire acceptable foods in socially acceptable ways" (Kendall & Kennedy 1998), is linked to individuals with limited financial resources (Booth & Smith 2001). Data from the 2008/2009 National Nutrition Survey showed that 7.3% of NZ households were classified as having low food security and was linked with higher rates of neighbourhood deprivation (University of Otago and Ministry of Health 2011). Similarly, Carter and colleagues (2010), analysed data from the longitudinal Survey of Families, Income and Employment (SoFIE) and found that 15% of the NZ population were food insecure in 2004/2005, with the strongest predictors being younger age groups, sole parenthood, unmarried status, Pacific peoples, and lower socioeconomic status (Carter et al. 2010). In comparison, the prevalence of food insecurity was higher in females (19%) than men (12%) (Carter et al. 2010). Similarly, other researchers have demonstrated the link between women and food insecurity (Parnell et al. 2001, Townsend et al. 2001). Plausible explanations for the higher prevalence of food insecurity are based on the social role of women, which involves caring for- and feeding their families (Olson 2005). Women in food insecure households may prioritise their children or husbands when it comes to food intake (Nord et al. 2009). Another explanation may be that women are more likely to be single parents, with 80% of single parents in NZ being women (McPherson 2006).

Overweight and obesity in women of a childbearing age (15 – 49 years) is especially a concern, with prevalence rising globally (Müller & Geisler 2017). Women in this life stage are especially vulnerable to weight gain. This is supported by several cohort studies demonstrating that this age range is associated with the greatest weight gain (Adamson et al. 2007, Gordon-Larsen et al. 2010, Dutton et al. 2016). In NZ, 32% of women are obese and 30% overweight (Ministry of Health 2019). Obesity during child-bearing years is linked with several long-term adverse health outcomes, including diabetes, hypertension, cancer, cardiovascular disease and overall mortality (Zheng et al. 2017). Maternal obesity is associated with ongoing adverse health outcomes for both the mother and baby. Obesity during pregnancy poses a greater risk for foetal defects, preterm birth, and congenital abnormalities, furthermore it is a significant factor for obesity in offspring and may result in long-term obesity for the mother (Mamun et al. 2011, Marchi et al. 2015).

2.2 Assessment of excessive adiposity

Body mass index (BMI) is often used to measure obesity. BMI is determined by dividing the body

weight in kilograms by the square of the height in meters (kg/m^2). It is an inexpensive and effective weight screening tool which gives an indication of relative body size (Lee 2007). BMI is best used in population studies and increased BMI correlates well with metabolic and fat mass diseases (De Lorenzo et al. 2016). A BMI of ≥25 kg/m^2 is classified as overweight and a BMI of ≥30 kg/m^2 is classified as obesity (Fitch & Bays 2022). However, BMI should not be used as the sole indicator of adiposity as it does not always reliably reflect body composition, metabolic disease and fat mass disease risk on an individual level. For example, BMI cannot distinguish between fat mass and fat free mass. This measurement may overestimate adiposity in muscular individuals or underestimate adiposity in sarcopenia patients (De Lorenzo et al. 2016). It is therefore suggested that in conjunction with BMI, other measurements such lean body mass (kg), whole body total fat mass (kg) (De Lorenzo et al. 2016) and percentage, android-, gynoid- and visceral fat mass be measured by dual-energy x-ray absorptiometry (DXA) as well as inexpensive methods such as waist- and hip circumference measurements (Krotkiewski et al. 1983, Sun et al. 2010, Hangartner et al. 2013, Chen et al. 2019, Gamboa-Gómez et al. 2019, Low et al. 2020, Seo et al. 2020). Collectively these measurements can give a comprehensive body composition profile and disease risk. It has been suggested that the distribution of body fat mass is more important in understanding the relationship between obesity and metabolic disease risks than considering only overall fat mass (Després and Lemieux 2006, Fox et al. 2007). For example, central obesity (measured by waist circumference and DXA) is a highly sensitive predictor of diabetes, insulin resistance and cardiovascular disease risk (Krotkiewski et al. 1983, Blouin et al. 2008, Sun et al. 2010) independent of BMI, while fat mass to fat-free mass (FM: FFM) is associated with insulin resistance, glucose homeostasis disorders, liver fat accumulation and metabolic syndrome (Chen et al. 2019, Gamboa-Gómez et al. 2019, Low et al. 2020, Seo et al. 2020).

2.3 Obesity aetiology

Obesity is defined by the Obesity Medicine Association as, *"... a chronic, progressive, relapsing, and treatable multi-factorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences*" (Bray et al. 2017).

The accumulation of excessive body fat seen in obesity develops slowly overtime and the excessive lipids are distributed to various body compartments such as the adipose tissues, skeletal muscle, and the liver. The metabolic profile of an obese individual differs from those with a healthy body weight. In individuals with a higher body fat percentage the following characteristics is often seen: a larger adipose and lean body mass; an increase in resting energy expenditure; higher cardiac output; elevated blood pressure and greater pancreatic beta cell mass. The Obesity Medicine Association define healthy metabolic metrics for individuals with a healthy body weight as follows (Puhl et al. 2013, Fitch & Bays 2022):

- Waist circumference (WC) <102 cm for males and <88 cm for females (excluding Asian individuals)
- Systolic blood pressure <120 mmHg
- Diastolic blood pressure < 80 mmHg
- Fasting glucose <5.56 mmol/L
- Glycated Hemoglobin (HbA1c) < 5.7%
- Triglycerides <8.33 mmol/L
- High-density lipoprotein cholesterol ≥2.22 for males and ≥2.78 mmol/L for females

Insulin is produced by the pancreatic β-cells and plays a crucial role in peripheral glucose homeostasis by regulating glucose uptake, glycogen storage as well as gluconeogenesis. In obese individuals, the deterioration of glucose and lipid metabolism contributes to the body cells becoming increasingly resistant to insulin. The pancreas in turn, overcompensates by increasing insulin secretion, resulting in elevated levels of circulating insulin and ultimately insulin resistance (Heymsfield & Wadden 2017). With the decline in insulin sensitivity, other metabolic alterations including oxidative damage (Hayes & Dinkova-Kostova 2014), systemic inflammation and an increase in secretion of the hormone leptin, all contribute to a decreased ability to metabolise lipids, leading to a default storage of energy as adipose tissue (Saltiel 2012).

Appetite regulation in obese individuals may also be affected, rendering successful weight loss challenging. The metabolic dysregulation of appetite control is often reflected in changes of the hormones, ghrelin and leptin (Steinert et al. 2017). The satiety hormone leptin is secreted by the adipocytes and together with ghrelin plays a role in hunger and satiety regulation (Farr et al. 2015). Any imbalances of these two hormones have consequences on energy homeostasis and therefore body weight regulation (Morioka et al. 2016). Circulating leptin plasma concentrations are proportional to fat mass and are elevated in obese individuals. Leptin crosses the blood-brain barrier (BBB) and binds to the receptors in the hypothalamus, giving information about the status of the body's energy stores. Leptin acts on the hypothalamus in the brain by inhibiting the neurotransmitter, neuropeptide Y (NPY) which results in a decrease in appetite and cessation of food intake (Klok et al. 2007). However, in obese individuals this is not the case despite elevated leptin levels (Morioka et al. 2016). Leptin resistance seen in obese individuals is due to either a decrease in leptin transport across the BBB or because of defective intracellular signaling via the leptin receptor (Banks 2012). Conversely, ghrelin, the appetite-stimulating hormone, is released from the stomach in response to fasting or low energy

intake. Ghrelin crosses the BBB, where it binds to receptors in the hypothalamus. As a result, NPY is released, which in turn triggers feelings of hunger and lead to food intake. In obese individuals, ghrelin levels are decreased. The decreased ghrelin levels in obese individuals may be attributed to defective genes coding for ghrelin and its receptors (Klok et al. 2007). Obesity may also lead to ghrelin resistance that results in reduced NPY responsiveness (Briggs, Enriori et al. 2010). Furthermore, it has been shown that obese individuals display smaller post-prandial reductions in ghrelin levels, which may not lead to the normal reduction in the speed of gastric emptying. Consequently, the obese individual may experience reduced feelings of satiety and consume more food than is required (Le Roux et al. 2005, Klok et al. 2007). Thus, the regulation of energy balance is biased toward protection against weight loss, further fat accumulation, and disease progression (Wells 2012).

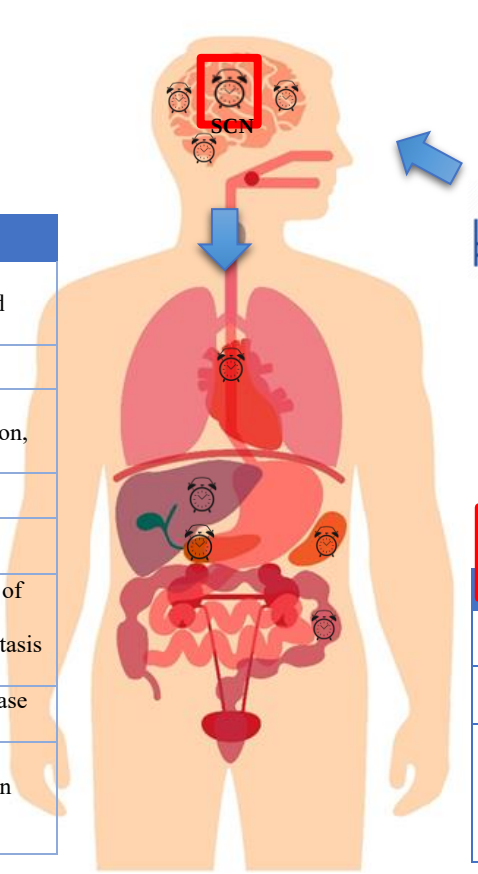
2.4 The role of circadian timing in obesity aetiology

2.4.1 The circadian timing system

Most organisms, including humans have evolved an internal time keeping system which involves internal circadian clocks that generates various rhythms or cycles of metabolism, behaviours (including sleeping, eating and activity), and gene expression (Ko & Takahashi 2006, Bass 2010, Garaulet & Madrid 2010, Huang et al. 2011). The internal circadian clocks produce rhythms with a period of approximately 24-hours, which explains the reasons why it is referred to as a "circadian" (from Latin, "about a day") clocks (Jiang & Turek 2018). On a molecular level, the molecules which oscillate in a circadian manner are called cellular 'clocks'; they generate self-sustained oscillations of 24-hours via different clock genes and proteins, including Circadian Locomotor Output Cycles Kaput (CLOCK), brain- and muscle aryl hydrocarbon receptor nuclear translocator-like protein-1 (BMAL1), Period (PER) and Cryptochrome (CRY) within cells. These genes and proteins work via autoregulatory feedback loops that takes approximately 24-hours to complete. (Schibler 2005, Bass 2010, Mohawk et al. 2012). These endogenous rhythms generated by the clock genes deliver intracellular and extracellular temporal signals (Armstrong 1980) which are repeated approximately every 24 hours. This circadian timing system consists of a core clock located in the suprachiasmatic nuclei (SCN) of the hypothalamus (Moore 1972) and body clocks in all peripheral tissues, organs and cells throughout the body. The peripheral clocks have been shown to be responsible for the various timed physiological functions within these specific tissues and organs, including cardiovascular function, glucose homeostasis, lipid metabolism, hormone secretion, digestive function, gastrointestinal motility and immune response (Bass 2010, Garaulet & Madrid 2010, Huang et al. 2011) (further functions are depicted in (**Figure 2.2**). The circadian rhythms of clocks are present in each cell of the body and brain and are controlled by the core /central clock in the SCN (Moore 1972). The timing (phase) of each circadian rhythm is defined by external factors (zeitgebers a German word for "time giver) in a process known as entrainment (Moore 1972, Moore 1973). External light is the primary zeitgeber for the central

clock and is synchronised to the 24-hour light/dark cycle of the earth's daily rotation on its axis (King et al 1997). The SCN receives external light input from the eyes via the optic nerve and synchronises the downstream peripheral body clocks by utilising various neural and hormonal signalling pathways, such as timed melatonin and cortisol secretion (Czeisler et al. 1999). While light is the primary zeitgeber for the central circadian clock, other external cues such food intake, including the timing and composition of the food intake, is capable of setting the rhythms in the peripheral clocks as well as the clock-controlled genes in the body tissues and organs (Reppert & Weaver 2002, Bass 2012, Youngstedt et al. 2016, Jiang & Turek 2017). These clock genes in turn influence the timing of digestion, nutrient uptake and metabolism, metabolite and hormonal regulation, food intake, behavior and appetite (Bass 2012, Jiang & Turek 2017). Timing of food intake during the 24-hour day as well as composition of food intake (particularly macronutrients) is therefore an important zeitgeber that sets the phases of the peripheral clocks (Manoogian & Panda 2017).

Organ/Tissue	Peripheral clock function
Brain	Regulates CNS functions of sleep/wake, mood, learning, reward and neurogenesis.
Heart	Regulates cardiac metabolism.
Liver	Role in metabolic processes of lipogenesis, xenobiotic detoxification, carbon oxidation and lipogenesis.
Pancreas	Regulates insulin secretion.
Kidney	Regulates water/electrolyte homeostasis and blood pressure.
Adipose tissue	Adipogenesis & the differentiation of white, brown and beige adipocytes which is crucial for energy homeostasis and thermogenesis.
Intestine	Regulates lipid absorption and release of gut hormones
Gut microbiota	May have their own endogenous clocks. Microbiota display circadian rhythmicity of composition and function.



Master clock
Entrained by the 24-hour light/dark cycle.
Synchronizes the downstream peripheral clocks.
Via various neural and hormonal signalling pathways and indirectly by sleep/wakefulness, fasting/feeding and body temperature cycles.

Figure 2.2 - *Physiological functions of the central & peripheral clocks adapted from (Jiang & Turek 2018)*
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2.4.2 Circadian rhythmicity of metabolic processes & endocrine regulation

The influence of the circadian rhythms in metabolism is evident by the diurnal rhythms seen in various metabolic parameters, such as circulating metabolite levels, insulin sensitivity and secretion as well as energy expenditure (Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997). In normal conditions, humans are diurnal in nature, meaning that, they usually spend about two thirds of their 24-hour day awake, being active, eating and storing energy (daytime). They usually spend one third asleep, being in a fasting state at night-time (Buxton et al. 1997). Humans are physiologically suited to a daytime-feeding and night-time-fasting pattern, depicted in **Figure 2.3**.

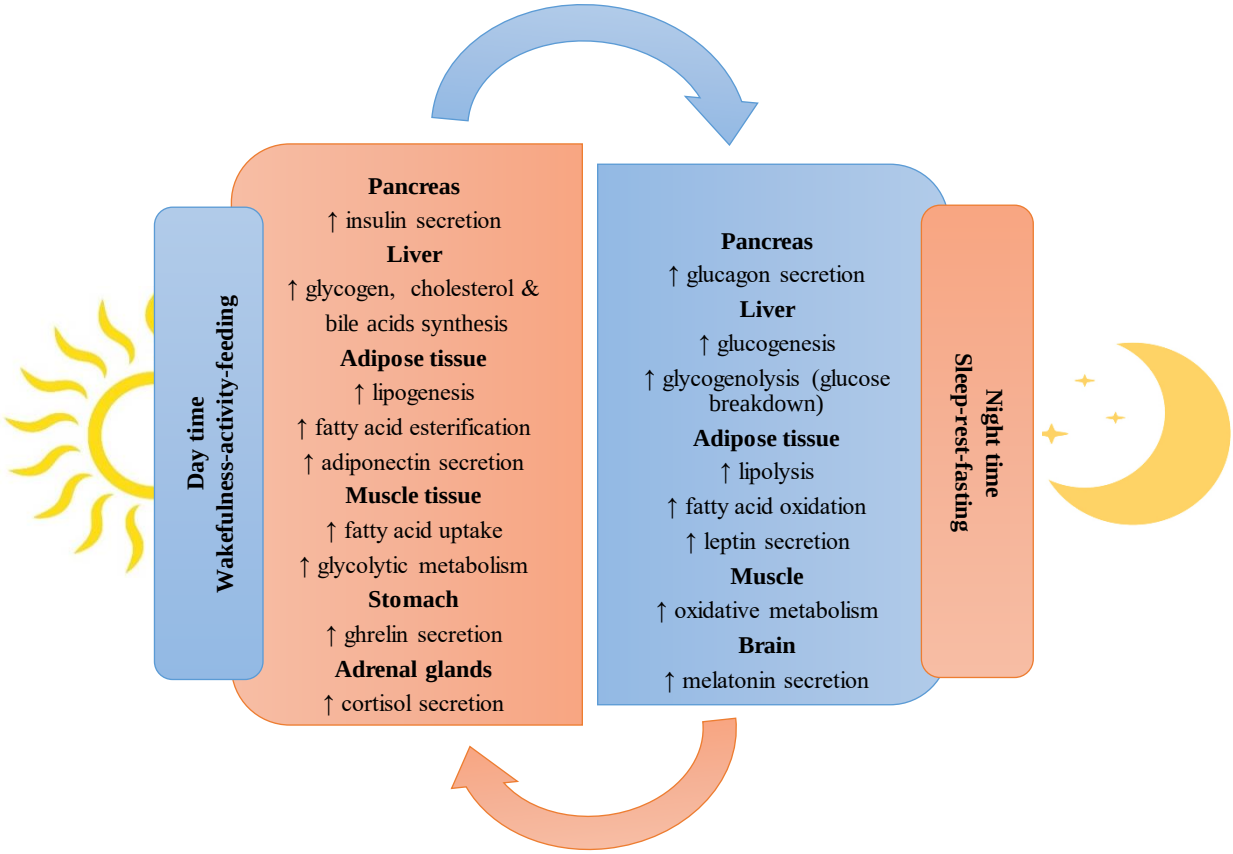

Figure 2.3 *- Circadian rhythmicity and interaction of metabolic processes & endocrine regulation adapted from (Covassin et al. 2016)*
Royalty-free stock illustration ID:1656431713, Moon crescent star (Kuzminka, T.) Shutterstock.com. 5 Royalty-free stock vector ID: 1378107854, Sun icon vector isolated (Maksym Drozd) Shutterstock.com. 2 June 2023 May 2023

During the daytime, metabolic processes involved in energy uptake and storage dominate (Cipolla-Neto et al. 2014). This is shown by the elevated peripheral and central insulin sensitivity and optimal glucose tolerance (Cipolla-Neto et al. 2014). Research has shown the diurnal rhythm of glucose concentrations, whereby glucose tolerance is the highest in the morning and lowest in the afternoon and evening which may be in part mediated by the diurnal rhythms of insulin sensitivity and β-cell responsivity (Roberts

1964, Van Cauter et al. 1997, Saad et al. 2012). Thereby showing that daytime, especially the morning, is the ideal phase of the 24-hour day for acquiring energy through dietary intake (Cipolla-Neto et al. 2014). The daytime is further ideal for storage of energy. This is shown by the upregulation of metabolic processes such as glycogen synthesis, glycolysis in the liver and skeletal muscles, increased lipogenesis, adiponectin synthesis and the downregulation of processes that creates energy, like hepatic gluconeogenesis (Cipolla-Neto et al. 2014). Additionally, the body requires more energy during the day, which is shown by the higher thermic effect of food in the morning and noon (Damiola et al. 2000); elevated energy expenditure; increased gut activity (Takahashi et al. 1968); upregulation of the hunger hormone, ghrelin and decreased secretion of the satiety hormone, leptin (Takahashi et al. 1968). In comparison, the resting phase is characterised by mobilisation of stored energy forms and hormones that encourage cessation of food intake. Metabolic processes that release energy are such as gluconeogenesis, glycogenolysis and lipolysis are upregulated (Cipolla-Neto et al. 2014). Lipid mobilisation is prominent at later in the day and night, shown by higher concentrations of circulating fatty acids (Takahashi et al. 1968), triglycerides (van Moorsel et al. 2016) and cholesterol. Arrendondo-Amador et al. recently found that the hormone-sensitive lipase (enzyme in control of lipid mobilisation) peaks around midnight, demonstrating that lipid mobilisation shown an endogenous circadian rhythm (2020). The secretion of the satiety hormone, leptin is upregulated, encouraging cessation of food intake (Cipolla-Neto et al. 2014). During this nightly prolonged fasting period, stored energy is thus utilised to maintain cellular homeostasis, making this phase suited for rest and fasting (Cipolla-Neto et al. 2014).

2.4.3 Measurement of circadian phase & chronotypes

Circadian phase in humans can be determined using different approaches. One approach is to use physiological markers for example, cortisol secretion in the morning, the onset of melatonin secretion in the evening or early night or measurements of core body temperature (Wirz-Justice 2007, Adan et al 2012, Burgess et al. 2015). Measuring the onset of melatonin secretion under dim light conditions in the evening (Lewy & Sack 1989) is the golden standard method of determining circadian phase. Melatonin concentrations generally increase two to four hours before the onset of nocturnal sleep (Burgess 2008). Therefore, dim light melatonin onset (DLMO) is used as a circadian marker and the term, phase angles of entrainment, indicate the time window between DLMO and habitual bedtime (Burgess et al. 2015). Measuring melatonin secretion onset time is not always feasible, due to the high costs, respondent burden and labour intensiveness for the researcher (Kantermann et al. 2015). Another approach is to use self-assessments from questionnaires and use habitual sleep-wake preferences as a proxy for circadian phase (Horne & Östberg 1976, Folkard et al. 1979, Torsvall & Åkerstedt 1980, Adan et al 2012). An individual's chronotype can be determined by these questionnaires. The term chronotype (Roenneberg et al. 2003) is widely used to describe the preferred sleep-wake timing of an individual relative to the light-dark cycle that influences the timing of their diurnal preferences and the

modulation of physiological functions and behaviour (Ehret 1974, Horne 1976, Klei et al. 2005, Adan et al 2012). There is also evidence of a genetic basis for an individual's chronotype (Hardin et al. 1990, Ebisawa et al. 2001, Mishima et al. 2005). Several questionnaires are available to estimate chronotype by assessing self-reported behavioural patterns, such as the Morningness-Eveningness Questionnaire (MEQ); the Diurnal Type Questionnaire; the Circadian Type Questionnaire as well as the Munich Chronotype Questionnaire (MCTQ) (Horne 1976, Folkard et al. 1979, Torsvall & Åkerstedt 1980, Adan et al 2012).

The MEQ and MCTQ are the most widely used questionnaires. The MEQ consists of 19 items that ask the respondent to indicate their preferred clock time for activities (such as work and physical activity) and preferred sleep times. The MEQ assesses morning appetite and alertness, dependency on alarm clocks and evening tiredness (Horne & Ostberg, 1976). The MEQ score is therefore regarded as a subjective preference for behaviour during wake times (diurnal preference). The MCTQ is a subjective assessment of habitual sleep/wake timing during the last four weeks. The MCTQ, was developed by Roenneberg and co-workers (2003) and focusses on habitual sleep/wake timing in the last four weeks, separately weighted for sleep timing on free and workdays. The MCTQ allows assessment of social jetlag, which is the difference between habitual sleep and wake times on free and workdays. Thus, one of the advantages of the MCTQ is, that it differentiates for sleep-duration between work and free days and can be also adjusted for age, which is not the case for the MEQ (Roenneberg et al. 2003).

This questionnaire assesses self-estimated habitual sleep-wake times of the last four weeks by differentiating between weekdays and work-free days. The parameters involved include midsleep (i.e., the midpoint time between sleep onset and waketime) on weekdays (MSW), mid-sleep on free days (MSF), and mid-sleep on free days, that is corrected for sleep duration on weekdays (MSFsc). The MCTQ responses are used to calculate MSW and MSF. In turn, MSW and MSF is used to calculate MSFsc. The questionnaire uses MSFsc as a chronotype predictor. Intrinsic sleep-wake time preferences in humans can be classified as either early chronotypes (=morning types; MT), intermediate chronotypes (IT) or late chronotypes (=evening types; ET (Ehret 1974, Horne 1976, Adan et al 2012). The MT habitually prefer an early bedtime and early morning rise time (Ehret 1974, Horne 1976, Adan et al 2012). On the other hand, ET, prefer a later bedtime and a late morning rise time (Ehret 1974, Horne 1976, Adan et al 2012). A higher MSFsc reflects later midsleep which is an indicator for late chronotypes, i.e., towards ET (Roenneberg et al. 2003) and vice versa for early chronotypes (MT); i.e.: midsleep time < 03:00 = MT; midsleep time between 03:00 and 05:00 = IT; midsleep > 05:00 = ET. Both MT and ET types have been shown to exhibit genetic differences in allele frequencies (Archer et al. 2003, Chang et al. 2019), and different intrinsic period length of the circadian clocks (Brown et al. 2008) as well as different phase angles of entrainment e.g., between circadian phase of the melatonin rhythms and sleep wake times (Duffy et al. 2001).

Scores from both the MCTQ and MEQ have been shown to correlate high with DLMO and can therefore be used as a proxy for the circadian phase of an individual (Kantermann et al. 2015, Randler & Engelke 2019). One limitation of both questionnaires is that they assess subjective measures which may not necessarily reflect objectively measured internal circadian phase. Nevertheless, when the MEQ scores and the MSFsc (derived from the MCTQ) were compared with the DLMO, the MSFsc revealed to be the strongest predictor for objectively assessed circadian phase, i.e., the DLMO (Kantermann et al. 2015). Both questionnaires showed also high association with each other (Kitamura et al. 2014).

2.4.4 Circadian misalignment

To optimise the body's biological functions and achieve ideal homeostasis, the circadian clock needs to be entrained by environmental and behavioural zeitgebers. In doing so, the internal rhythms are synchronised both within the body and with the external environment. However, the central clock and the peripheral clocks can become misaligned when their respective zeitgebers (e.g., light input vs timing of food intake) are out of sync. A zeitgeber can aid in resetting the circadian clock, but it can also become a *desynchroniser*, when provided or experienced at the incorrect time of the daily cycle (Salgado-Delgado et al. 2010, Nelson & Chbeir 2018, Challet 2019). Circadian misalignment may disrupt proper metabolic function, since the two clock systems jointly coordinate various metabolic pathways, ultimately resulting in metabolic changes including insulin resistance, impaired glucose homeostasis, dyslipidaemia as well as weight gain and obesity (Reppert & Weaver 2002, Qin 2003, Farshchi et al. 2005, Scheer et al. 2009, Antunes et al. 2010, Roenneberg et al. 2012, Wang et al. 2014, Parsons et al. 2015, Mota 2017). In today's modern society work schedules and social demands challenge the circadian clock. Some individuals, (ET) prefer to rise late in the morning and extend their wakefulness into the night, meaning that evenings are no longer set aside for only rest and sleep, but daytime activities such as work, and food intake also occur (Ehret 1974, Horne 1976, Adan et al 2012). Other individuals (MT) prefer to wake early in the morning and go to bed early at night (Ehret 1974, Horne 1976, Adan et al 2012), ceasing their food intake early. The majority of individual chronotypes lie in a range between MT and ET. Individuals at the ends of the distribution, manifests as either extreme MT or extreme ET and may experience substantial misalignment between their internal circadian rhythms and work and social demands, resulting in potentially adverse health outcomes (Covassin et al. 2016).

Individuals, like ET, who prefer to extend their wakefulness to the night are exposed to light at night, which is known to delay melatonin onset (biological marker of impending sleep onset) as well as decrease total melatonin secretion (Boivin & Czeisler 1998, Obayashi et al. 2013). Melatonin plays a regulating role in glucose and lipid metabolism and a decreased secretion may lead to metabolic dysfunction and lipid accumulation (National Sleep Foundation 2011). The adverse hormonal- and metabolic responses of nocturnal light exposure include increased insulin resistance, elevated

postprandial insulin, glucose, GLP-1 concentrations and high body weight and waist circumference (Albreiki et al. 2017). Individuals, like ET, who tend to delay and restrict their sleep period during weekdays, will often exhibit "catch-up" sleep behaviour over weekends. This time difference between workdays and free days is called social jetlag because it reveals an abrupt change of sleep-wake timing which is often also due to social activities (Wittmann et al. 2006). Studies have shown positive associations between social jetlag and an increased body weight, fat accumulation and metabolic derangement such as inflammation, impaired glucose regulation (Wittmann et al. 2006, Roenneberg et al. 2012, Broussard 2016) metabolic syndrome and type two diabetes mellitus (Al-Naimi et al. 2004, Parsons et al. 2015, Koopman et al. 2017, Boivin & Czeisler 1998, Garaulet 2020).

Another source of circadian misalignment besides nocturnal light exposure and social jetlag is the diet. When ET have a later wake and sleep time, mealtimes also shift to later in the day. Therefore, along with sleep and wakefulness, food intake also occurs in misalignment with the normal day/light cycle of metabolic processes and endocrine regulation. Scheer et al. showed the detrimental effects of shifting normal sleep-, wake- and mealtimes by 12 hours. Participants showed reduced glucose tolerance, increased blood pressure and decreased leptin concentration (2009). Therefore, organising daily sleep, wakefulness and food intake relative to the circadian timing system is vital to prevent misalignment.

An individual's chronotype may thus be considered a driver of sleep-, wake- (Duffy et al. 2001) and mealtimes. When these are experienced at the incorrect time, they become *desynchronisers* of the circadian timing system. The timing and composition of food intake relative to daily activities serve as a powerful zeitgeber. Therefore, eating at an inappropriate time, e.g., during the night, may have a desynchronising effect on the biological clocks and in the long-term, may result in adverse health outcomes e.g., weight gain, obesity, and poor metabolic function (Salgado-Delgado et al. 2010, Nelson & Chbeir 2018, Challet 2019). The diet is complex and involves other factors besides meal timing, such as nutrient content and diet quality (Leech et al. 2015), all of which may be influenced by chronotype (Sato-Mito et al 2011, Kanerva et al. 2012, Reutrakul et al. 2013, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018). To comprehend the role of the diet in circadian misalignment as well as the effect of chronotype as driver, firstly the role of the diet in obesity development must be discussed.

2.5 The role of the diet in obesity

Nutritionally sound diets are the foundation of health and wellbeing (Leech et al. 2015). Obtaining essential nutrients and dietary components are vital for survival and can prevent and/or promote disease development. It is well-established that a poor diet contributes to adverse health outcomes and consequently increases the risk of diseases such as inflammatory diseases, hypertension, diabetes and obesity (McHill et al. 2011). The role of dietary intake in obesity aetiology is often simplistically described as excessive energy intake, although the diet can impact health status and energy balance

beyond merely providing energy. Body weight regulation is dependent on a homeostatic feedback system that acts on energy intake and energy expenditure to maintain a stable body weight (Park 2019). Various factors can influence the 'intake' and 'expenditure' sides of the energy balance equation, including total energy intake, dietary macronutrient composition (percentage of energy from carbohydrates, fat and protein) (Galgani & Ravussin 2008, Flatt 2011), and the energy density of the diet (Rolls et al. 2006, Ledikwe et al. 2007, Galgani & Ravussin 2008).

In recent years, a gradual shift in diet-disease- risk research has occurred. Dietary assessment approaches have moved away from a simplistic single-nutrient-only approach to new assessments that take into consideration the nutrient interactions and bioactive compounds within the whole diet (Leech et al. 2015). This new food-based approach takes into consideration that individuals don't eat singular nutrients, they consume a variety of foods typically grouped together at different eating occasions (as meals or as snacks) across the 24-hour day This represents an eating pattern for referral to eating occasions across the day, or a meal pattern which refers to the combination of foods consumed at one meal or eating occasion (Kant 2018).

Eating patterns are important determinants of energy and nutrient intake as well as diet quality. The term *eating patterns* is used to describe an individual's food intake at the level of a meal. The term eating pattern is used to describe dietary intake in terms of *format* (food combinations and nutrient content) and *patterning* (spacing, skipping and timing). Dietary guidelines for a healthy body weight and metabolic profile, frequently include recommendations such as intake of a variety of foods (Ministry of Health 2022), three or more regular meals (Speechly & Buffenstein 1999) and breakfast consumption.

The format of meals in terms of the food type and the contributing macronutrient content forms an important part of an eating pattern. It is well known that body composition is influenced by a range of interplaying factors including the types and combinations of foods consumed together (Miller et al. 1990, Satia-Abouta et al. 2002, Newby et al. 2003, Ahluwalia et al. 2009, Paradis et al. 2009, Austin et al. 2011, Suliga et al. 2015). It is often recommended that a healthy eating pattern should include a variety of micronutrient rich foods (Ministry of Health 2022), while being low in overall energy density (Pérez-Escamilla et al., Obbagy et al. 2012, Rouhani et al. 2016). The higher prevalence of weight gain and obesity in recent years is largely attributed to an increased intake of energy dense foods (such as fast foods and sugar sweetened beverages) in large portion sizes (Rouhani et al. 2016). Globalisation has contributed to a shift away from "traditional dietary patterns" (Popkin 2006, Kearney et al. 2010) to more "unhealthy dietary patterns" that are high in energy dense foods such as refined grains, sugar-sweetened beverages, animal products and vegetable oils (Hu 2011, Basu et al. 2013). These unhealthy foods are highly palatable, ultra-processed, inexpensive, and widely available, contributing to their high intake among populations (Malik et al. 2013, Vandevijvere et al. 2015, Rico-Campà et al. 2019) and

especially those with a lower socio-economic status (Anand et al. 2019). A meta-analysis by Rouhani et al. concluded that high dietary energy density is associated with an elevated adiposity risk and is directly associated with weight gain in both men and women (2016) as shown by cohort studies. Furthermore, high energy density is associated with an increased risk of metabolic syndrome and diabetes (Wang et al. 2008, Esmaillzadeh & Azadbakht 2011).

Often the recommended intervention strategies for obesity, include energy restricted diets and increased PA, although these interventions have largely been unsuccessful in promoting long-term weight loss (Taubes et al. 2013). It is suggested that aside from the energy content of the diet, the quality and distribution of macronutrients are important determinants in weight regulation (Miller et al. 1990). The precise role of carbohydrates and lipids in obesity development is controversial due to mixed results from the literature (Miller et al. 1990, Satia-Abouta et al. 2002, Ahluwalia et al. 2009, Van Baak & Astrup 2009, Austin et al. 2011, Yancy et al. 2014, San-Cristobal et al. 2020). Cross-sectional studies found that protein and carbohydrate intakes are inversely associated with BMI, while fat intake is positively associated with BMI (Miller et al. 1990, Satia-Abouta et al. 2002, Ahluwalia et al. 2009, Austin et al. 2011). However, RCTs found mixed results, with some showing low-fat diets (Saris et al. 2000) more beneficial and another showing low-carbohydrate diets more beneficial for weight loss (Brehm et al. 2003). A meta-analysis considered the evidence from 19 RCT's that compared the effects of low carbohydrate vs isoenergetic balanced weight loss diets. The meta-analysis concluded that both diets had short-term weight loss success, but little or no differences were seen after two years follow up in weight loss and cardiovascular risk factors (Naude et al 2014). Another meta-analysis by Ge et al. showed similar results, with both the low carbohydrate and low-fat diets resulting in weight loss at six months (2020). These findings suggest that the quantity of carbohydrate and fat in the diet is not the most decisive factor for weight loss.

Apart from the quantity of carbohydrates and fats in the diet, it has been proposed that the quality of these macronutrients is a crucial determining factor of the effect of the diet on body weight regulation (Aller et al. 2011, Forouhi et al. 2018, Reynolds et al. 2019). High-fibre carbohydrates with a low glycaemic index are associated with a reduced risk of obesity (Aller et al. 2011), while a diet rich in simple carbohydrates, such as sweets and sugar-sweetened beverages are associated with increased obesity risk (Malik et al. 2010). Similarly, the quality of dietary fat may be more important than the quantity consumed. For example, in the case of population groups that consume a diet favouring unsaturated fatty acids (FA) intake (Bes-Rastrollo et al. 2006, Gulati & Misra 2017, Beulen et al. 2018) rather than saturated FA intake, such as the Mediterranean diet, are at a lower risk of developing obesity and cardiovascular disease. It is also shown that the health effects of substituting refined carbohydrates or saturated fatty acids (SFA) depend on the quality of dietary fat or carbohydrate. For example, when either refined carbohydrates or SFA is replaced with either polyunsaturated fatty acids **(**PUFA),

monounsaturated fatty acids (MUFA) or complex carbohydrates, a reduction in cardiovascular risk is seen. However, when refined carbohydrates is substituted with SFA and trans fatty acids (TFA) no change or an increased risk of cardiovascular risk is seen. Similarly, when SFA is replaced by refined carbohydrates or TFA no change, or an increased risk of cardiovascular risk is seen (Alexander et al. 2022). This evidence confirms the importance of carbohydrate and fat quality in the diet. The proportion of protein in the diet also has a role in body weight via appetite regulation. Diets that are high in protein has been shown to reduce the feelings of hunger and increase energy expenditure (Veldhorst et al. 2010), with some research suggesting a higher intake of protein is beneficial in overweight and obese individuals (Arciero et al. 2013).

In recent years, research has shifted away from the single-nutrient studies to a whole-diet assessment such as dietary pattern analysis, (Krebs-Smith et al. 2015) identifying dietary patterns based on the combinations of foods and nutrients consumed together (Frank 2002, Krebs-Smith et al. 2015, Liese et al. 2015). Dietary pattern analysis studies have identified "unhealthy dietary patterns" including the "western-" (Malik et al. 2013, Vandevijvere et al. 2015, Rico-Campà et al. 2019), "animal protein" (Bosetti et al. 2013, Vajdi et al. 2020) and "starch rich" (Bosetti et al. 2013) food and nutrient patterns in adults. This "western dietary pattern" is characterised by energy dense and micronutrient poor foods such as sugar sweetened beverages, fast foods, refined grains, red- and processed meats, and confectionery. This pattern is associated with weight gain and obesity as well as an elevated risk for the development of lifestyle diseases (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015). The "animal protein dietary patterns", are high in cholesterol, fat, SFA and animal protein and is associated with greater odds of metabolic syndrome, especially elevated triglyceride concentrations (Vajdi et al. 2020) and pancreatic cancer risk (Bosetti et al. 2013). Lastly the "starch rich pattern" and "refined and processed" is high in carbohydrates and is associated with increased pancreatic cancer risk (Bosetti et al. 2013).

Dietary pattern analysis has identified several healthy patterns such as the "prudent dietary pattern", "micronutrient rich" (Palli et al. 2001), and "vitamins and fibre" (Bosetti et al. 2013, Schrijvers et al. 2016). These patterns are considered healthy due to the high micronutrient and fibre content and related food such as whole grains, fruits, vegetables, legumes, and fish. The "prudent dietary pattern" is associated with a lower waist circumference and BMI as well as lower risk for disease development (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015). The micronutrient dietary patterns were shown to be associated with a lower risk of pancreatic and gastric cancer, most likely due to the ability of the antioxidants to clear free radical damage (Palli et al. 2001, Bosetti et al. 2013).

Another recommendation of a healthy diet is to consume regular meals (usually three main meals with snacks in between) (Speechly & Buffenstein 1999). A range of studies in adults (study samples between 641 – 28 098) have shown that consuming between three and six meals per day reduces the risk for

obesity (Fabry et al. 1964, Ma et al. 2003, Holmbäck et al. 2010, Aljuraiban et al. 2015), improves lipid profiles (Fabry et al. 1964, Edelstein et al. 1992, Titan et al. 2001), and glucose tolerance (Fabry et al. 1964). Regular meals are thought to increase dietary induced thermogenesis (energy dissipated as heat after food intake) (Tai et al. 1991, Bellisle et al. 1997, Westerterp-Plantenga et al. 2003), increase satiety (Speechly and Buffenstein 1999, Leidy & Campbell 2011) and delay gastric emptying (Jackson et al. 2007), thereby both increasing energy expenditure and reducing energy intake, although there is insufficient evidence to support this hypothesis. A large study conducted in the United States of America (n = 7791), concluded that a higher meal frequency was associated with a lower energy density and BMI (Zhu & Hollis 2016). Fewer studies have found results that showed the opposite, where a lower meal frequency of less than two per day is protective against obesity (Howarth et al. 2007, Bes-Rastrollo et al. 2010, Kahleova et al. 2017). The positive associations between higher meal frequency and higher risk for obesity may be due to the overall higher energy intake (Howarth et al. 2007, Mills et al. 2011, Casazza et al. 2015) seen in the high meal frequency group. Additionally, studies that have investigated the associations between meal frequency and body composition vary largely in their methodology including covariates adjusted for and whether participant underreporting of energy intake was addressed (Flegal 1999). Another consideration is the timing of meals. A large study of 50660 adults not only found that a longer overnight fasting period (≥ 18 hours) was associated with a lower BMI but also that breakfast skippers had a higher BMI (Kahleova et al. 2017).

The daily consumption of a healthy breakfast is shown to be a protective measure against weight gain and obesity (Purslow et al. 2008, Szajewska & Ruszczyński 2010, Kahleova et al. 2017, Gwin & Leidy 2018) as well as coronary heart disease (Cahill et al. 2013). When breakfast is not consumed, the overnight fasting period is prolonged and an increase in postprandial insulin concentrations and fat oxidation is seen (Nas et al. 2017). Ultimately low-grade inflammation and impaired glucose metabolism result in the development of metabolic inflexibility (Nas et al. 2017). Another proposed mechanism by which breakfast consumption protects against weight gain (Purslow et al. 2008) is, by causing satiety early in the day and thus reducing the desire to overconsume food later within the day to make up for the missed early meal (Casazza et al. 2015, Tani et al. 2015). Regular breakfast eaters also have a higher diet quality due to the high fibre and nutrient rich foods that are regularly consumed at breakfast (Timlin & Pereira 2007). These finding suggest that besides the meal frequency, the timing of meals are important considerations in body weight regulation.

2.6 The role of eating patterns as zeitgeber

Eating patterns are determinants of both the quantity and quality of nutrients in the diet and have varying effects on body weight regulation. Eating patterns in terms of format and patterning can also serve as a powerful zeitgeber. There is clear evidence that macronutrient metabolism and energy expenditure exhibit a marked morning-evening difference (see **Figure 2.3**). Therefore, the physiological responses

to food consumed at different time points in the day may differ and eating patterns may either have a synchronising or desynchronising effect on the circadian timing system. Various animal and human studies have demonstrated that food intake at an inappropriate time of the day can have a desynchronising effect on the biological circadian clocks, resulting in adverse health outcomes including weight gain, obesity as well as poor metabolic health outcomes (Salgado-Delgado et al. 2010, Nelson & Chbeir 2018, Challet 2019).

Animal studies

Animal studies provided the first evidence for the role of circadian rhythms with regards to food intake and body weight control. Professor Fred Turek and his colleagues demonstrated the importance of properly timed food intake in nocturnal rodent models (Arble et al. 2009, Fonken et al. 2010, Salgado-Delgado et al. 2010). Nocturnal rodents typically consume about 70 – 80% of the food during their active cycle, which is during the night (Arble et al. 2009). The study showed that nocturnal rodents that were fed a high-fat diet (HFD) during their normal sleep-resting phase (during the day), weighed more than the rodents who were fed the same diet but during the "right time" of their feeding cycle (during the night). The difference in weight was observed, despite the energy intake and PA levels being the same for both groups (Arble et al. 2009, Fonken et al. 2010, Salgado-Delgado et al. 2010). These findings suggests that the timing of food intake, together with dietary intake and PA is relevant in body weight control, likely by ensuring energy balance and optimal metabolism (Fonken et al. 2010, Collado et al. 2018). Interestingly, when feeding is restricted to the rodent's "correct" circadian phase (during the night), circadian rhythms are more robust and seem to be protective against HFD-induced adverse effects, such as obesity, leptin resistance, glucose intolerance and liver damage (Hatori et al. 2012). This evidence suggests that time-restricted feeding (restricting food intake to the correct circadian phase) may be an effective therapeutic intervention for treating obesity and metabolic disorders in humans (Jiang & Turek 2017). These findings in animal models (Arble et al. 2009, Fonken et al. 2010, Salgado-Delgado et al. 2010) resonate with findings from human studies (Garaulet et al. 2013, Reutrakul et al. 2014, Muñoz et al. 2017, Raynor et al. 2018, Leung et al. 2020).

Morning intakes

Breakfast is often referred to as "the most important meal of the day". It is believed that individuals who omit breakfast tend to be hungrier later in the day, leading to an overcompensation of energy intake, especially during the evening (Stanton Jr 1989). The higher energy intake during the night (the normal resting and fasting cycles), result in enhanced fat storage and ultimately obesity. This phenomenon may explain the reason why late-night eaters and breakfast skippers are prone to obesity and related comorbidities (Fong et al. 2017). It is further supported by McHill et al. who showed that obese individuals typically consume most of their energy an hour closer to the melatonin secretion onset time

in the evening, in comparison to lean individuals (2017). Metabolically, ingested calories are optimally used during the morning in comparison with the evening (Cipolla-Neto et al. 2014, Roberts 1964, Van Cauter et al. 1997, Saad et al. 2012, Takahashi et al. 1968). The mechanisms responsible for the enhanced morning energy metabolism is not yet fully understood. Yosida et al. demonstrated the importance of breaking this overnight fast with food intake at the start of the active phase, by using a nocturnal mice model (2012). Their study concluded that short term early nocturnal fasting (equivalent to breakfast skipping) resulted in an increased energy intake, elevated lipogenesis and weight gain (Yoshida et al. 2012). Similar to animal models, the importance of breakfast consumption in preserving the clock-controlled gene activity was demonstrated in humans by Jakubowicz et al. showing that acute breakfast omission led to changes in the expression of clock genes and consequently, disruption of the circadian rhythms (2017). With alterations of the circadian clock and the clock-controlled genes, a change in metabolism is expected and, in this study, an enhanced postprandial glycaemic response was exhibited in both the healthy and type two diabetes groups (Jakubowicz et al. 2017).

Late lunch eating

The adverse effects of late lunch eating are supported by evidence from weight loss trials. A weight loss intervention study by Garaulet et al. demonstrated a difference in weight loss success between early (before 15:00) and late lunch eaters (after 15:00) (2013). The early eaters lost more weight in comparison with the late eaters on the same energy restricted diet, despite being of a similar age and having a similar energy intake, macronutrient distribution, estimated energy expenditure, appetite hormone values and sleep duration (Garaulet et al. 2013). Similarly, a late lunch time was associated with less successful weight loss in bariatric surgery patients (Ruiz-Lozano et al. 2016). The results from these two studies prompted the researchers to investigate the reason why meal timing affects weight loss. The study consisted of 32 lean, young women, who received standardised meals at two different times: early eating (lunch at 13:00) and late eating (lunch at 16:30). After one week of eating lunch later, the lean women showed metabolic changes that are typically seen in obese women such as a decrease in resting energy expenditure, decrease in carbohydrate oxidation, decrease in glucose tolerance as well as blunted daily cortisol (Garaulet 2020).

Night vs day eating

Human studies have shown the adverse effects of consuming food during the night in studies with night shift workers (Morikawa et al. 2008, Mota et al. 2013, Balieiro et al. 2014, Samhat et al. 2020), RCT's (Jakubowicz et al. 2013, Raynor et al. 2018) and an observational study (Bo et al. 2014).

Night shift workers often experience chronic circadian misalignment (Chaput et al. 2022), as they are required to work during the night which is the normal sleep-rest phase. This is usually accompanied by

food intake during the night, thereby requiring the body to metabolise nutrients during the normal fasting cycle (Dumont et al. 2001, Waterhouse et al. 2003, Sharifian et al. 2005, Leung et al. 2020). Studies have found adverse effects of shift work in terms of on body composition, including, a higher risk of weight gain, obesity and metabolic syndrome (Morikawa et al. 2008, Antunes et al. 2010, Roenneberg et al. 2013). Other metabolic changes include increased LDL-cholesterol, triglycerides, insulin, and total cholesterol concentrations (Ghiasvand et al. 2006, Biggi et al. 2008). When investigating dietary intake, a meta-analysis found there are no differences in total energy intake between day and night shift workers (Bonham et al. 2016). There are also some differences in the types of foods consumed. Night shift workers are more likely to make unhealthy food choices and are shown to consume more servings of oils, meats, sweets, SFA, dietary cholesterol, and caffeine, while having fewer servings of fruits and vegetables in comparison with day shift workers (Morikawa et al. 2008, Mota et al. 2013, Balieiro et al. 2014).

Demonstrating the importance of the nightly fasting period, a randomised crossover trial rearranged the meal and snack times of night shift workers with central obesity (Leung et al. 2020). The new meal and snack distribution ensured a five-hour long fasting period between 01:00 and 06:00. At the end of the four-week intervention, body weight was significantly lower, despite the daily energy intake being similar prior to the intervention period. The results from this trial demonstrated the effectiveness of rearranging meal and snack times in obesity treatment by ensuring that the body experiences the normal fasting cycle during the biological night (Leung et al. 2020). The evidence from the studies discussed above, accentuates that food intake at the incorrect time as well as unhealthy food choices (Morikawa et al. 2008, Mota et al. 2013, Balieiro et al. 2014), play a role in obesity and the metabolic derangement seen in shift workers (Morikawa et al. 2008, Mota et al. 2013, Roenneberg et al. 2013, Balieiro et al. 2014).

Similar to the findings from shift work studies are observational studies that have found a positive association between higher energy intake during the night and increased risk of obesity, metabolic syndrome (Bo et al. 2014) and hypertension (Almoosawi et al. 2013). Others have also shown the effect of late-night eating on weight loss success. Jakunowicz et al. randomised overweight and obese women with metabolic syndrome into two weight loss groups (breakfast vs dinner group) (2013). One group consumed 50% of their daily calories at breakfast and the other group consumed 50% of their daily calories at dinner for 12 weeks. In the large breakfast group, a greater reduction of fasting glucose, insulin, HOMA-IR, body weight and waist circumference were seen (Jakubowicz et al. 2013). Similarly, a randomized controlled eight-week pilot study demonstrated that overweight adults who ate 50% of their calories at breakfast, 30% at lunch and 20% at dinner lost more weight in comparison with those who consumed 20% of their calories at breakfast, 30% at lunch and 50% at dinner (Raynor et al. 2018).

Chronotype-specific eating patterns

In recent years, an increased number of studies have examined the relationship between chronotype and dietary intake, with a main focus on absolute food intake (Sato-Mito et al 2011, Kanerva et al. 2012, Reutrakul et al. 2013, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018) and to a lesser extent on dietary intake distribution (Muñoz et al. 2017); a clear link between chronotype and the diet is yet to be established. These studies demonstrated that ET and MT tended to consume a similar amount of total daily energy (Sato-Mito et al 2011, Reutrakul et al. 2013, Muñoz et al. 2017). However, there are variances in terms of the clock timing of meals (Reutrakul et al. 2013), macronutrient distribution (Muñoz et al. 2017), meal skipping (Reutrakul et al. 2014), total micronutrient (Sato-Mito et al 2011, Kanerva et al. 2012), total macronutrient (Kanerva et al. 2012) and food group intakes (Sato-Mito et al 2011, Kanerva et al. 2012, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018). The majority of studies used a single 24-hour recall (Reutrakul et al. 2013, Reutrakul et al. 2014, Muñoz et al. 2017) or a one month diet history (Sato-Mito et al 2011) to determine dietary intakes and may therefore not be representative of habitual dietary intake. Also, some of these included study populations with diagnosed acute, pre-existing and chronic diseases, such as type two diabetes (Reutrakul et al. 2013, Reutrakul et al. 2014, Nimitphong et al. 2018) and surgical participants (Ruiz-Lozano et al. 2016). This is of importance as the disease aetiology and possible medication use may influence an individual's preference for sleep/wakefulness and consequently fasting/feeding behaviour. In comparison with MT, ET tended to display a more unhealthy eating pattern such as delayed clock times for breakfast and dinner (Reutrakul et al. 2013) as well as more frequent breakfast skipping (Nakade et al. 2009, Reutrakul et al. 2014). The delay in food intake in ET may be due to their later awakening time (Dashti et al. 2015) and may be explained by the two to three hour phase differences in sleep- and wake times of ET vs MT. Therefore, ET are more likely to experience misalignment of their internal clock as a result of the mismatch between their preferred circadian timing and social demands such as work schedules (Wittmann et al. 2006).

Differences in food group intake among chronotypes is the most explored dietary aspect (Sato-Mito et al 2011, Kanerva et al. 2012, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018). Several studies have demonstrated that ET consume more energy dense foods such as sweets and confectionery (Kanerva et al. 2012), sugar sweetened beverages (Kanerva et al. 2012, Muñoz et al. 2017), alcohol (Kanerva et al. 2012) added fats (Muñoz et al. 2017) and consume less micronutrient-rich foods such as fruits (Kanerva et al. 2012, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018), vegetables (Sato-Mito et al 2011, Kanerva et al. 2012, Patterson et al. 2016, Patterson et al. 2018), dairy (Sato-Mito et al 2011) and wholegrains (Kanerva et al. 2012), which may account for the lower total micronutrient intakes reported by Sato-Mito et al. (2011) and Kanerva et al. (2012). It is well known that such an eating pattern high in energy dense foods is associated with weight gain (Rouhani et al.

2016) and an increased risk of metabolic syndrome and diabetes (Wang et al. 2008, Esmaillzadeh & Azadbakht 2011).

Studies have yielded varying results with regards to total macronutrient intakes. The study by Sato-Mito et al. found no differences in total macronutrient intakes between chronotypes with the exception of higher protein and cholesterol intake among MT (2011). Similarly, Kanerva et al. found higher protein but also higher carbohydrate intakes among MT, with higher fat, SFA and sucrose intake among ET (2012). On the other hand, Muñoz et al. reported a higher protein and carbohydrate intake among normal weight ET (2017). Only one study explored the impact of energy and macronutrient distribution on BMI. This study by Muñoz et al. found that lean ET consumed more of their energy at dinnertime and in contrasts overweight and obese ET consumed more energy at lunch time (2017). This raises the question of, whether energy intake in "misalignment with one's chronotype" is an important factor when considering the effect on body weight (Muñoz et al. 2017). The question rests on the basis that energy expenditure and utilisation after a meal varies depending on the circadian phase of the day (Asher 2011, Scheer et al. 2013) and that different chronotypes represent different phases of entrainment (Roenneberg et al. 2007). This may lead to the possibility that optimal meal timing may differ among individuals in accordance with their chronotype (Xiao et al. 2019).

2.7 Eating behaviours that contribute to obesity

Over the past decades, energy dense and palatable foods have become increasingly available, which makes food choices difficult for those seeking to control their body weight (Tuschl et al. 1990). Individuals who are prone to weight gain are more vulnerable to problematic eating behaviours and disturbed eating attitudes (**Table 2.1)** (Dykes et al. 2004, de Lauzon-Guillain et al. 2006, Kruger et al. 2016). The term eating behaviours is broad and encompasses a range of concepts relating to dieting, food choices and motives, feeding practises and eating-related problems such as eating disorders and obesity (LaCaille 2013). Validated questionnaires are available to measure eating behaviours and eating attitudes, each with their own description of behaviours (Garner et al. 1982, Gormally et al. 1982, Stunkard & Messick 1985, Van Strien et al. 1986). For the purpose of this PhD work the Three-Factor Eating Questionnaire (TFEQ) (Stunkard & Messick 1985) and the Eating Attitudes Test-26 (EAT-26) were used (Garner et al. 1982).

The TFEQ was developed in the 1980s to measure eating behaviours in lean and overweight/obese individuals (Stunkard & Messick 1985). The questionnaire consists of three main eating behaviours domains, namely cognitive eating restraint- which is the conscious restriction of food intake to control body weight and shape; disinhibition of control- the loss of control of food intake that leads to overconsumption of food; and susceptibility to hunger- feelings and subjective perceptions of hunger that leads to food consumption (Stunkard & Messick 1985). Each of the three main categories have

subcategories, which is depicted in **Figure 2.4** below.

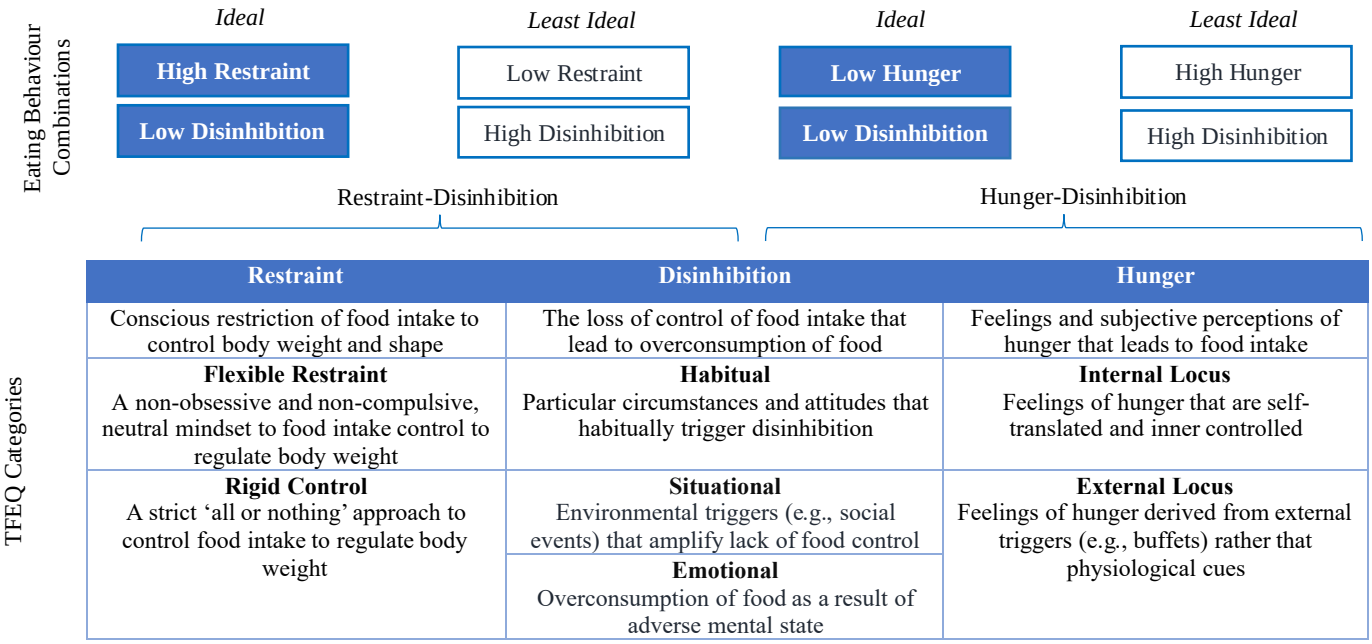

***Figure 2.4** - The Three Factor Eating Questionnaire main categories, subcategories & eating behaviour combinations adapted from (Stunkard & Messick 1985, Westenhoefer et al. 1999, Lesdéma et al. 2012)*

Several researchers, including a NZ based study in women, have demonstrated that these eating behaviours are predictors of body composition including BMI (Dykes et al. 2004, Kruger et al. 2016) and BF% (Kruger et al. 2016), body adiposity (de Lauzon-Guillain et al. 2006), body weight gain (Provencher et al. 2003). It is suggested eating behaviours like those described by the TFEQ affects energy intake (Provencher et al. 2003) by possibly influencing the types and amounts of foods eaten, timing of food intake and where food intake occurs (French et al. 2012). **Table 2.1** depicts the specific eating behaviours and the association with body composition, dietary intake, the hunger and satiety hormones.

Restraint is often used as a dieting strategy to control body weight (Stunkard & Messick 1985) and has been associated with lower body composition (McGuire et al. 2001), energy and fat intakes (McGuire et al. 2001, Provencher et al. 2003). Therefore, those who are more preoccupied with restricting their food intake, consume less food and weigh less than those who don't. The type of *restraint* is shown to be an important determinant of body composition, with higher *flexible control* associated with a lower body composition and greater weight loss success (McGuire et al. 2001, Provencher et al. 2003, Hays & Roberts 2008), likely due a more balanced approach to dieting (Westenhoefer 1991). On the other hand, higher *rigid control* is associated with less successful weight loss attempts (Westenhoefer et al. 1999) and higher body composition (McGuire et al. 2001, Provencher et al. 2003), showing that

individuals who employ strict dieting strategies, are more susceptible to higher *disinhibition* which leads to overconsumption of food (Westenhoefer 1991, Mills et al. 2018).

High *disinhibition* has consistently been shown to be associated with both a higher body composition (Provencher et al. 2003, Bellisle et al. 2004, Dykes et al. 2004, French et al. 2014) and higher energy and fat intakes (Provencher et al. 2003, Bellisle et al. 2004). The subcategories of *situational*, *habitual*, and *emotional disinhibition* have been linked with higher BMI (Hays & Roberts 2008). Both *habitual* and *emotional disinhibition* have been associated with greater energy and fat intakes (Provencher et al. 2003, Bellisle et al. 2004), with *habitual disinhibition* being the strongest predictor of weight gain (Hays & Roberts 2008).

High scores of *hunger* are associated with higher body composition variables (Provencher et al. 2003, Dykes et al. 2004, French et al. 2014) most plausibly due to higher energy intakes (Provencher et al. 2003). The subcategories of hunger have shown less associations with body composition outcomes in the literature. *External locus for hunger* is associated with a lower BMI (Hays & Roberts 2008) but higher fat intake (Provencher et al. 2003). While *internal locus for hunger is* associated higher energy intake (Provencher et al. 2003) and more weight gain in adult women (Hays & Roberts 2008).

The TFEQ's eating behaviours do not act in isolation, but instead *disinhibition* has an interaction effect with either a low or a high level of *restraint* and *hunger* (Hays 2002). This is reflected in findings from studies that showed that *disinhibition* is correlated with *restraint* and *hunger* respectively (Bellisle et al. 2004, Dykes et al. 2004). Body composition is also associated with specific eating behaviours combinations. A high BMI is associated with the least ideal eating behaviours combination of high *disinhibition* - low *restraint* (Dykes et al. 2004), while a normal BMI and BF% are associated with the ideal eating behaviours combination of low *disinhibition* - high *restraint* (Kruger et al. 2016). Additionally, the least ideal eating behaviours combination of high *disinhibition* - high *hunger* is associated with high BMI (Provencher et al. 2003), implying that the ideal combination may be low *hunger* - high *disinhibition*.

The satiety and hunger hormones of leptin and ghrelin play an important role in energy homeostasis. Any imbalances of these hormones consequently effect body weight regulation (Morioka et al. 2016). A small number of researchers have shown associations between these hormones and eating behaviours. Higher ghrelin concentrations are associated with higher *restraint* (Schur et al. 2008) and *hunger* scores (Langlois et al. 2011). While lower ghrelin (Blundell et al. 2008) but higher leptin concentrations (Benbaibeche et al. 2021) are associated with higher *disinhibition* (Blundell et al. 2008), suggesting that elevated appetite (higher ghrelin) does not necessarily lead to loss of control over food intake. It is suggested that individuals with elevated appetite (higher ghrelin) consciously increase their *restriction* of food intake as a counteractive measure to maintain body weight (Schur et al. 2008).

Table 2.1 *- Eating behaviours & associations with body composition, energy intake, hunger & satiety hormones: summary of studies*

Eating behaviours	Association with body composition	Association with macronutrient intake	Association with hunger & satiety hormones
Restraint	- BMI (McGuire et al. 2001) - Weight (McGuire et al. 2001) - WC (McGuire et al. 2001)	- Energy (McGuire et al. 2001, Provencher et al. 2003) - Fat (McGuire et al. 2001, Provencher et al. 2003) + Protein (McGuire et al. 2001) + Carbohydrates (McGuire et al. 2001)	+ Ghrelin (Schur et al. 2008) NS Leptin (Schur et al. 2008)
Flexible Restraint	+ Weight loss (McGuire et al. 2001) - BF (Provencher et al. 2003) - WC (McGuire et al. 2001, Provencher et al. 2003) - BMI (McGuire et al. 2001, Hays & Roberts 2008) - Weight (McGuire et al. 2001)	- Energy (Provencher et al. 2003) - Fat (McGuire et al. 2001) + Carbohydrates (McGuire et al. 2001)	NR
Rigid Control	+ BMI (McGuire et al. 2001, Provencher et al. 2003) + BF (Provencher et al. 2003) + WC (McGuire et al. 2001, Provencher et al. 2003) + VAT (Provencher et al. 2003) + SAT (Provencher et al. 2003) - WC (McGuire et al. 2001)	- Energy (McGuire et al. 2001) - Fat (McGuire et al. 2001) + Protein (McGuire et al. 2001) + Carbohydrates (McGuire et al. 2001)	NR
Disinhibition	+ BMI (Provencher et al. 2003, Bellisle et al. 2004, Dykes et al. 2004, French et al. 2014) + Weight (Dykes et al. 2004)	+ Energy (Provencher et al. 2003, Bellisle et al. 2004) + Fat (Provencher et al. 2003)	- Ghrelin (Blundell et al. 2008) + Leptin (Blundell et al. 2008, Benbaibeche et al. 2021)

	+ WC (Provencher et al. 2003, Dykes et al. 2004) + HC (Dykes et al. 2004) + WtHR (Dykes et al. 2004) + BF (Provencher et al. 2003) + VAT (Provencher et al. 2003) + SAT (Provencher et al. 2003)		
Habitual Disinhibition	+ Weight gain (Hays & Roberts 2008) + BMI (Hays & Roberts 2008)	+ Energy (Provencher et al. 2003, Bellisle et al. 2004) +Fat (Provencher et al. 2003)	NR
Situational Disinhibition	+ BMI (Hays & Roberts 2008)	+ Energy (Provencher et al. 2003) + Fat (Provencher et al. 2003)	NR
Emotional Disinhibition	+ Weight gain (Hays & Roberts 2008) + BMI (Hays & Roberts 2008)	NR	NR
Hunger	+ BMI (Provencher et al. 2003, Dykes et al. 2004, French et al. 2014) + Weight (Dykes et al. 2004) + WC (Dykes et al. 2004, Provencher et al. 2003) + HC (Dykes et al. 2004) + WtHR (Dykes et al. 2004) + BF (Provencher et al. 2003) + VAT (Provencher et al. 2003) + SAT (Provencher et al. 2003)	+ Energy (Provencher et al. 2003)	+ Ghrelin (Langlois et al. 2011) + Leptin (Blundell et al. 2008)
Internal Locus for Hunger	+ Weight gain (Hays & Roberts 2008)	+ Energy (Provencher et al. 2003)	NR
External Locus for	- BMI	+ Fat (Provencher et al. 2003)	NR

Hunger	(Hays & Roberts 2008)		
Eating behaviour combinations			
Low disinhibition - low restraint	- BMI (Dykes et al. 2004) - BF% (Dykes et al. 2004, Kruger et al. 2016)	NR	NR
High disinhibition - low restraint	+ BMI (Dykes et al. 2004)	NR	NR
High disinhibition - high hunger	+ BMI (Provencher et al. 2003)	NR	NR
High disinhibition - low hunger	NR	NR	NR

References	**Study population**	**Study design**	**Study aim**
(McGuire et al. 2001)	1044 Healthy adults	Cohort	Evaluate the associations between restraint, flexible- and rigid restraint and shifts in weight and behaviours over a three year period.
(Provencher et al. 2003)	596 Healthy adults	Cross-sectional	Investigate the relationship between dietary intake and anthropometrical variables with eating behaviours. To assess if obesity status and gender influence the identified associations.
(Schur et al. 2008)	24 Normal & overweight adults	Cross-sectional	Investigate possible associations between eating *restraint* and leptin, insulin, or ghrelin.
(Hays & Roberts 2008)	535 Healthy women	Cohort	Investigate the association of the subcategories of eating behaviours and BMI as well as weight gain over time.
(French et al. 2014)	233 Healthy adults	Cross-sectional	Compared eating behaviours with laboratory-based measures of food preference and -reward.
(Bellisle et al. 2004)	2509 Healthy adults	Cohort	Explore the varying levels of restraint, disinhibition, and hunger, among individuals with a range of BMIs.
(Dykes et al. 2004)	1470 Healthy women	Cross-sectional	Investigate the associations between the TFEQ and body weight and size. As well as assess the effect of the employment on body measurements.
(Blundell et al. 2008)	Obese & lean females	Cross-sectional	Investigate whether appetite peptides control

			eating behaviour traits.
(Benbaibeche et al. 2021)	190 Healthy adults	Cross-sectional	Assess eating behaviours in adults and measure changes in eating behaviours scores according to BMI category.
(Langlois et al. 2011)	308 Healthy adults	Cross-sectional	Assess the association of eating behaviours with ghrelin.
(Kruger et al. 2016)	116 Healthy women	Cross-sectional	Assess the associations between BF%, BMI and eating behaviours.

+ indicates a positive association; - indicates a negative association; NR = not reported; NS = not significant; (VAT) Visceral adipose tissue; (SAT) Subcutaneous adipose tissue; (WC) Waist circumference;(BMI) Body mass index; (HC) Hip circumference; (WtHR); Waist-to-height ratio; (BF); Body fat; (BF%) Body fat percentage.

Eating behaviours are often associated with disturbed eating attitudes (Keeler et al. 2015, Vinai et al. 2015, Linardon & Mitchell 2017), for example, those with high *restraint* and *disinhibition* are shown to have a higher risk of developing eating disorders (Allison & Heshka 1993). Eating disorder assessment is often not conducted in the obese individual (Rukavishnikov et al. 2021). A large cohort of overweight and obese individuals who sought medical weight loss intervention was shown to have high EAT-26 scores. The EAT-26 measuring tool does not diagnose eating disorders, but rather identifies eating attitudes that are related to eating disorders. The measuring tool involves a total score out of 26 and three sub-categories: *dieting* (preoccupation with being thinner and avoidance of fattening foods); *bulimia & food preoccupation* (thoughts about food and bulimia) and o*ral control* (self-control about food and social pressures to gain weight). A score of ≥ 20 denotes more disturbed eating attitudes. A small number of studies have explored these associations between eating behaviours and eating attitudes. Those diagnosed with binge eating disorder have been shown to have higher score of eating behaviours that result in overconsumption of food such as *disinhibition* and *hunger* (Vinai et al. 2015). Likewise, Keeler et al. showed that participants with high *disinhibition* also scored high on the EAT-26 questionnaire (≥ 20) (2015). Furthermore, individuals who show high cognitive *restraint* of food intake are more likely to have disturbed eating attitudes (Linardon & Mitchell 2017). Especially those with high *rigid control*- the strict all or nothing approach to dieting- which is linked with increased binge eating (Bryant et al. 2019).

Therefore, exploring eating attitudes related to eating disorders along with eating behaviours is of high importance and will create a clearer picture of factors that drive weight gain and obesity.

2.8 Chronotype-specific eating behaviours & attitudes

To date, the association between chronotype, eating behaviours (Schubert & Randler 2008, Lázár et al. 2012, Meule et al. 2012, Lai & Say 2013, Vera et al. 2018, Beaulieu et al. 2020), eating attitudes and related behaviours (Natale et al. 2008, Schmidt & Randler 2010, Harb et al. 2012, Muñoz et al. 2017) have largely been unexplored, especially by using the TFEQ (Schubert & Randler 2008) and EAT-26 (Harb et al. 2012, Muñoz et al. 2017). Exploring the difference between chronotypes in terms of eating behaviours is important as eating behaviours may alter energy intake by influencing the types and amounts of foods eaten, timing of food intake and where food intake occurs, ultimately affecting body weight (French et al. 2012). The studies that have utilised the TFEQ solely investigated the three main categories namely *disinhibition, restraint* and *hunger* (Schubert & Randler 2008) or the total TFEQ score (Beaulieu et al. 2020), while those who used the EAT-26 (Harb et al. 2012, Muñoz et al. 2017), only explored the total score of this test. Both of these questionnaires/tests consist of several subcategories that can be used to create a comprehensive eating behaviours (Stunkard & Messick 1985, Westenhoefer et al. 1999, Lesdéma et al. 2012) and eating attitude profile (Garner et al. 1982). The study by Schubert et al. have found that ET tend to display more unfavourable eating behaviours such

as higher perceived *hunger* and *disinhibition* (2008). On the other hand, MT seem to have better control over their eating behaviours and to have higher *restraint* and *flexible control* strategies (Schubert & Randler 2008), while another study found no association between chronotype score and TFEQ score (Beaulieu et al. 2020). From the two studies that investigated eating attitudes in chronotypes, the one study found a positive correlation between ET and a higher EAT-26 score (Harb et al. 2012), while the other found no differences in total score between MT and ET (Muñoz et al. 2017). Furthermore, the relationship between chronotype, eating behaviours, eating attitudes and body composition have been unexplored.

2.9 Conclusion

From this literature review it is evident that obesity aetiology is complex and stretches beyond excessive energy intake that is mismatched with energy expenditure (Taubes et al. 2013, Heymsfield & Wadden 2017, Schwartz et al. 2017). Rather, the aetiology includes various underlying factors including dietary factors such as eating patterns in terms of patterning (spacing, skipping and timing), and format (food combinations and nutrient content of meals) (Leech et al. 2015, Schwartz et al. 2017) and eating behaviours which all influence each other. Shedding further light on these dietary factors is the circadian timing system that plays a role in the timing of digestion, nutrient uptake and metabolism, metabolite and hormonal regulation, food intake, behaviour, and appetite (Bass 2012, Jiang & Turek 2017). Timing of food intake as well as composition of food intake (particularly macronutrients) is an important zeitgeber for the circadian timing system (Manoogian & Panda 2017). Furthermore, an individual's circadian preference, that is to say chronotype, may modulate their sleep wake cycles (Duffy et al. 2001) as well as eating patterns and eating behaviours.

Although the number of studies that has examined the relationship between chronotype, dietary intake, health and body composition outcomes are growing, key questions regarding the role that chronotype has on these outcomes have emerged. Such as, the question of whether optimal meal timing may differ among individuals in accordance with their chronotype (Muñoz et al. 2017, Xiao et al. 2019) or is it possible that food intake should simply be prioritised to "day" hours and limited during "night" hours, because ingested calories are better metabolised during the day (Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Covassin et al. 2016). Furthermore, is chronotype a determinant of eating patterns (both patterning and format of meals) as well as eating behaviours which ultimately affects body composition and metabolic health (Almoosawi et al. 2019).

A systematic review will be conducted to identify the research gaps as well as to answer one of the above outlined questions of whether chronotype indirectly impacts eating behaviours, dietary intake (timing, choice, nutrients), and biomarkers leading to body composition outcomes in healthy adults.

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3 Chapter 3: Methodology

Chapter 3 gives a brief description of the methodology followed in this thesis. This thesis utilised data from the cross-sectional study; PRedictors linking Obesity and gut MIcrobiomE (PROMIsE). This study was funded by the Health Research Council of New Zealand and conducted at Massey University between May 2016 until May 2017. In this method section the PROMIsE methods which are relevant to this PhD research are outlined. A more detailed description of the PROMIsE study is reported elsewhere (Kindleysides et al. 2019). Forming the foundation of this PhD, is a systematic review that provided vital background knowledge and identified the research gaps that guided the statistical analysis of the PROMIsE data. Furthermore, the data processing and analyses completed for this PhD research are discussed.

3.1 Systematic review - chronotype differences in body composition, dietary intake and eating behaviour outcomes

To fulfil objective one a systematic literature review that provided background knowledge and served as the foundation of this PhD research was completed. The systematic review further identified the research gaps that guided the statistical analysis of the PROMIsE data. The review is registered with PROSPERO (CRD42020219754) and reported in accordance with the Preferred Reporting Items (2009) for Systematic Reviews and Meta-Analyses (PRISMA) checklist. The aim of this first part of the thesis was to systematically determine whether chronotype indirectly impacts eating behaviours, dietary intake (timing, choice, nutrients), and biomarkers leading to body composition outcomes in healthy adults. The systematic review question was formulated using the participants, interventions, comparisons, outcomes and study design (PICOS) approach (Boland et al. 2017). The methodology and rationale of the published systematic review is further discussed in chapter four.

3.2 The PRedictors linking Obesity and the MIcrobiomE (PROMIsE)

3.2.1 Study design & aim

This was a single centre, cross-sectional study conducted at the Human Nutrition Unit at Massey University, Albany. The original objective of the PROMISE study was to characterise the gut microbiome in two populations (Pacific and NZ European women) with different metabolic disease risk and body weight profiles (normal and obese); and to further explore the association with dietary intake, physical activity, taste perception, eating behaviour, and sleep. This PhD research utilised selected data (sleep, anthropometry, body composition, biomarkers, and diet) from the PROMIsE study to explore the diet in-depth (eating patterns and eating behaviours) as it relates to different chronotypes and

metabolic health markers of NZ Europeans and Pacific women with different body fat profiles and varied metabolic disease risk factors (Kindleysides et al. 2019).

3.2.2 Study population & recruitment

The study aimed to recruit 272 post-menarche and premenopausal healthy women between the ages of 18 and 45 years – 136 of NZ European and 136 of Pacific ethnicity (established by self-determination and at least one parent of the same ethnicity). Participants were recruited from the wider Auckland region, either in-person, via the phone or online. NZ European women were mainly recruited through web-based advertisements via platforms such as work - and university email lists, Facebook community - and public figure pages (well-known NZ dietitians and nutritionists). Recruiting Pacific women proved to be more challenging as little interest for participation was generated from advertisements on Pacific Facebook pages and websites. Pacific women were mainly recruited in-person or via phone by a specially appointed Pacific nurse who was also available during all data collection procedures to support them. Furthermore, Pacific women were successfully recruited at cultural events and festivals and via University Pacific networks. Finally, two recruitment services were employed to aid in recruiting Pacific women with a BMI of < 25 kg/m^2. Initial screening of BMI, based on self-reported weight and height, was conducted either online, in-person or via the phone. Research staff utilised self-reported weight and height to calculate BMI to screen eligible participants to invite to participate in the study. Participants were excluded if they were pregnant or lactating; diagnosed with chronic illness (e.g., diabetes and cardiovascular disease); underwent bariatric surgery; had severe food allergies; used medication that could interfere with appetite or the immune system (e.g., appetite suppressants and corticosteroids); were current smokers; had severe dietary restrictions or avoidances (e.g., vegan) A total of 351 women were eligible to participate in the study, although 304 fully completed all the data collection aspects and met the inclusion criteria. Thus, the final recruited sample were 304 women of which 162 were of NZ European and 142 of Pacific ethnicity (**Figure 3.1**) (Kindleysides et al. 2019).

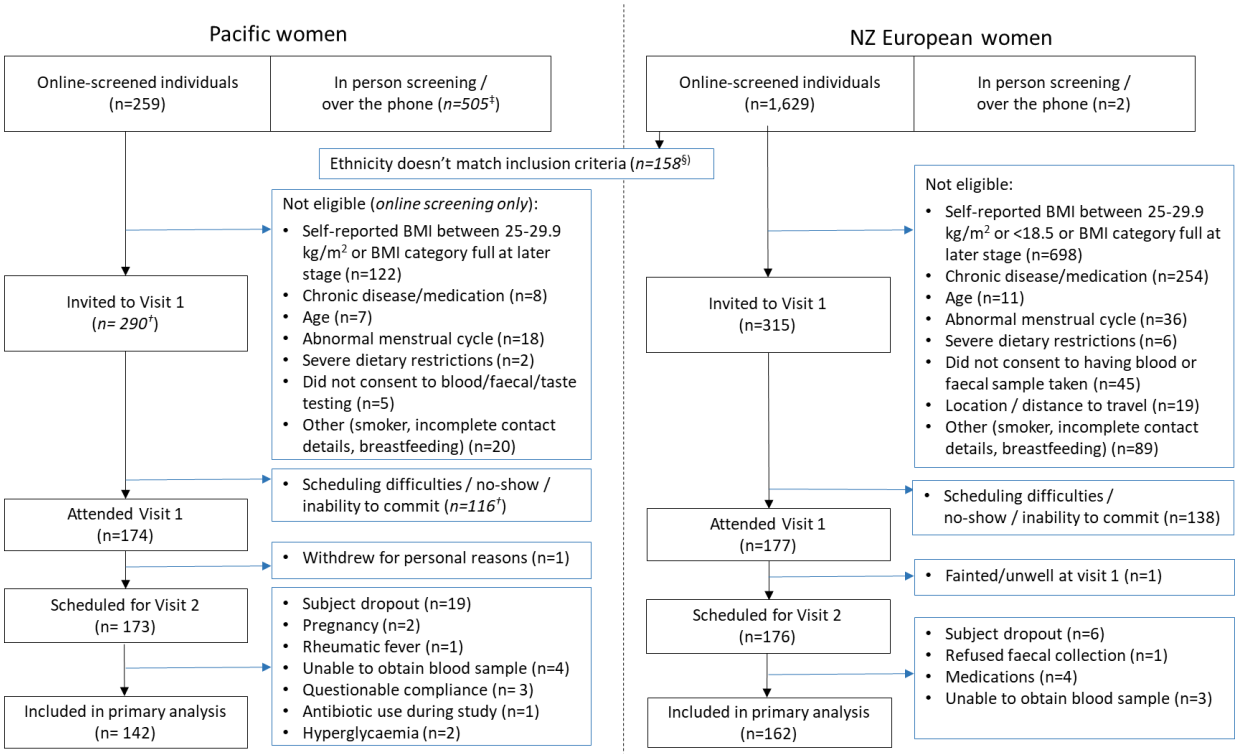

Figure 3.1 *- PROMIsE study recruitment process (Kindleysides et al. 2019) Reproducible under the terms of Creative Commons Attribution 4.0 license.*

3.2.3 Ethical considerations

The Southern Health and Disability Ethics Committee granted the PROMIsE study ethical approval (16/STH/32) (**Appendix A.1**). The study was conducted according to the guidelines of the declaration of Helsinki. The trial was registered at anzctr.org.au (ACTRN12618000432213). Participants were provided with detailed information regarding the study procedures and measures and had the opportunity to ask questions before commencing the study (see **Appendix A.2** for the information sheet). Informed and written consent was obtained from all participants (**Appendix A.3**). Data were restricted to the research team and each participant was allocated a unique study ID, which was used for all analyses to ensure participant data were deidentified (Kindleysides et al. 2019, Renall 2020).

3.2.4 Study procedure & data collected

The participants attended two visits as well as completed at home data collection between the study visits. The time in between study visits were usually no longer than 14 days and took place from May 2016 until May 2017. A Pacific nurse was present throughout the Pacific participant's first visit and addressed any questions and concerns. To minimise dropouts, Pacific participants were offered transport to and from the research unit. Data collected at each visit and general phenotype characteristics and deprivation index of study participants are shown in **Table 3.1**. See **Appendix A.4** for the complete list of data collected (Kindleysides et al. 2019).

Table 3.1 *- Data collected at each visit (Kindleysides et al. 2019). Reproducible under the terms of Creative Commons Attribution 4.0 license.*

Visit 1 (fasted) *Arrival time: 7:30 – 8:45 am*			**Visit 2 (non-fasted)**	
10 min	Informed consent, allocation of study identifier		5 min	Welcome and return of accelerometers, diet record and faecal samples
10 min	Anthropometric measurements (height, weight, bioelectrical impedance analysis body composition)	11 – 14 days at home data collection	10 min	Anthropometric measurements (weight, waist and hip circumference, bioelectrical impedance analysis body composition)
5- 10 min	Blood sample	**Diet record:** 5-day estimated non-consecutive food record. **Accelerometers:** hip and wrist mounted (7 consecutive days). Sleep and physical activity diary. **Faecal sample:** Collection after food record completion and stored in freezer at home (-20ºC) until visit 2.	5 min	Blood pressure
5- 10 min	Urine sample		Online questionnaire (monitored by researchers)	
25 min	Taste perception testing		25 min	Food frequency questionnaire (FFQ)
15- 20 min	Breakfast		12 min	Recent physical activity questionnaire
Online questionnaire (monitored by researchers)			5 min	Eating Attitudes test (EAT-26)
10 min	Dietary diversity questionnaire (DDQ)		25 – 30 min	One-on-one food record interview with New Zealand Registered Dietitian
10 min	Pittsburgh Sleep Quality Index, Munich Chronotype questionnaire, Berlin questionnaire Shift work questionnaire		10 – 15 min	Dual X-Ray Absorptiometry (DXA) whole body scan
10 min	Three factor Eating Questionnaire		3 min	Sagittal height measurement
3 min	Retrospective 7-day, sleep and work questionnaire			
10 min	Demographic interview (one-on-one)		3 min	Participant thanked for their participation and provided with voucher
10 min	Instructional video for diet record			
12 min	Participant provided with a home pack, personalized verbal instructions and written, at home data collection instructions			
Total: 2.5 hours			**Total: 2 hours**	

3.2.5 Demographic information & deprivation index

Demographic information, including health information (including information on menstruation, disease conditions, mediations, previous surgery, and supplement use), age, alcohol consumption, smoking habits, address, occupation, personal/household income, number of children, dietary

supplement use, birth weight and delivery method and work patterns was collected during a one-on-one interview with a researcher. The NZ Deprivation Index 2013 (NZDep2013) was used as a measure of socio-economic status. The NZDep2013 is determined by combining census information such as home ownership, housing, qualifications, employment, income, access to transport, communications, and family structure. The deprivation scores are grouped into deciles, where one represents the geographical area with the least deprived scores and 10 the areas with the most deprived scores (Crampton et al. 2020, Kindleysides et al. 2019, Renall 2020).

3.2.6 Anthropometrical measurements

All anthropometrics measurements were conducted according to the International Society for the Advancement of Kinanthropometry (ISAK) protocols by level one ISAK trained research staff (Stewart et al. 2011) and collected during study visit one and two. Height and weight were used in the Quetelet index (weight/height^2) to determine BMI (stratified as low < 25 kg/m^2 and high ≥ 25 kg/m^2). Waist- and hip circumferences were measured with a Lufkin W600PM flexible steel tape with the participant in a relaxed standing position with their arms folded across their chest. Waist-to-hip and waist-to-height ratio was determined by dividing waist circumference by hip circumference (cm) and height (cm), respectively. Dual-energy x-ray absorptiometry (DXA; Hologic QDR Discovery A, Hologic Inc with APEX V. 3.2 software) were used to assess lean body mass (kg), whole body total fat mass (kg) and percentage, android-, gynoid- and visceral fat (Kindleysides et al. 2019). Bioelectrical impedance analysis (InBody230) was used to determine muscle and body fat mass (kg) (Kindleysides et al. 2019).

3.2.7 Blood samples

Blood samples were collected to analyse the following biomarkers (e.g., plasma glucose, insulin, glycated haemoglobin, lipids, and liver function tests), inflammation markers (e.g., high-sensitivity C-reactive protein, interleukin 6 and tumour necrosis factor alpha), and endocrine regulators (e.g., GLP-1, ghrelin, leptin, and adiponectin). Fasted blood samples (10 -15 hours overnight) were collected between 07:30 and 09:00 during visit one. A phlebotomist applied a tourniquet to the arm of participants before performing a venipuncture. The blood was then drawn from the same arm into 4 vacutainers with a maximum volume of 30 ml. This was used to obtain plasma and serum for the analysis of metabolic markers and endocrine regulators. For whole blood, the sample was drawn into Ethylenediaminetetraacetic acid (EDTA) 10 mL vacutainers (Becton Dickinson), that was stored immediately in a −80° C freezer. The serum vacutainer (10 mL, Becton Dickinson) was kept a room temperature (18°C) for 30 - 60 minutes to allow for clotting before centrifugation. A plasma sample (2mL Becton Dickinson vacutainer P800 EDTA, aprotinin, and dipeptidyl peptidase IV) was collected to analyse endocrine regulators. All vacutainers were immediately placed on wet ice after collection until centrifugation. Within 1 hour of collection, all four vacutainer tubes were centrifuged at 1500 g

for 15 minutes at 4°C. Plasma and serum samples were frozen at -80° C until further analysis (Kindleysides et al. 2019).

Blood samples were analysed by the Liggins Institute, Auckland, New Zealand using the following standard procedures. Whole blood and HbA1c concentrations were measured by turbidimetric inhibition immunoassay method (Roche Diagnostic, Mannheim, Germany) on a Hitachi c311 auto-analyser (Hitachi High Technologies Corporation, Tokyo, Japan). While total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) (enzymatic colorimetric method) and serum glucose (enzymatic UV method) were measured using a Hitachi c311 auto-analyser (Hitachi High Technologies Corporation, Tokyo, Japan) with Roche Diagnostic reagents (Mannheim, Germany) for all analyses. Serum levels of insulin were measured on the Cobas e411 analyser (Hitachi High Technologies Corporation, Tokyo, Japan), using the electrochemiluminescence immunoassay method (Roche Diagnostics, Mannheim, Germany). Homeostasis model assessment (HOMA-IR) index for insulin resistance was calculated as fasting blood glucose [mmol/L] X fasting plasma insulin [μU/mL]/22.5 (Kindleysides et al. 2019).

3.2.8 Dietary intake

Dietary intake for energy (kJ), macro- and micronutrient intakes (g), and distribution of food intake throughout the day (clock times) were assessed by using 5-day food records (5DFR) that included two weekend days and three weekdays (**Appendix A.5**). Portion sizes were estimated, and participants received training for estimating and documenting portions (10-minute instructional video). Participants had the opportunity to ask questions regarding the recording process, which was answered by trained research staff. The food record was reviewed by a NZ registered dietitian before a one-on-one, in-depth discussion with the participant to clarify portions, cooking methods, brands of food products reported during visit two. Visual portion book aids, household measures (e.g., metric cups and spoons), and Web-based tools were provided to participants to confirm specific portion sizes and brands consumed.

The computer software, FoodWorks10 (Xyris Software (Australia) Pty Ltd, Queensland, Australia) was used to analyse the energy, macro- and micronutrient content of the 5DFR. The 5DFR information for each participant was entered into the software by PROMIsE researchers and NZ registered dietitians. A vast range of food items from the food composition tables were used. If an appropriate compositional match for an item was not found in the NZ FOODFiles 2016, a food item from the Xyris brand name database AusFoods 2017 (based on the Australian food composition databases AUSNUT 2011-13, developed by Food Standards Australia New Zealand) was selected. Recipes were handled in two ways. Firstly, if a participant was unable to recall the purchased food item (brand, store, etc.) or recall the recipe (ingredients, cooking methods, etc.) of a food item (e.g., tuna and rice salad) a standard recipe was created from the available food items listed in the databases. Secondly, if a direct compositional

match for a participant's recipe (e.g., vanilla cake) was available from the NZ FOODfiles2016, it was selected. If appropriate, cooked portions were entered into FoodWorks. For the raw weights of ingredients, the cooking factors from McCance and Widdowson's (Finglas et al. 2015), were used to convert raw to estimated cooked weights. Weekly meetings with the research team were held to discuss and resolve issues relating to data entry. A record detailing the food items reported in the 5DFR and the corresponding selected options within FoodWorks was created (Kindleysides et al. 2019).

Following, food items from the 5DFR were categorised into 66 food groups (g/day) based on similar nutritional composition and characteristics by previous PROMIsE researchers (**Appendix A.6**). For example, individual food items such as cream cheese was categorised in the "high fat cheese" group and green banana was categorised in the "starchy vegetable" group. Participant recipes were handled in three ways. Firstly, recipes that represented an individual food item were allocated to the appropriate food groups in their entirety (e.g., chocolate cake was allocated to the group "cakes and biscuits", and bliss balls were allocated to "sweet snacks"). Secondly, recipes that represented a mixed dish/beverage such as beef lasagne or tea with sugar and milk were allocated as individual food items to the corresponding food groups. For example, beef lasagne was handled as follows: the wheat pasta was allocated to the "refined grains", beef mince to "red meat", canned tomatoes to "tomatoes", and onion to "other-non starchy vegetables" groups. Another example is tea with milk and sugar. Earl grey tea was allocated to the "tea" group, full fat milk was allocated to the "full fat milk" group and white sugar to the group named "sugar added to food and beverages". However, if specific details such as weights of raw ingredients and cooking methods were not provided by the participant, a third method, where the participant recipe was allocated to the appropriate food groups in its entirety, was used. For example, the participant recipe, tuna and rice salad, was allocated to the "white meat mixed dishes" food group (Kindleysides et al. 2019).

This researcher (CvdM) revised all the 5DFR as well as allocation of food items to the food groups and corrected mistakes such as missing entries; double entries; incorrect portion sizes; missing mealtimes; and portions that were entered according to the imperial system were changed to the metric system. Furthermore, changes to the method of handling participant recipes that represented a meal or mixed dishes were made to allow for more distinction between food groups. For example, the following entry "tuna and rice" were entered into FoodWorks as a recipe. However, this recipe and others like it, contained full portions/exchanges of each individual food item and there was no need to enter it as a recipe into FoodWorks. Rather, the food items were entered separately and not as a recipe. This enabled the accurate allocation of food items into food groups. For example: fried rice and chicken were entered into FoodWorks as a recipe (by the previous researchers) and allocated to the "white meat mixed dishes" food group. However, this recipe contains "mixed vegetables", "chicken", and "white rice" which all equal full portions. Therefore, the "rice" and "mixed vegetables" and "chicken" were treated as separate

entries. After separating the three food items from the recipe, the “white rice”, “mixed vegetables” and “chicken” was allocated into the corresponding food groups namely: “white meat”, “other non-starchy vegetables”, and “refined grains”.

Dietary data were checked for plausibility of intakes. Participants were excluded if they under- or over reported energy intakes. The energy reporting cut offs of <2100 kJ/day and >27000 kJ/day were used to identify under- and over reporters - overreporting cut offs were extended similar to those previously used for ethnic minority populations (Kolonel et al. 2000, George et al. 2004, Willett 2012). From the 5DFR daily mean dietary intakes (energy, micro- and macronutrients, food groups) were determined for participants allocated to each chronotype group. The average hourly energy and macronutrient intakes for each chronotype group was also determined. Thereafter, four dietary windows were created (window 1: 03:00 – 09:59, window 2: 10:00 – 14:59, window 3: 15:00 – 19:59, window 4: 20:00 – 02:59) based on previous studies and mean intakes were determined for each window (Baron et al. 2011, Baron et al. 2013, Maukonen et al. 2017). The clock time for the first and last eating occasion of the day was determined for each chronotype. An eating occasion was defined as 210 kJ that was at least fifteen minutes apart from the next eating occasion. This energy caption excluded low energy beverage-only eating occasions such as tea with milk. The first eating occasion was defined as the first 210 kJ consumed from wake time and the last eating occasion was defined as the last 210 kJ consumed before sleep onset (Leech et al. 2015, Kant 2018). From this information the overnight fasting period was determined for each chronotype (**Appendix C.1**). The 66 food groups previously developed were condensed to 33 food groups including both healthy and discretionary food choices from all food groups to allow for dietary pattern analysis by principal component analysis (details reported in chapter 6).

3.2.9 Chronotype and shift work

The MCTQ is used to subjectively assess an individual’s chronotype (Roenneberg et al. 2003, Roenneberg et al. 2007). The shortened version of the MCTQ was utilised for this study (**Appendix A.7**). The questionnaire involved six items regarding subjective habitual bedtime (clock hour of going to bed), sleep latency (amount of minutes it takes to fall asleep; SLat), and habitual wake time (clock hour of wake time) on free days (work-free days) and workdays (scheduled workdays) from the preceding four weeks. From these 6 items, related parameters such as sleep onset (SO), sleep duration (SD), sleep latency (SL), midsleep (MS) and midsleep corrected for sleep debt (MSFsc) and social jetlag was calculated. The MSFsc is used as a proxy for chronotype. **Table 3.2** outlines the parameters calculated from the MCTQ.

Table 3.2 *- Munich Chronotype Questionnaire parameters (Roenneberg et al. 2003)*

Parameters	Description	Calculation
BT	Clock time of going to bed (hh:mm)	-
WT	Clock time of wake-up time (hh:mm)	-
SD	Sleep duration (SD) is the time range between sleep onset (SO; see below) and wake time (WT) (hh:mm)	WT minus SO *(for workdays and free days respectively)*
SL	Sleep Latency (SL) reflects the time it takes to fall asleep (hh:mm) after bedtime (BT).	SO minus BT *(for workdays and free days respectively)*
SO	Sleep onset (SO) is the clock hour of the onset of actual sleep (hh:mm).	BT plus SL *(for workdays and free days respectively)*
MS (MSW, MSF)	Midsleep (MS) is the clock time after half of the habitual sleep episode which occurs between habitual sleep onset (SO) and wake time (WT; hh:mm); MSW = midsleep for workdays; MSF = midsleep for free days)	(WT minus SO)/2 *for workdays (MSW) and free days (MSF) respectively*
Social jetlag	The time difference between habitual midsleep on free days (MSF) and workdays (MSW; hh:mm)	MSF minus MSW
MSFsc	MSFsc indicates midsleep time (in decimals) corrected for sleep duration on free days. A MSFsc of > 5 reflects a late chronotype (ET), while a MSFsc < 3 indicates an early chronotype (MT) and a score of 3 -5 indicates intermediate chronotypes (IT)	If SD on free days ≤ SD on workdays, then MSFc = MSF If SD on free days > SD on workdays, the MSFsc = MSF minus (SD on free days minus SD during the week*)/2

*(BT) bedtime, (WT) wake time, (SD) sleep duration, (SL) sleep latency, (SO) sleep onset, (MS) midsleep/midpoint of sleep, (MSFsc) midsleep corrected for sleep debt, (ET) evening type, (MT) morning type, (IT) intermediate types; *SD week = (SD x number of work days per week plus SD x number of free days per week) / 7*

A retrospective seven-day, sleep and work questionnaire (a week before the commencement of the study) was also used to collect information on sleep and wake times as well as work schedules. In addition, there was also a shift work questionnaire, which consisted of five items including the number of workdays per week; hours worked per day; description of work pattern; description of usual work pattern and information on number of night shifts. For the purpose of this PhD work, 17-night shift workers were excluded from the final analysed sample. Night shift workers exhibit altered sleep-wake and fasting-feeding cycles due to work obligations and not necessarily because of their chronotype and was therefore excluded from this PhD work. Information from the seven-day retrospective sleep and work questionnaire was used to confirm the self-reported wake and bedtimes in the MCTQ. For example, if a participant reported an improbable wake time of 13:30 on workdays,

the wake time reported in the retrospective sleep and work questionnaire was checked. If the wake time differed in the retrospective questionnaire, an average wake time was calculated from the retrospective questionnaire. The clock times reported in the MCTQ was further checked for AM and PM reporting mistakes (e.g., 7 AM reported as bedtime), which was compared against the retrospective questionnaire and corrected if necessary.

3.2.10 Eating behaviour

Eating behaviour was measured by using the three-factor eating questionnaire (TFEQ) *(with a total of 51 items)*. The questionnaire examines three eating behaviour domains, namely cognitive eating *restraint (21 items)* - which is the conscious restriction of food intake to control body weight and shape; *disinhibition* of control *(16 items)*- the loss of control of food intake that leads to overconsumption of food; and susceptibility to *hunger (14 items)*- feelings and subjective perceptions of hunger that leads to food intake (**Appendix A.8**). Responses were scored 0 or 1, with a higher score indicating higher levels of said eating behaviour (Stunkard & Messick 1985). *Restraint* and *disinhibition* were scored low (≤ 7) or high (>7) and *hunger* was scored as low (≤ 5) and high (> 5) (Lesdéma et al. 2012). Each of the three eating behaviour domains include sub-categories. The sub-categories of restraint include *flexible control (7 items)*- a non-obsessive and non-compulsive, neutral mindset to food intake control to regulate body weight; and *rigid control (7 items)* – a strict 'all or nothing' approach to control food intake to regulate body weight. Disinhibition consists of three subcategories namely *habitual disinhibition (5 items)* –particular circumstances and attitudes that habitually trigger disinhibition; *emotional disinhibition (3 items)* - overconsumption of food as a result of adverse mental state; and *situational disinhibition (5 items)* – environmental triggers (e.g., social events) that amplify lack of food control. Lastly, the two sub-categories of hunger include *internal locus for hunger (6 items)* – self-translated and controlled inner hunger and *external locus for hunger (6 items)* - derived from external triggers (e.g., buffets) rather that physiological cues (Westenhoefer et al. 1999).

Restraint and *disinhibition* were scored as low (≤ 7) or high (>7) and *hunger* was scored as low (≤ 5) and high (> 5) (Lesdéma et al. 2012). From this, four eating behaviour combination categories were created for levels of *restraint* and *disinhibition* ranging from *high restraint – low disinhibition* (the ideal eating behaviour combination) to *low restraint - high disinhibition* (least ideal eating behaviour combination). Four combination categories were also created for levels of hunger and disinhibition ranged from *low hunger – low disinhibition* (the ideal eating behaviour combination) to *high hunger - high disinhibition.*

3.2.11 Eating Attitudes

Eating attitudes related to eating disorders were assessed by using the Eating Attitudes 26 Test (EAT-26), which includes a total score out of 26; three sub-categories (**Appendix A.9**). The three sub-categories include: *dieting (13 items)* - preoccupation with being thinner and avoidance of fattening foods; *bulimia and food preoccupation* (6 items) - thoughts about food and bulimia (including binge eating); and *oral control (7 items)* - self-control about food and social pressures to gain weight. Responses are rated on a 6-point Likert scale and a score of ≥ 20 indicates disturbed eating attitudes with the three sub-categories creating a profile (Garner et al. 1982).

3.2.12 Statistical analysis

The following section gives an overview of the statistical analysis conducted for this thesis work. Specific details regarding statistical methods utilised for each of the chapters are reported in chapter 5 to 7 as part of the relevant chapter.

3.2.13 Data handling: descriptive statistics

Analyses were conducted using IBM SPSS Statistics for Windows version 28 (Armonk, NY: IBM Corp) and a p-value of < 0.05 was accepted as statistically significant. The distribution of the different variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests, boxplots, and histograms. Normally distributed data were presented as means and standard deviations, non-normally distributed data as medians and 25th and 75th percentiles and categorical data as frequency summary statistics. Log transformed data were back transformed to the original scale and reported as geometric mean [95% CI]. Welche's T-tests were used on normally distributed data and Mann-Whitney U tests on non-normal data for comparing two groups. When comparing more than two groups, ANOVA tests (with Tukey HSD as post-hoc tests) were used for normally distributed data and Kruskal - Wallis tests (with multiple comparison rank test, adjusted by Bonferroni correction) for non-normally distributed data.

The population was stratified by chronotype groups namely: ET (MSFsc >5), IT (MSFsc = 3 - 5) and MT (MSFsc <3). Chronotype groups (MT, IT and ET) were not further stratified by ethnicity as the number of participants in each chronotype groups was insufficient for analysis. For most analyses, MT and IT were also grouped together since there was (as expected) a much higher proportion among the (young) study sample of ET. **Appendix A.10** depicts descriptive statistics differences between ethnic groups.

Regression analyses were used to assess the association between chronotype groups (independent variable) and the dependent variables of interest (e.g., timing of dietary intake (chapter 5), dietary

pattern (chapter 6) or eating behaviours (chapter 7). The analyses were adjusted for age, ethnicity, and deprivation index (chapters 5 to 7). Regression models were not adjusted for sleep duration and regularity for the reason that the main focus of the analysis was not to investigate sleep-related eating behaviours. Subjectively reported sleep duration was indirectly taken into account by using the MSFsc derived from the MCTQ and is reported on page 351 of the thesis. Regression analyses were repeated within low and high body composition groups (BMI; normal <25 versus high ≥25 kg/m^2 and BF%; normal <35 % or high ≥35 %) (chapter 5 and 7) (Fitch & Bays 2022). Some participants did not estimate their self-reported height and weight correctly and as a result, a range of BMIs across categories were found following actual measurements. Therefore, participants were stratified into the BMI groups of low <25 and high ≥25 kg/m^2, thus capturing both overweight and obesity. Participants were also stratified into body fat percentage as normal <35 % or high ≥35 %), as defined by the Obesity Medicine Association (Fitch & Bays 2022). Android to gynoid percentage fat (AG) ratio was determined by diving android fat by gynoid fat percentage times 100 and stratified as normal <0.8 or high ≥ 0.8. Fat mass (FM) to fat free mass (FFM) measured by DXA was determined by dividing whole body total fat mass (kg) by lean body mass (kg). Furthermore, FM to FFM ratio (FM: FFM) was calculated by dividing FM by FFM (Chen et al. 2019, Gamboa-Gómez et al. 2019, Low et al. 2020, Seo et al. 2020). Pearson's correlation analysis was utilised to perform additional exploratory analyses to assess correlations between continuous MCTQ scores (MSFsc) and the variable of interest (e.g., biomarkers (chapter 5) or eating behaviours (chapter 7).

Principle component analysis (PCA) with varimax rotation was used to extract dietary patterns (chapter 6). The suitability of the data sets for PCA was determined with the Kaiser-Meyer-Olkin Measure of Sampling Adequacy and Bartlett's test of sphericity. The factors retained for the dietary patterns were based on the break-even point of the scree plot and Eigenvalues > 1. 0 (chapter 6).

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4 Chapter 4: Chronotype differences in body composition, dietary intake and eating behaviour outcomes – a scoping systematic review

van der Merwe, C., M., Münch, M. & Kruger, R. (2022). Chronotype Differences in Body Composition, Dietary Intake and Eating Behavior Outcomes - A Scoping Systematic Review. Advances in Nutrition. https://academic.oup.com/advances/article/13/6/2357/6679275

Abstract

The timing and nutritional composition of food intake are important zeitgebers for the biological clocks in humans. Thus, eating at an inappropriate time, e.g., during the night, may have a desynchronising effect on the biological clocks and in the long-term, may result in adverse health outcomes e.g., weight gain, obesity, and poor metabolic function. Being a very late or early chronotype not only determines preferred sleep- and wake-times but may also influence subsequent mealtimes which may impact the circadian timing system differently. In recent years, an increased number of studies have examined the relationship between chronotype and health outcomes, with a main focus on absolute food intake and metabolic markers and to a lesser extent on dietary intake distribution and eating behavior. Therefore, this scoping review aimed to systematically determine whether chronotype indirectly impacts eating behaviors, dietary intake (timing, choice, nutrients), and biomarkers leading to body composition outcomes in healthy adults. A systematic literature search on electronic databases (PubMed, CINAHL, MEDLINE, SCOPUS, Cochrane library) was performed (PROSPERO number: CRD42020219754). Only studies which included healthy adults (>18 years), classified according to chronotype and body composition profiles, using outcomes of dietary intake, eating behavior and/or metabolic biomarkers, were considered. Out of 4404 articles; 24 met the inclusion criteria. The results revealed that late (=evening types; ET) compared to early (=morning types; MT) chronotypes were more likely to be overweight/obese with poorer metabolic health. Both MT and ET had similar energy and macronutrient intakes, consuming food during their preferred sleep-wake timing, later for ET than MT. Most of the energy and macronutrient intakes were distributed towards nighttime for ET and were exacerbated by unhealthy eating behaviors and unfavorable dietary intakes. These findings from our systematic review give further insight why higher rates of overweight/obesity and unhealthier metabolic biomarkers are more likely to occur in ET.

Keywords: Morning type, evening type, circadian, meal timing, nutritional intake, eating habits.

Statement of significance: This scoping systematic review exemplifies differences in food choice, timing and distribution during the day, nutritional quality and eating behaviours, between chronotypes. This is the first systematic review which comprehensively compares not only dietary patterns, and food composition, but also eating behaviour and metabolic outcome markers between morning and evening types. Our findings highlight that it might be important for long-term metabolic health to include someone's chronotype when tailoring meal and food plans for healthy cohorts but also for patients.

Running title: Chronotype impact on body composition and diet.

4.1 Introduction

Most organisms, including humans have evolved an internal time keeping system that generates circadian rhythms of metabolism, gene expression and behaviours (Ko & Takahashi 2006, Bass 2010, Garaulet & Madrid 2010, Huang et al. 2011, Bass 2012). The circadian rhythms of clocks in each cell are controlled by the central clock located in the suprachiasmatic nuclei (SCN) in the hypothalamus of the brain (Moore 1972). In turn, the SCN is entrained to the earth's 24-hour light/dark cycle (King et al. 1997) as it receives external light input via the eyes and optic nerve and synchronises the downstream peripheral cell and tissue clocks (Czeisler et al. 1999, Dibner et al. 2010). Environmental light is the primary Zeitgeber (time cue) for the central circadian clock, however other external cues such as food intake, including the timing and composition of food intake is capable of setting the rhythms in the peripheral clocks as well as the clock-controlled genes in the body tissues and organs (Bass 2012, Youngstedt et al. 2016, Jiang & Turek 2017). These clock genes in turn influence the timing of digestion, nutrient uptake and metabolism, metabolite and hormonal regulation, food intake, behaviour, and appetite (Bass 2012, Jiang & Turek 2017). Timing of food intake as well as composition of food intake (particularly macronutrients) is therefore an important zeitgeber for the circadian timing system (Manoogian & Panda 2017).

Humans are physiologically suited to spend about two thirds of their 24-hour day awake, being active and eating and storing energy. They usually spend one third asleep, being in a fasting state at night-time (Buxton et al. 1997). During the day, ingested food provides energy to support metabolic processes whilst during the night, when sleep usually occurs, stored energy is mobilised to maintain homeostasis (Damiola et al. 2000, Cipolla-Neto et al. 2014). Thus, eating at an inappropriate time can have a desynchronising effect on the biological circadian clocks, resulting in adverse health outcomes including weight gain, obesity as well as poor metabolic health outcomes (Salgado-Delgado et al. 2010, Nelson & Chbeir 2018, Challet 2019). Studies not considering different chronotypes have shown that a higher energy intake during the biological night (the normal resting and fasting cycles), results in enhanced fat storage and ultimately obesity (Fong et al. 2017, McHill et al. 2017). It is further supported by McHill et al. who showed that obese individuals typically consume most of their energy an hour closer to the melatonin secretion onset time (circadian phase marker which usually occurs two to four hours before sleep onset) in comparison to lean individuals (2017). Additionally, eating later in the day is associated with an increased risk for type 2 Diabetes Mellitus (St-Onge et al. 2017), as well as metabolic alterations including impairment of lipid profiles, daily cortisol levels and glucose tolerance (Scheer 2010, Grimaldi et al. 2016, Morris et al 2016, Collado et al. 2018, Depner et al. 2018).

Evidence from shift work studies have further accentuated that incorrect timing of food intake in combination with other dietary factors such as poor food choices, eating behaviours as well as meal and snack frequency play a role in the adverse health outcomes seen in individuals (Morikawa et al. 2008,

Antunes et al. 2010, Roenneberg et al. 2013). An eating pattern that is high in energy dense foods such as sugar sweetened beverages, fast foods, and fatty foods and low in micronutrient-rich foods such as fruits, vegetables, and fibre, is associated with weight gain (Rouhani et al. 2016) and an increased risk of metabolic syndrome and diabetes (Wang et al. 2008, Esmaillzadeh & Azadbakht 2011). Furthermore, disinhibited, or restrained eating behaviours are known to affect energy intake by influencing the types and amounts of foods eaten, the timing of food intake and the eating occasion or where food intake occurs (French et al. 2012). This ultimately leads to increases in BMI and body fat percentage (Kruger et al. 2016) as well as subsequent detrimental metabolic health outcomes such as poor glycaemic control (Martyn-Nemeth et al. 2014). These findings can be explained by the various metabolic processes and hormones involved in energy expenditure that are governed in precise timed relationships with each other across a 24-hour day (Damiola et al. 2000, Cipolla-Neto et al. 2014).

This altered timing of food intake, poor food choices and behaviours are influenced by various other factors, such as work schedules, social events, but likely also by individual chronotypes. The term chronotype (Roenneberg et al. 2003) is widely used to describe the preferred sleep-wake timing of an individual relative to the light-dark cycle that influences the timing of their diurnal preferences and the modulation of physiological functions and behaviour. Intrinsic sleep-wake time preferences in humans can be classified as either early [morning - (=MT)], intermediate - (=IT)] or late [evening types (=ET)] chronotypes (Ehret 1974, Horne 1976, Adan et al 2012). The MT habitually prefer an early bedtime and early morning rise time (Ehret 1974, Horne 1976, Adan et al 2012). On the other hand, ET, prefer a later bedtime and a later morning rise time (Ehret 1974, Horne 1976, Adan et al 2012). Morning and evening types have also been shown to exhibit genetic differences in allele frequencies (Archer et al. 2003, Chang et al. 2019), and different intrinsic period length of the circadian clocks (Brown et al. 2008) as well as different phase angles of entrainment e.g., between circadian phase of the melatonin rhythms and sleep wake times (Duffy et al. 2001). Chronotype may therefore drive not only sleep and wake time (Duffy et al. 2001), but also the timing of food intake (fasting or eating).

Assessment of a person's chronotype can thus be used as a proxy for the phase of entrainment between the external 24-hour cycle and the internal circadian phase of sleep and wakefulness. Hence, some of the assessment instruments, (e.g., the Munich Chronotype Questionnaire; MCTQ), use mid-sleep as proxy for chronotype (which is the midpoint of the sleep episode after habitual sleep onset and wake up times on free and workdays). Such differences in sleep-wake timing consequently led to differences in food intake (Muñoz et al. 2017). However, not only the shift in mealtimes seem to be of different between chronotypes, but also nutrient and food choices, behaviours and consequently biomarkers may also be important (Schubert & Randler 2008, Meule et al. 2012, Muñoz et al. 2017). There appears to be a difference in inherent eating patterns displayed between MT and ET (Kanerva et al. 2012, Muñoz et al. 2017), although the number of studies is limited. One study for example has shown that normal

weight MT consume more energy earlier during the day, whilst normal weight ET consume food later during the day (Muñoz et al. 2017), while another study has found no association between chronotype and BMI (Kanerva et al. 2012). One study has found that ET have a poorer lipid profile in comparison with MT (Muñoz et al. 2017), but this has not yet been extensively studied in a healthy population. Furthermore, the ET tend to display unhealthy eating behaviours leading to less control over their dietary intake, that may favour a dietary pattern that results in weight gain and obesity (Schubert & Randler 2008, Meule et al. 2012), although the effect on body composition has not been explored.

A limited number of systematic reviews have been conducted regarding chronotype and diet (Almoosawi et al. 2019, Mazri et al. 2020, Lotti et al. 2021, Phoi et al. 2021). The majority of these reviews had a specific focus on disease conditions (Almoosawi et al. 2019, Lotti et al. 2021), or included unhealthy (type 2 diabetes mellitus) or specific populations (e.g., post-bariatric surgery), night shift workers (Mazri et al. 2020), or investigated eating patterns including behaviour related to temporal eating patterns (meal frequency and skipping) and energy intake (Phoi et al. 2021). The number of studies investigating the potential link between different chronotypes, and the diet have grown in the last 10 years. This systematic review identified as a gap that the associations with individual dietary aspects and health outcomes have not been explored extensively, nor does a comprehensive framework exist that presents the dietary components beyond energy intakes together with eating behaviours, as a whole. Therefore, the aim of this scoping systematic review was to systematically determine whether chronotype indirectly impacts eating behaviour, dietary intake (timing, choice, nutrients), and biomarkers leading to body composition outcomes in healthy adults.

4.2 Methods

4.2.1 Study design

This systematic review was designed, as a scoping review. It was conducted and reported according to the Preferred Reporting Items (2009) for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (see **Appendix B.1** for PRISMA checklist) (Moher et al. 2010). The main research question which was aimed to be answered was: “Is body composition, dietary intake, eating behaviour and biomarker outcomes in healthy adults dependent on chronotype?” The systematic review protocol was registered prospectively in the International Prospective Register of Systematic Reviews (PROSPERO) number: (CRD42020219754) and can be accessed at https://www.crd.york.ac.uk/PROSPERO/display_record.php?RecordID=219754.

4.2.2 Search strategy & eligibility criteria

A systematic literature search was conducted in May 2020 followed by a re-run in November - December 2020. The following electronic databases were searched: PubMed, CINAHL, MEDLINE,

SCOPUS, and Cochrane library. The search was limited to articles published in English, published within the last 10 years reflecting the surge of chronotype research, and including studies with participants older than 18 years. The search strategy was based on the following categorical keywords and their synonyms [adults], [chronotype], [body composition], [dietary intake], [eating behaviour] and [biomarkers] (see **Appendix B.2** for the full list of search terms). All relevant study designs except for conference proceedings, editorial letters, review articles and pharmacological studies were included. Studies that determined BMI and used this anthropometrical measurement as a comparator were included. Studies that recruited adults < 18 years; pregnant and lactating women, night shift workers (these individuals already exhibit altered sleep-wakefulness and fasting-feeding cycles due to work obligations and not necessarily because of their chronotype), and participants with diagnosed acute, pre-existing and chronic conditions that may influence sleep/wake timings (e.g., eating disorders, bariatric patients, mental illness, sleep disorders, diabetes, etc.) were excluded.

4.2.3 Study selection

In order to answer the research question (see above) the PICOS criteria, [Population Intervention, Comparison, Outcomes and Study] (Moher et al. 2010) were used from primarily retrieved publications:

1) *Population:* adults

2) *Intervention:* chronotype assessment

3) *Comparisons*: body composition measures including BMI and BF% categories, waist circumference and weight change

4) *Outcomes:* dietary intake, eating behaviour and biomarkers)

5) *Study design*: all relevant design except for conference proceedings, editorial letters, review articles and pharmacological studies; see **Appendix B.3**.

All records retrieved from the databases were exported using the Endnote X9 citation management software (Clarivate Analytics) (Team 2013). Duplicates were removed using Endnote and the remaining references were exported into Rayyan QCRI (Ouzzani et al. 2016). Two authors (CVDM, RK) independently screened the titles, abstracts and full text for eligibility using the PICOS criteria before final inclusion in the review (CVDM, RK). In the case of conflicting decisions, a third reviewer (MM) participated. Studies were included if participants were classified according to chronotypes, and their body composition profiles were compared. Studies were included if they reported at least one variable from the following four outcomes:

1) Dietary intake: i.e., diet composition: energy, macro- and micronutrients; food groups or food

and drink categories (e.g., fruits and vegetables, sugar, fiber, alcohol, starch, meat and dairy), and portion sizes.

2) Eating occasions: i.e., meal timing, frequency or skipping.

3) Eating behaviour: i.e., dietary restraint (conscious restriction of food intake to control body weight and shape), disinhibition (loss of control of food intake that leads to overconsumption), binge eating, perceived hunger.

4) Biomarkers: e.g., glucose, insulin, lipid profiles and blood pressure and genetic profiles (such as genotyping of the PERIOD3 clock gene (PER3)).

Studies were excluded if:

- they did not include at least one of the predefined outcomes;
- were not designed to compare body composition profiles; or
- did not analyse night shift workers separately from day workers.

Detailed reasons for exclusion of studies are reported in the Prefer Reporting Items for Systematic Reviews and Meta-Analyses and a PRISMA flow diagram outlines the study selection for this review (**Figure 4.1**) (Moher et al. 2010).

4.2.4 Data extraction

The following data were extracted by CVDM in table format with the following variables: authors, publication year, country, study design, number of participants, type of participants, age, body composition, method of chronotype classification, distribution of chronotype. The study had the following outcomes: dietary intake, eating behaviours and biomarkers. The information in the tables were organized into the specific outcome categories and then presented according to the differences between chronotypes. Additional analysis such as correlation analysis were also reported next to each outcome category. A second researcher (RK) reviewed the extracted data for accuracy by using the full text articles.

The quality of each study was assessed using the appropriate Joanna Briggs Institute (JBI) checklists for Analytical Cross-sectional studies, Cohort studies, and Randomized Controlled Trials (Moola et al. 2020). Each study was assessed independently by two authors (CVDM, RK) using the appropriate checklist for each study design assessing issues of bias, data collection, analysis and reporting. Studies were allocated a score according to the number of JBI checklist criteria that were met (Moola et al. 2020) (**Appendix B.4-6**).

Only statistically significant differences between chronotypes derived from the included articles and statistically significant associations (correlations), including significant linear relationships (*P*-T*rend* analyses) are reported in the text, but all *P*-values and additional analysis are reported in the tables. If the mean differences (absolute or in percentage), for example calories of energy intake per chronotype group, were not directly available for this systematic review, they were calculated based on information from the tables or figures in the original journal papers (marked with ‡ in the text).

4.3 Results

A total of 4404 articles were initially identified of which 339 were duplicates. After screening the remaining ones (title and abstract), 106 full text articles were assessed by applying the eligibility criteria. Finally, 24 full text articles were eligible for inclusion in this review. The main reasons for exclusion of studies were that either participants with acute, pre-existing and chronic diseases were included (n = 27) and/or incorrect intervention (n = 20), and/or incorrect publication type (n = 16) was used (Figure 4.1, based on pre-defined exclusion criteria).

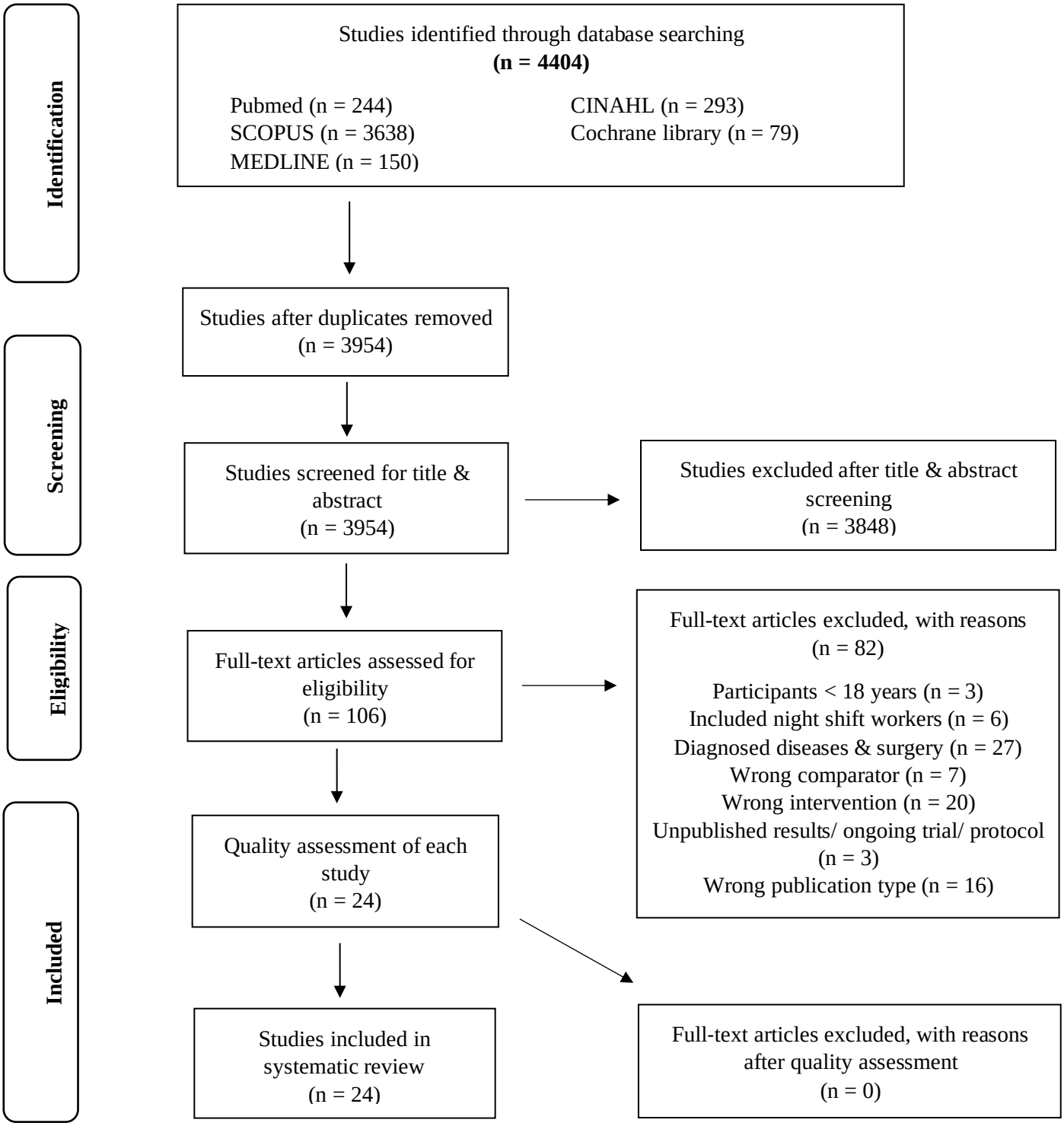

Figure 4.1 *- PRISMA 2009 flow diagram (adapted) illustrating the identification and selection process*

4.3.1 Study and participant characteristics

The sample size of the 24 studies varied between 44 and 3304 participants (men and women), of which three studies recruited women only (Sato-Mito et al. 2011, Muñoz et al. 2020, Zerón-Rugerio et al. 2020). Most of the included studies (n = 20) had a cross-sectional design (Baron et al. 2011, Sato-Mito

et al. 2011, Lázár et al. 2012, Lai & Say 2013, Baron et al. 2013, Culnan et al. 2013, Lucassen et al. 2013, Silva et al. 2016, Maukonen et al. 2016, Mota et al. 2016, Yoshizaki et al. 2018, Li et al. 2018, Teixeira et al. 2018, Vera et al. 2018, Zeron-Rugerio et al. 2019, Najem et al. 2020, Beaulieu et al. 2020, De Amicis et al. 2020, Muscogiuri et al. 2020, Zerón-Rugerio et al. 2020). The remaining studies included one randomised controlled trial (RCT) (Muñoz et al. 2020), two cohort studies (Maukonen et al. 2019, Xiao et al. 2019) and one population-based study (Maukonen et al. 2017) (**Table 4.1**).

The included studies assessed chronotypes using one of two validated questionnaires: the Morningness–Eveningness Questionnaire (MEQ) (Horne & Östberg 1976) or the Munich Chronotype Questionnaire (MCTQ) (Roenneberg et al. 2007). Some of the studies used a mixed methodology by calculating midsleep from rest-activity recordings or using sleep and wake timings (from sleep logs) to create MT and ET categories. Most of the studies (n = 18) included in this review determined chronotype using the MEQ (Lázár et al. 2012, Lai & Say 2013, Culnan et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Teixeira et al. 2018, Maukonen et al. 2019, Najem et al. 2020, Beaulieu et al. 2020, De Amicis et al. 2020, Muñoz et al. 2020, Muscogiuri et al. 2020, Zerón-Rugerio et al. 2020), one study used the MCTQ (Xiao et al. 2019), four studies used calculations of midpoint of sleep from rest-activity recordings (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Silva et al. 2016), and one used sleep and wake timings to create four categories (Zerón-Rugerio et al. 2020) (**Table 4.1**). Therefore, there was some heterogeneity among the classification of the different chronotypes.

Most studies (n = 15) used the MEQ cut-off values to classify chronotypes (Lázár et al. 2012, Lai & Say 2013, Culnan et al. 2013, Lucassen et al. 2013, Mota et al. 2016, Maukonen et al. 2017, Li et al. 2018, Teixeira et al. 2018, Maukonen et al. 2019, Zeron-Rugerio et al. 2019, Najem et al. 2020, Beaulieu et al. 2020, De Amicis et al. 2020, Muñoz et al. 2020, Muscogiuri et al. 2020). However, Xiao et al. (2019), and Vera et al. (2018), classified participants as ET and MT based on the median chronotype score instead of using the cut-off values from the MEQ (i.e., IT = scores between 42–58, MT = scores >58 or ET = scores <42) or the short version of the MEQ (see supplement) and the MCTQ (i.e., midsleep time < 3 = MT; midsleep time between 3 - 5 = IT; midsleep > 5 = ET). Two studies used tertiles of chronotype scores using the MEQ (Maukonen et al. 2016, Yoshizaki et al. 2018). For a detailed overview of the chronotype assessment methods used in each publication and thresholds of scores see **Appendix B.7** as well as **Table 4.1**.

Table 4.1 *- Study and participant characteristics*

Author, year, country	Study population N	Sex N (%)		Age (years)	Chronotype assessment method	Chronotype distribution N (%)		
		Women	Men			MT	IT	ET
(Xiao et al. 2019) United States of America	Middle-to-older-aged adults 872	443 (51)	429 (49)	MT: 62.3 ± 6.0 ET: 63.8 ± 5.7	MCTQ	436[a]	-	436[b]
(Sato-Mito et al. 2011) Japan	Dietetic students 3304	3304 (100)	-	Range: 18–20 18.1 ± 0.3	Midpoint of sleep quintiles	534 (16)[c]	2169 (66)[d-f]	601(18)[g]
(Vera et al. 2018) Spain	Overweight & obese adults 2126	1722 (81)	404 (19)	40.5 ± 12.4	MEQ	1098 (51.6)	-	1028 (48.4)
(Najem et al. 2020) Lebanon	Adult university students 644	453 (70.3)	190 (29.5)	20.2 ± 1.8	MEQ	56 (8.7)	452 (70.2)	132 (20.5)
(Lázár et al. 2012) United Kingdom	Healthy adults 675	262 (38.8)	413 (61.2)	Range: 20-35 26.1 ± 4.0	MEQ & the total score and the single question from the MCTQ referring to self-assessed chronotype	208 (31)	227 (34)	228 (34)
(Yoshizaki et al. 2018) Japan	Nurses 2559 Nurses: day workers 1095	1095 (100)	-	Range: 20 - 59 41.2 ± 9.4	MEQ	336 (31)[h]	359 (33)[i]	400 (37)[j]
(Silva et al. 2016) Brazil	University students 204	112 (55)	92 (45)	Range: 18 - 39 21.6 ± 3.9	Mid-sleep (MSFsc) midsleep corrected for sleep duration on free days	Not reported		

Author, year, country	Study population N	Sex N (%)		Age (years)	Chronotype assessment method	Chronotype distribution N (%)		
		Women	Men			MT	IT	ET
(Lai & Say 2013) Malaysia	Tertiary students 1118	632 (56.5)	486 (43.5)	Range: 18 - 27 20.1 ± 1.53	MEQ	-	2 (0.2)	1116 (99.8)
(Muñoz et al. 2020) Spain	Overweight & obese adults 200 Chrono-group: 102	102 (100)	-	Range: 18 - 65	MEQ	61 (60)	-	41 (40)
(Lucassen et al. 2013) United States of America	Obese men & pre-menopausal women 119	92 (77.3)	27 (22.7)	Range: 18 to 50	MEQ	80 (67)	-	39 (33)
(Mota et al. 2016) Brazil	Healthy university medical residents 72	52 (72.2)	20 (27.8)	29.2 ± 2.0	MEQ	26 (36)	36 (50)	10 (14)
(Zeron-Rugerio et al. 2019) Spain	University students 534	137 (25.7)	397 (74.3)	Range: 18 – 25 21.5 ± 3.0	MEQ	91 (17.0)	333 (62.4)	110 (20.6)
(Maukonen et al. 2019) Finland	Adults 1097	619 (56.4)	478 (43.6)	Range: 25 to 74	MEQ	552 (50.3)	433 (39.5)	112 (10.2)
(Maukonen et al. 2017) Finland	Adults 1854	1003 (54.1)	851 (45.9)	Range: 25 to 74 MT: 53.4 ± 0.4 IT: 48.4 ± 0.5 ET: 43.9 ± 0.9	MEQ	904 (49)	726 (39)	224 (12)
(Maukonen et al. 2016) Finland	Adults 4421	2408 (54.5)	2013 (45.5)	Range: 25 - 74	MEQ	1655 (37)	1529 (35)	1237 (28)

Author, year, country	Study population N	Sex N (%)		Age (years)	Chronotype assessment method	Chronotype distribution N (%)		
		Women	Men			MT	IT	ET
(Teixeira et al. 2018) Brazil	Undergraduate students 721	488 (67.7)	233 (32.3)	>18	MEQ	151 (21)	446 (62)	124 (17)
(Li et al. 2018) China	Undergraduate students 788	517 (65.6)	271 (34.4)	19.8 ± 1.1	MEQ	172 (21.8)	495 (62.8)	121 (15.45)
(De Amicis et al. 2020) Italy	Adults 416	289 (69.5)	127 (30.5)	50 ± 13	MEQ	135 (32.5)	243 (58.1)	38 (9.1)
(Culnan et al. 2013) United States of America	University undergraduates 135	79 (58)	56 (40.9)	18.25 ± 0.56	MEQ	7 (5)	65 (48)	64 (47)
(Baron et al. 2011) United States of America	Adult, volunteers 52	25 (48)	27 (52)	Range: 18–71 31 ± 12	Midpoint of sleep	-	28 (54)[k]	23 (44)[l]
(Baron et al. 2013) United States of America	Adults 52	25 (48)	27 (52)	Range: 18 – 71 31 ± 12	Midpoint of sleep	-	28 (54)[k]	23 (44)[l]
(Beaulieu et al. 2020) England	Adults 44	28 (63.6)	16 (36.4)	Range: 18 - 25	MEQ	22 (50)	-	22 (50)
(Muscogiuri et al. 2020) Italy, Naples	Middle-aged adults 172	123 (71.5)	49 (28.5)	51.8 ± 15.7	MEQ	100 (58.1)	50 (29.2)	22 (12.8)
(Zerón-Rugerio et al. 2020) Mexico	Undergraduate students 133	133 (100)	-	Range: 18-25	Median splits of the time in which each participant went to bed and woke up	34 (25.6)[m]	66 (49.6)[n,o]	33 (24.8)[p]

[1] ET, Evening type, IT, Intermediate Type, MCTQ, Munich Chronotype Questionnaire, MD, Mediterranean diet, MEQ, Morning-Eveningness Questionnaire, MT, Morning type, RCT, Randomised controlled trial.

[2] [a]Early sleep was defined as a chronotype earlier than the median (3:04 AM), [b]Late sleep was defined as a chronotype later than the median, [c]Based on earliest midpoint of sleep quintiles, [d]Based on midpoint of sleep quintiles 2, [e]Based on midpoint of sleep quintiles 3, [f]Based on midpoint of sleep quintiles 4, [g]Based on latest midpoint of sleep quintiles, [h]Based on MEQ score tertile 1: 34–53, [i]Based on MEQ score tertile 2: 54–59, [j]Based on MEQ score tertile 3: 60–76, [k]Based on normal sleep timing (midpoint 4:08 am), [l]Based on late sleep timing (midpoint of sleep 7:15 am), [m]Based on wakeup time <7:52 h and early-bedtime <23:48 h and defined as early-bedtime/early-rise (EE), [n] Based on early-bedtime (<23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as early-bedtime/late-rise (EL), [o] Based on late-bedtime (≥23:48 h) and wakeup time (<7:52 h) and defined as late-bedtime/early-rise (LE),[p] Based on late-bedtime (≥23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as late-bedtime/late-rise (LL).

4.3.2 Differences between chronotype & body composition or biomarkers

From the 24 included studies, 21 found significant differences between body composition outcomes and the different chronotypes (**Table 4.2**).

BMI, Weight & Height. In comparison with other chronotypes, two of the studies reported a higher BMI in ET (Xiao et al. 2019, Zeron-Rugerio et al. 2019); one study reported that ET vs MT women had greater increase in BMI (0.7 kg/m^2 vs. −0.1 kg/m^2, P = 0.024) (Maukonen et al. 2019); one study reported a linear relationship towards a higher BMI in ET (MT: 30.99 kg/m^2 vs ET: 31.3 kg/m^2 (Vera et al. 2018); and two studies reported a correlation between ET and a higher BMI, ranging between 26.0 - 32.6 kg/m^2 (Baron et al. 2011, Muscogiuri et al. 2020). Three studies showed that being an ET were associated with an increase in BMI points of a 0.50 – 1.2 kg/m^{2‡} (Culnan et al. 2013, Lucassen et al. 2013, Maukonen et al. 2019). In contrast to these findings, four studies reported a higher BMI in MT than other chronotypes (Maukonen et al. 2016, Li et al. 2018, Beaulieu et al. 2020, Zerón-Rugerio et al. 2020).

Weight loss/gain. Four studies reported on weight gain/loss over time between ET and MT (Mota et al. 2016, Maukonen et al. 2019, De Amicis et al. 2020, Muñoz et al. 2020), of which only one study by Maukonen et al. (2019) reported that ET vs MT women had a greater mean weight gain (+ 2.4 kg vs. + 0.3 kg, P = 0.016) over a seven-year follow-up period.

Biomarkers. Only four studies investigated chronotype differences/associations with biomarkers (Lázár et al. 2012, Lucassen et al. 2013, Vera et al. 2018, Muñoz et al. 2020) (Table 4.2). Investigating the differences in lipid profile and glucose homeostasis, Vera et al. (2018) found that ET compared with MT had higher concentrations of triglycerides (105 ± 1.79 vs 101 ± 1.71 mg/dL, P = 0.009), and lower high-density lipoprotein (HDL) (55.6 ± 0.48 vs 57.1 ± 0.46 mg/dL, P = 0.026). The ET had higher fasting blood insulin (7.62 ± 0.23 vs 7.40 ± 0.22 µUI/ml, P < 0.001) and Homeostatic Model Assessment – Insulin Resistance (HOMA-IR) scores (1.68 ± 0.06 vs 1.61 ± 0.05) than MT. Vera et al. (2018) also calculated the metabolic syndrome score, which was higher for ET vs MT (2.16 ± 0.04 vs 2.06 ± 0.04, P = 0.011). Lucassen et al. (2013) investigated resting heart rate, epinephrine and morning plasma adrenocorticotropic hormone concentrations and found this to be higher in ET (P = 0.007, P = 0.039, P = 0.019, respectively) compared to IT/MT.

Table 4.2 *- Differences between chronotype and body composition or biomarkers*

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
BMI, weight & height					
(Xiao et al. 2019)	Normal BMI: Women: 65.5% Men: 34.5% Overweight BMI: Women: 40.1% Men: 59.9% Obese BMI: Women: 52.8% Men: 47.2%	Not reported	Not reported	Overweight (3.1 ± 1.0 h) and obese (3.2 ± 1.1 h) participants had on average a later chronotype in comparison with those with a normal BMI (2.9 ± 1.0 h) [b]	$P < 0.007$
		Not reported	Not reported	Overweight (3.5 ± 0.9) / obese (3.6 ± 1.0) had a later midpoint of time in bed during weekends (3.6 ± 1.0) h in comparison with those with a normal BMI (3.3 ± 1.0) [b]	$P < 0.001$
(Sato-Mito et al. 2011)	Height: 158 ± 5.3 cm Weight: 52.2 ± 7.6 kg BMI: 20.9 ± 2.8 kg/m^2	MT[c] 21.2 ± 3.1 kg/m^2 Underweight:14.2% Normal: 77.3% Overweight: 8.4%	IT[d,e,f] 20.9 ± 2.8 kg/m^2 Underweight: 14.6% Normal: 78.9% Overweight: 6.6%[d] 20.8 ± 2.5 kg/m^2 Underweight: 13.8% Normal: 79.4% Overweight: 6.9%[e] 20.9 ± 2.7 kg/m^2 Underweight: 15.3% Normal: 77.8% Overweight: 6.9%[f]	ET [g] 20.9 ± 2.7 kg/m^2 Underweight: 14.0% Normal: 79.0% Overweight & Obese: 7.0%	*P-Trend* = 0.30
(Vera et al. 2018)	BMI: 31.3 ± 5.41 kg/m^2	40.0 ± 0.16 kg/m^2	Not Reported	31.3 ± 0.16 kg/m^2 ET showed a linear association towards higher BMI	$P = 0.16$ *P-Trend* = 0.02

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Najem et al. 2020)	BMI: 22.3 ± 3.61 kg/m^2 Range: 15.6 – 38.6 kg/m^2				*Other analysis:* No correlation (r = Not Reported0.025, *P* = 0.54) between BMI and ME scores
(Lázár et al. 2012)	BMI: 23.7 ± 2.8 kg/m^2 Range: BMI 18 – 30 kg/m^2	Not Reported	Not Reported	Not Reported	Not Reported
(Yoshizaki et al. 2018)	BMI day workers: 21.2 ± 2.7 kg/m^2	[h] 21.2 ± 2.7 kg/m^2	[i]21.2 ± 2.6 kg/m^2	[j]21.1 ± 2.8 kg/m^2	*P-Trend* = 0.33
(Silva et al. 2016)	BMI: 22.8 ± 3.2 kg/m^2 Overweight & obese: n = 47 (23%)	Not Reported	Not Reported	Not Reported	Not Reported
(Li et al. 2018)	BMI: Underweight: n = 270 Normal: n = 585 Overweight: n= 181 Obese: n = 82	Not Reported	Not Reported	Not Reported	*Other analysis:* BMI not correlated (r = 0.04; *P* = 0.15) with MES scores
(Muñoz et al. 2020)	Range: BMI > 25 kg/m^2 Chrono group: 30.37 ± 2.56 kg/m^2	BMI changes: -3.4 ± 1.0 kg/m^2	Not Reported	BMI changes: -2.9 ± 0.6 kg/m^2	*P* = 0.219
(Lucassen et al. 2013)	BMI: 38.5 ± 6.4 kg/m^2 BMI Range: 30 – 55 kg/m^2	38.2 ± 6.3 kg/m^2	Not Reported	39.1 ± 6.6 kg/m^2	*P* = 0.47 *Other analysis*: Scores towards ET were associated with an increase in BMI (*P* = 0.05, R^2 = 0.06) Effect size: 10-unit change in chronotype score was associated with a change of 1.2 kg/m^2 in BMI
(Mota et al. 2016)	BMI: 22.9 ± 3.4 kg/m^2 BMI ≥25 kg/m^2: 33.4%	Chronotype scores not associated with BMI (β - coefficient = −0.01)			*P* = 0.98

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Zeron-Rugerio et al. 2019)	BMI: n = 21.7 (3.1%) kg/m^2 Underweight: n = 54 (10.1%) Normal weight: n = 413 (77.3%) Overweight: n = 56 (10.5%) Obese: n = 11 (2.1%)	Not Reported	Not Reported	ET had a higher BMI (β - coefficient = -0.03)	$P = 0.04$
(Maukonen et al. 2019)	BMI: MT: 26.5 (0.2) kg/m^2 IT: 26.6 (0.2) kg/m^2 ET: 26.7 (0.4) kg/m^2	No increase in BMI over 7- year follow-up period	Not Reported	Mean increase in BMI over 7- year follow-up period: 0.4 kg/m^2	$P = 0.23$
		Proportion of subjects with BMI increases of ≥ 5% over the 7-year follow-up period: 22%	Not Reported	Higher proportion of subjects (33%) with BMI increases of ≥ 5% over the 7-year follow-up period	$P > 0.05$
		Obese at end of the follow-up: 17% of subjects	Not Reported	Obese at end of the follow-up: 26% of subjects	$P = 0.061$
		Increase in BMI of MT women: −0.1 kg/m^2	Not Reported	ET women had a greater increase in BMI (0.7 kg/m^2) that MT women	$P = 0.024$
(Maukonen et al. 2017)	Not Reported	27.1 ± 0.2 kg/m^2	26.7 ± 0.2 kg/m^2	27.6 ± 0.3 kg/m^2	$P = 0.44$
		Not associated with chronotype score			*P-Trend* = 0.66
(Maukonen et al. 2016)	BMI: MT: 27.2 (SE 0.13) kg/m^2 IT: 27.1 (SE 0.09) kg/m^2 ET: 26.9 (SE 0.16) kg/m^2	No difference in both genders			$P > 0.05$
		Chronotype score was positively associated with BMI in men MT were associated with a higher BMI in men (β - coefficient = 0.05)	Not Reported	Not Reported	$P = 0.04$
(Teixeira et al. 2018)	Not Reported	22.6 ± 3.2 kg/m^2	22.3 ± 3.8 kg/m^2	22.2 ± 3.6 kg/m^2	$P = 0.71$
		Overweight: n = 14.6 (22%)	Overweight: n = 18.8 (84%)	Overweight: n = 20.2 (25%)	*P = 0.41*

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Li et al. 2018)	Weight: Under: n = 158 (20.1%) Normal: n = 585 (74.2%) Over: n = 32 (4.1%) Obese: n = 13 (1.6%)	Not Reported	Not Reported	Not Reported	*Other analysis:* Positive correlation between chronotype and BMI. MT was associated with a higher BMI (r = 0.51, $P < 0.01$)
(De Amicis et al. 2020)	Not Reported	29.7 ± 5.6 kg/m^2	29.1 ± 6.1 kg/m^2	29.4 ± 6.1 kg/m^2	$P > 0.05$
(Culnan et al. 2013)	Weight - baseline: 139 ± 28.8 kg Weight - follow-up: 143 ± 29.5 kg BMI - baseline: 22.0 ± 3.26 kg/m^2 BMI - follow-up: 22.9 ± 3.41 kg/m^2	Baseline: Chronotype not associated with weight (unstandardized β = -1.70)			$P > 0.05$
		Baseline: Chronotype not associated with BMI (unstandardized β = - 0.26)			$P > 0.05$
		Not Reported	Not Reported	8-weeks follow-up: increase in BMI of 0.50 BMI points (Unstandardized β = 0.50, 95% CI: [0.04, 0.95])	$P = 0.03$
(Baron et al. 2011)	BMI: IT[k]: 23.7 ± 3.2 kg/m^2 ET[l]: 26.0 ± 6.9 kg/m^2	Not Reported	2 out of 27 IT[k] reported BMI ≥30	6 out of 22 ET[l] reported BMI ≥30	$P = 0.15$ *Other analysis:* BMI positively correlated with ET[l] ($p < 0.01$)
(Baron et al. 2013)	BMI: IT[k]: 23.7 ± 3.2 kg/m^2 ET[l]: 26.0 ± 6.9 kg/m^2	Not Reported	Not Reported	Not Reported	*Other analysis:* BMI moderately positive correlated with midpoint of sleep (r = 0.35, $P < 0.05$)
(Beaulieu et al. 2020)	BMI: 24.5 ± 3.2 kg/m^2	24.1 ± 2.7 kg/m^2	Not Reported	24.9 ± 3.6 kg/m^2	$P = 0.01$ *Other analysis:* Inverse relationship between MEQ score and BMI (ET showing a lower BMI r = −0.37, $P = 0.01$)
	Weight: 72.9 ± 11.4 kg	73.4 ± 10.3 kg	Not Reported	72.4 ± 12.7 kg	$P > 0.05$

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Muscogiuri et al. 2020)	BMI: (32.1 ± 6.3) kg/m^2 Normal 18 (10.5%) Overweight: 47 (27.3%) Obesity: class I: 58 (33.7%) class II: 29 (16.9%) class III: 20 (11.6)	31.4 ± 5.8 kg/m^2 Normal BMI: 10 (10.0%) Overweight BMI: 33 (33.0%) Obesity: class I: 32 (32.0%) class II: 15 (15.0%) class III:10 (10.0%)	33.1 ± 7.3 kg/m^2 Normal BMI: 7 (14.0%) Overweight BMI: 9 (18.0%) Obesity: class I: 15 (30.0%) class II: 11 (22.0%) class III: 8 (16.0%)	32.6 ± 5.5 kg/m^2 Normal BMI:1 (4.5%) Overweight BMI: 5 (22.7%) Obesity: class I:11 (50.0%) class II: 3 (13.6%) class III: 2 (9.1%)	$P = 0.27$ *Other analysis:* Chronotype was inversely correlated to BMI (r = −0.16, $P = 0.04$) MTs were associated with a lower BMI
	Weight	82.9 ± 19.0 kg	88.1 ± 20.6 kg	83.7 ± 12.5 kg	$P = 0.29$
(Zerón-Rugerio et al. 2020)	BMI: 23.7 ± 4.0 kg/m^2	25.4 ± 4.0 kg/m^{2m}	23.8 ± 4.5 kg/m^{2n} 23.0 ± 3.0 kg/m^{2o}	22.5 ± 3.8 kg/m^{2p}	*P = 0.02* *P-Trend* = 0.002
		Associated with increased BMI 2.3 kg/m^{2m}	Not Reported	Not Reported	$P < 0.05$
Body fat %, abdominal, visceral & subcutaneous adipose tissue					
(Vera et al. 2018)	BF%: 37.2 ± 6.71 %	BF%: 37.0 (0.19) %	Not Reported	BF%: 37.0 (0.19) %	$P = 0.85$ *P-Trend* = 0.54
(Muñoz et al. 2020)	Not Reported	BF% changes between baseline and end point: -4.2 ± 2.3	Not Reported	BF% changes between baseline and end point: - 3.2 ± 2.1	$P = 0.28$
(Maukonen et al. 2016)	Not Reported	BF%: 35.2 (0.23) %	BF%: 35.2 (0.15) %	BF%: 35.3 (0.24) %	$P = 0.92$
(Teixeira et al. 2018)	Not Reported	Inadequate abdominal fat: n = 17.9 (27 %)	Inadequate abdominal fat: n = 23.5 (105 %)	Inadequate abdominal fat: n = 25.8 (32 %)	$P = 0.24$
(De Amicis et al. 2020)	Not Reported	SAT: 2.6 ± 1.3 cm	SAT: 2.5 ± 1.1 cm	SAT: 2.5 ± 1.3 cm	$P > 0.05$
		VAT: 5.1 ± 2.3 cm Lower abdominal VAT for every 1 point of rMEQ score MT were associated with lower VAT −0.06 (-0.11, -0.01) cm	VAT: 5.1 ± 2.5 cm	VAT: 5.2 ± 2.9 cm	$P > 0.05$ $P < 0.05$

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Beaulieu et al. 2020)	Body fat: 27.7 ± 8.3 %	27.3 ± 8.4%	Not Reported	28.2 ± 8.4 %	$P > 0.05$
(Zerón-Rugerio et al. 2020)	Not Reported	Fat mass: 32.2 ± 7.4 %[m]	Fat mass: 31.5 ± 7.8 %[n] 30.5 ± 5.3 %[o]	Fat mass: 29.5 ± 6.4%[p]	$P = 0.39$ *P-Trend* = 0.08
Waist circumference					
(Silva et al. 2016)	Abdominal obesity: 31(15%)	Not Reported	Not Reported	Not Reported	Not Reported
(Muñoz et al. 2020)	Not Reported	Changes between end point and baseline: -9.8 ± 2.7 cm	Not Reported	Changes between end point and baseline: -8.8 ± 3.6 cm	$P = 0.44$
(Lucassen et al. 2013)	Not Reported	113 ± 13.6 cm	Not Reported	115 ± 11.5 cm	$P = 0.51$
(Mota et al. 2016)	WC >94 cm in males and >30 cm in females: 33.3%	Chronotype scores were not associated with WC (β-coefficient = 0.09)			$P = 0.41$
(Maukonen et al. 2019)	MT: 89.8 (SE 0.5) cm IT: 90.8 (SE 0.6) cm ET: 92.3 (SE 1.1) cm	Mean increase: 2.2 cm for both types over the 7-year follow-up period			$P = 1.00$
		Proportion of subjects whose WC increased with ≥ 5% over 7-year follow-up period: 33%	Not Reported	Proportion of subjects whose WC increased with ≥ 5% over 7-year follow-up period: 39%	$P > 0.05$
(Maukonen et al. 2016)	MT: 86 ± SE 0.42 cm IT: 86.5 ± SE 0.27 cm ET: 86.9 ± SE 0.43 cm	No difference in both genders			$p > 0.05$
(Teixeira et al. 2018)	Not Reported	78.3 ± 8.3 cm	79.0 ± 11.3 cm	79.0 ± 11.6 cm	$P = 0.75$
(De Amicis et al. 2020)	Not Reported	98.4 ± 13.2 cm WC decreases by -0.19 as rMEQ score increases MT associated with a lower WC	97.8 ± 14.5 cm	99.6 ± 13.5 cm	$P > 0.05$ $P < 0.01$
(Beaulieu et al. 2020)	84.3 ± 7.9 cm	84.2 ± 6.2	Not Reported	84.3 ± 7.9 cm	$P > 0.05$

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Muscogiuri et al. 2020)	Not Reported	103 ± 16.4 cm	103 ± 17.3 cm	105 ± 11.8 cm	*P* = 0.89 *Other analysis:* Chronotype not correlated with WC (r = −0.04, *P* = 0.57)
(Zerón-Rugerio et al. 2020)	Not Reported	98.4 ± 6.9 cm[m]	76.2 ± 9.7 cm[n] 74.9 ± 8.4 cm[o]	72.8 ± 7.4 cm[p]	*P = 0.06* *P-Trend = 0.01*
		Associated with increased WC of 5.2 cm[m]			*P-Trend* < 0.05
Hip circumference					
(Beaulieu et al. 2020)	98.4 ± 6.9 cm	99.2 ± 4.8 cm	Not Reported	97.6 ± 8.6 cm	*P* > 0.05
(Zerón-Rugerio et al. 2020)	Not Reported	99.5 ± 7.7 cm[m]	97.3 ± 10.7 cm[n] 96.3 ± 6.8 cm[o]	95.2 ± 7.3 cm[p]	*P* = 0.19 *P-Trend* = 0.03
Waist-to-hip ratio					
(Beaulieu et al. 2020)	0.86 ± 0.06	0.85 ± 0.07	Not Reported	0.86 ± 0.06	*P* > 0.05
Neck circumference					
(Lucassen et al. 2013)	Not Reported	38.8 ± 3.8 cm	Not Reported	39.6 ± 3.8 cm	*P* = 0.34 *Other analysis:* - Scores towards evening-ness was associated with a larger NC (*P* = 0.03) - Effect size: a 10-unit change in chronotype score was associated with a change of 0.6 cm in NC
Weight loss/gain					
(Muñoz et al. 2020)	Not Reported	Total weight loss: 10.2 ± 2.6 %	Not Reported	Total weight loss: 9.6 ± 1.8 %	*P* = 0.52
(Mota et al. 2016)	Not Reported	Not Reported	Not Reported	Chronotype scores (MT, IT, ET) not associated with weight gain	*P* = 0.48

Reference	Body composition distribution	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
				after the beginning of residency (β-coefficient = −0.10)	
(Maukonen et al. 2019)	Not Reported	Mean weight gain: 0.6 kg	Not Reported	Mean weight gain: 1.4 kg	$P = 0.35$
	Not Reported	Proportion of subjects who gained weight of ≥ 5% over the 7-year follow-up period: 22%	Not Reported	Proportion of subjects who gained weight of ≥ 5% over the 7-year follow-up period: 37%	$P > 0.05$
	Not Reported	Weight gain in MT women over the 7-year follow-up period: 0.3 kg	Not Reported	Weight gain in ET women over the 7-year follow-up period: 2.4 kg	$P = 0.02$
(Culnan et al. 2013)	Not Reported	Not Reported	Not Reported	8-w follow-up: weight gain of 2.35 pounds (1.07 kg) (Unstandardized β = 2.35 pounds 95% CI: [-1.62, 4.87])	$P = 0.07$

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
Biomarkers					
(Vera et al. 2018)	- Fasting glucose: glucose oxidase method - Triglycerides and HDL-c: Commercial kits - Arterial pressure: mercury sphygmomanometer. - MetS score: IDF criteria; summing MetS components - Fasting insulin: solid-phase, two-site chemiluminescent immunometric assay - Insulin resistance: (HOMA-IR; fasting glucose × fasting insulin/22.5) - Blood samples via standard procedures: DNA isolation and genotyping and GRS	- Triglycerides levels: 101 ± 1.71 mg/dL - Mets Scores: 2.06 ± 0.04 - HDL-c levels: 57.1 ± 0.46 mg/dl - Insulin levels: 7.40 ± 0.22 µUI/ml - HOMA-IR levels: 1.61 ± 0.05 Not Reported	Not Reported	- Triglycerides levels: 105 ± 1.79 mg/dL - Mets Scores: 2.16 ± 0.04 - HDL-c levels: 55.6 ± 0.48 mg/dl - Insulin levels: 7.62 ± 0.23 µUI/ml - HOMA-IR levels: 1.68 ± 0.06 - Higher evening genetic risk score	*P* = 0.01 *P* = 0.01 *P* = 0.03 *P-Trend* <0.001 *P-Trend* = 0.002 *P* = 0.04
(Lázár et al. 2012)	Genotyping of the PER3 VNTR was performed according to standard procedure	Frequency of $PER3^{5/5}$ genotype: 15.4%	Not Reported	Frequency of $PER3^{5/5}$ genotype: 7.5%	NA
		Genotype: effect on diurnal preference measured by MEQ (F2,619 = 4.43)			*P* = 0.01
		Genotype: marginal effect on diurnal preference measured by MCTQ			*P* = 0.06
		The main effect of genotype was significant for MEQ (F2,636 = 5.97)			*P* = 0.003
		The main effect of genotype was significant for the self-assessment question from the MCTQ (F2,642 = 4.12)			*P* = 0.02

Reference	Body composition distribution	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) & other analysis**
(Lucassen et al. 2013)		- 24h-urinary epinephrine concentrations 3 (2 -5) ug/24h	Not Reported	- 24h-urinary epinephrine concentrations: 4 (3 - 7) ug/24h; 0–30% higher	*P* = 0.04
		HDL-cholesterol: 48 (42–58) mg/dL	Not Reported	HDL-cholesterol: 49 (41–52) mg/dL	*P* = 0.51
		Resting heart rates: 68.4 ± 10.1 beats/min	Not Reported	Resting heart rates: 74.0 ± 10.1 beats/min	*P* = 0.01
		Plasma ACTH: 17 (12-24) pg/ml	Not Reported	Plasma ACTH: 21 (16 – 32) pg/ml	*P* = 0.02
		24h urinary norepinephrine: 39 (28 – 56) ug/24h	Not Reported	24h urinary norepinephrine: 45 (37 – 61) ug/24h	*P* = 0.05

[1] BMI, Body mass index, BMI, underweight <18.5 kg/m^2, BMI, normal <18.5 to <25 kg/m^2, BMI, Overweight & Obese ≥25 kg/m^2, FFM, fat free mass, HC, Hip circumference, NC, Neck circumference, OR [95% CI], P-Trend refers to the continuous association between the MEQ or MCTQ score and exposures of interest, SAT, Subcutaneous adipose tissue, %TWL, total body weight loss, Values reported as mean and SD unless stated otherwise, VAT, Visceral adipose tissue, WC, Waist circumference, MCTQ, Munich Chronotype Questionnaire, MEQ, Morning-Eveningness Questionnaire, MetS, Metabolic Syndrome, HDL-Cholesterol, High Density Lipoprotein – Cholesterol, ACTH, adrenocorticotropic hormone, HOMR-IR, Homeostatic Model Assessment – Insulin Resistance.

[2] [a]Early sleep was defined as a chronotype earlier than the median (3:04 AM), [b]Late sleep was defined as a chronotype later than the median, [c]Based on earliest midpoint of sleep quintiles, [d]Based on midpoint of sleep quintiles 2, [e]Based on midpoint of sleep quintiles 3, [f]Based on midpoint of sleep quintiles 4, [g]Based on latest midpoint of sleep quintiles, [h]Based on MEQ score tertile 1: 34–53, [i]Based on MEQ score tertile 2: 54–59, [j]Based on MEQ score tertile 3: 60–76, [k]Based on normal sleep timing (midpoint 4:08 am), [l]Based on late sleep timing (midpoint of sleep 7:15 am), [m]Based on wakeup time <7:52 h and early-bedtime <23:48 h and defined as early-bedtime/early-rise (EE), [n] Based on early-bedtime (<23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as early-bedtime/late-rise (EL), [o] Based on late-bedtime (≥23:48 h) and wakeup time (<7:52 h) and defined as late-bedtime/early-rise (LE),[p] Based on late-bedtime (≥23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as late-bedtime/late-rise (LL).

4.3.3 Differences between chronotype & dietary intake

Total daily energy. Fourteen studies reported total energy intake among chronotypes (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Teixeira et al. 2018, Vera et al. 2018, Maukonen et al. 2019, Xiao et al. 2019, Beaulieu et al. 2020, Muñoz et al. 2020, Zerón-Rugerio et al. 2020) (**Table 4.3**). Only one (Mota et al. 2016) found that chronotype scores (towards ET) were negatively associated with energy intake/day, thus ET consumed significantly more energy than MT ($P = 0.02$). However, across the other 13 studies, there were no differences in energy intakes between chronotypes (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Vera et al. 2018, Maukonen et al. 2019, Xiao et al. 2019, Beaulieu et al. 2020, Muñoz et al. 2020, Zerón-Rugerio et al. 2020). Furthermore, Teixeira et al. found that if ET also skipped breakfast, they would have a higher total energy intake per day (2018).

Energy distribution. Calculated across studies, ET consumed an overall mean intake of 6 – 90 kcal‡ less during the morning/at breakfast/by 10:00 (clock hour) (Baron et al. 2011, Maukonen et al. 2017, Maukonen et al. 2019, Zerón-Rugerio et al. 2020) and a total mean intake of 102 - 378 kcal‡ more energy during the evening/at dinner/ after 20:00, than MT (Baron et al. 2011, Lucassen et al. 2013, Maukonen et al. 2017, Maukonen et al. 2019, Zerón-Rugerio et al. 2020) (Table 4.3). Xiao et al. (2019) showed a significant linear association between energy distribution and being an ET and the likelihood of being overweight or obese. The MT consumed more energy in the morning (within two hours after waking up) and were less likely to be overweight or obese (OR: 0.32; 95% CI: 0.16, 0.66; $P = 0.0006$). The ET consumed more energy in the evening (within two hours before bedtime) and were 4.94 times more likely to be overweight or obese than MT (OR:4.94; 95% CI: 1.61, 15.1; *P-Trend* = 0.01) (Xiao et al. 2019).

Total daily carbohydrate, protein & fat intake. Eight out of (Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2017, Yoshizaki et al. 2018, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019, Muñoz et al. 2020) of 11 studies (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Teixeira et al. 2018, Vera et al. 2018, Muñoz et al. 2020) reported macronutrient intakes, however found no differences/associations between chronotypes. Three studies (Sato-Mito et al. 2011, Maukonen et al. 2017, Vera et al. 2018), reported significantly higher carbohydrate intakes in MT, (230 g/day‡) compared with ET (217 g/day‡), two studies (Mota et al. 2016, Teixeira et al. 2018) reported that ET had a higher carbohydrate intake, and one reported this was only found in ET that also skipped breakfast regularly ($p < 0.05$) (Teixeira et al. 2018). Maukonen et al. (2017) also found that sucrose intakes increased for ET on weekdays ($P = 0.02$) in comparison with other chronotypes.

Two studies reported that MT had a significantly higher daily intake of 4 g/day‡ of total protein (Sato-Mito et al. 2011, Mota et al. 2016) than IT and ET. Another study reported a higher intake of 3 g/day only over the weekend in MT in comparison with IT and ET (Maukonen et al. 2017).

Two studies reported that ET vs MT were significantly associated with a higher total fat intake of 1 g/day‡ (Sato-Mito et al. 2011, Maukonen et al. 2016), although Maukonen et al. (2016) reported this linear association in ET women (vs MT and IT women) only (31 E% vs 30 E%; *P-Trend* = 0.018). Teixeira et al. (2018) reported a higher total fat intake in ET who regularly skip breakfast, whilst Maukonen et al. (2017) reported an inverse relationship between total fat intake over weekends and ET in comparison with MT and IT ($P < 0.05$).

Daily carbohydrate, protein & fat distribution. Only three studies investigated the distribution of macronutrient intakes between chronotypes throughout the day (Baron et al. 2013, Maukonen et al. 2017, Xiao et al. 2019). Both carbohydrate and protein intakes after 20:00 were higher in ET than in MT (Baron et al. 2013, Maukonen et al. 2017). Maukonen et al. (2017) found that ET consumed more fat after 20:00 on both weekdays (10 g more‡) and weekends (19 g‡ more) compared with other types. They also showed that ET compared with other types consumed more saturated fatty acids (SFA) on both weekdays (3.4 g‡) and weekends (8 g‡) after 20:00 (Maukonen et al. 2017). Similarly, Baron et al. (2013) found that being ET vs IT were associated with a higher fat intake (14 g‡) after 20:00 (Baron et al. 2013). Interestingly in the morning, ET vs other types consumed 3.5 g‡ less fat on weekends by 10:00 (Maukonen et al. 2017) and 1 g less‡ fat in the four hours before habitual bedtime (Baron et al. 2013).

Xiao et al. (2019) also demonstrated that MT that consumed more carbohydrates and protein during the morning (within two hours after getting out of bed), were 80 % (*P-Trend* < 0.0001) and 61 % (*P-Trend* = 0.03) less likely to be overweight or obese than ET (Xiao et al. 2019). Conversely, ET that consumed more carbohydrates and protein in the evening (two hours before bedtime) were respectively 4.5 (OR: 4.48; CI 95%: 1.64, 12.2; *P-Trend* = 0.009) and 3.7 times (OR: 3.74; CI 95%: 1.33, 10.5; *P-Trend* = 0.02) more likely to be overweight or obese (Xiao et al. 2019) (**Table 4.3**).

Total daily micronutrient intake. Only one study by Sato-Mito et al. (2011) reported on micronutrient intakes. Being ET was associated with significantly lower potassium, calcium, magnesium, iron, zinc, vitamin A, thiamine, riboflavin, pyridoxine, folate, and vitamin D intakes compared with MT (*P-Trend* < 0.05 **Table 4.3**).

Total daily food group intake. Thirteen of the included studies reported on total food group intakes (Baron et al. 2011, Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Silva et al. 2016, Maukonen et al. 2016, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Vera et al. 2018, Najem et al. 2020, Muscogiuri et al. 2020). The ET consumed larger quantities of

energy-dense foods such as confectionery and sweets (Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Muscogiuri et al. 2020), sugar-sweetened beverages (Baron et al. 2011, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020), butter, cream, margarine (Muscogiuri et al. 2020), cholesterol-rich foods (Mota et al. 2016), meat (Sato-Mito et al. 2011, Silva et al. 2016, Muscogiuri et al. 2020), fast foods (Baron et al. 2011), caffeine (Lázár et al. 2012, Najem et al. 2020) and alcohol (Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Vera et al. 2018) in comparison with other chronotypes. Four studies reported that ET consumed fewer healthy foods such as, fish (6 g/day less, *P-Trend* < 0.05) (Maukonen et al. 2017), cereals (Maukonen et al. 2016, Vera et al. 2018) and vegetables (17g/1000 kcal‡, $P < 0.001$) (Sato-Mito et al. 2011) in comparison with other chronotypes. Similarly, Baron et al. (2011) reported that ET consumed only 1.9 servings/week of fruit and vegetables compared with 3.4 servings/week in IT (Baron et al. 2011). The MT consumed more nutrient-dense foods such rice and potatoes (Sato-Mito et al. 2011, Yoshizaki et al. 2018), fibre (Maukonen et al. 2017), vegetables (Sato-Mito et al. 2011, Yoshizaki et al. 2018), pulses (Sato-Mito et al. 2011), eggs (Sato-Mito et al. 2011), dairy (Sato-Mito et al. 2011), fruits, algae (*P-Trend* < 0.05) (Yoshizaki et al. 2018), and wine (Muscogiuri et al. 2020) than ET.

Table 4.3 *- Differences between chronotype & dietary intake*

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) and other analysis
		Morning type	Intermediate type	Evening type	
Total daily energy intake					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	2114.5 ± 634 kcal/d[a]	Not Reported	2147.4 ± 588 kcal/d[b]	*P-Trend* = 0.33
(Sato-Mito et al. 2011)	Dietary history questionnaire	1836 ± 20 kcal/d[c]	1776 ± 16 kcal/d[d] 1803 ± 17 kcal/d[e] 1814 ± 17 kcal/d[f]	1768 ± 18 kcal/d[g]	*P-Trend* = 0.10
(Vera et al. 2018)	Single 24-hour recalls	1972.8 ± 23.8 kcal/d	Not Reported	1918.6 ± 24.68 kcal/d	*P* = 0.12 *P-Trend* = 0.94
(Yoshizaki et al. 2018)	A semi-quantitative food frequency questionnaire	1854 ± 29 kcal/d[h]	1853 ± 27 kcal/d[i]	1825 ± 26 kcal/d[j]	*P-Trend* = 0.47
(Lucassen et al. 2013)	Three-day food recall diary	Working day: (2129 ± 631) kcal Non-working day: (2383 ± 928) kcal	Not Reported	Working day: (2276 ± 815) kcal Non-working day: (2378 ± 883) kcal	*P* = 0.37 *P* = 0.92
(Mota et al. 2016)	3- day self-administered food diary	Not Reported	Not Reported	Chronotype scores (towards ET) were negatively associated with daily energy intake; kcal/kg/day ET were associated with higher intake (β coefficient = −0.28)	*P* = 0.02
(Maukonen et al. 2019)	48-h dietary recalls over two previous consecutive days	7709 (SEM 97) kJ	Not Reported	7679 (SEM 215) kJ	*P* = 1.00
(Maukonen et al. 2017)	48-h dietary recalls	7808 (SEM 170) kJ on weekdays	7960 (SEM 171) kJ on weekdays	7881 (SEM 210) kJ on weekdays	*P* = 1.00
		7841 (SEM 283) kJ on weekends	7871 (SEM 283) kJ on weekends	7992 (SEM 367) kJ on weekends	*P* = 1.00

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Maukonen et al. 2016)	Food frequency questionnaire; Baltic Sea diet score	Men: 11597 (SEM 130) kJ/d Women: 9489 (SEM 103) kJ/d	Men: 11676 (SEM 90) kJ/d Women: 9433 (SEM 64) kJ/d	Men: 11776 (SE 159) kJ/d Women: 9389 (SE 105) kJ/d	*P-Trend* = 0.43 *P-Trend* = 0.54
(Teixeira et al. 2018)	24-hour recall	1552.8 [1233.4 – 2090.6] kcal/d	1734.2 [1356.3 – 2218.3] kcal/d	1692.9 [1333.8 – 2197.9] kcal/d	$P = 0.07$
		Not Reported	Not Reported	Breakfast skippers were negatively associated with energy intake (kcal/day) ET breakfast skippers had higher intake β = −0.25	$P < 0.001$
(Baron et al. 2011)	7-day food logs	Not Reported	1905 ± 526 kcal/d[k]	2153 ± 524 kcal/day[l] 248 kcal/d[d]	$P = 0.10$
(Baron et al. 2013)	7-d food logs	Not Reported	1905 ± 526 kcal/d[k]	2153 ± 524 kcal/d[l]	$p > 0.05$
(Beaulieu et al. 2020)	24-hour dietary record tool (myfood24, Leeds, UK)	1843 ± 681 kcal/d	Not Reported	1737 ± 659 kcal/d	$p > 0.05$
(Zerón-Rugerio et al. 2020)	6-d food logs	1517 ± 404 kcal/d[m]	1596 ± 425 kcal/d[n] 1555 ± 412 kcal/d[o]	1676 ± 420 kcal/d[p]	$P = 0.45$
Total daily carbohydrate intake					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	Carbohydrate: 240.5 ± 79.0 g/d[a]	Not Reported	Carbohydrate: 244.9 ± 72.3 g/d[b]	*P-Trend* = 0.27
		Sugar: 103 ± 46.6 g/d[a]		Sugar: 109 ± 43.9 g/d[b]	*P-Trend* = 0.02
		Fiber:19.9 ± 8.1 g/d[a]		Fiber: 19.8 ± 7.7 g/d[b]	*P-Trend* = 0.96
(Sato-Mito et al. 2011)	Dietary history questionnaire	56.3 ± 0.3 E% [c]	55.9 ± 0.2 E%[d] 55.5 ± 0.3 E%[e]	55.1 ± 0.3 E%[g]	*P-Trend* < 0.01

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
			55.4 ± 0.2 E%[f]		
(Vera et al. 2018)	Single 24-hour recalls	205 ± 3.07 g/d	Not Reported	194 ± 3.18 g/d	*P* = 0.02 *P-Trend* = 0.67
(Yoshizaki et al. 2018)	A semi-quantitative food frequency questionnaire	235 ± 4.30 g/d[h]	237 ± 4.0 g/[i]	230 ± 3.9 g/d[j]	*P-Trend* = 0.50
(Lucassen et al. 2013)	Three-day food recall diary	No significant differences in total intakes before and after 20:00			*P* = 0.84
(Mota et al. 2016)	3- day self-administered food diary	Not Reported	Not Reported	Chronotype score were negatively associated with carbohydrate (g/kg/d) ET had a higher intake (β = -0.26)	*P* = 0.03
(Maukonen et al. 2017)	48-hour dietary recalls	Weekdays: 48.6 (0.6) E%	Weekdays: 48.1 (0.6) E%	Weekdays: 48.8 (0.7) E%	*P* = 1.00
		Weekends: 49.6 (0.8) E%	Weekends: 48.8 (0.8) E%	Weekends: 47.8 (1.0) E%	*P* = 0.09
		ME score was positively associated with carbohydrate intakes on weekends; MT was associated with higher intake on weekends	Not Reported	Not Reported	*P-Trend* = 0.04
		Fiber: 2.5 (0.1) E% on weekdays	Fiber: 2.4 (0.1) E% on weekdays	Fiber: 2.5 (0.1) E% on weekdays	*P* = 1.00
		Fiber: 2.5 (0.1) E% on weekends	Fiber: 2.4 (0.1) E% on weekends	Fiber: 2.4 (0.1) E% on weekends	*P* = 1.00
		Fiber: ME score was positively associated with fiber intakes on weekends;	Not Reported	Not Reported	*P-Trend* = 0.04

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
		MT was associated with higher intake			
		Sucrose: 9.5 (0.4) E% on weekdays	Sucrose: 9.4 (0.4) E% on weekdays	Sucrose: 10.1 (0.5) E% on weekdays	$P = 0.46$
		Not Reported	Not Reported	Sucrose: Intakes increased with lower ME scores (ET) on weekdays	*P-Trend* = 0.02
		Sucrose: 10.3 (0.5) E% on weekends	Sucrose: 10.0 (0.5) E% on weekends	Sucrose: 9.7 (0.7) E% on weekends	$P = 0.91$
(Teixeira et al. 2018)	24-hour food recall	Carbohydrate: 198.6 [155.6 – 275.1] g/d	Carbohydrate: 226.4 [169.2 – 295.5]	Carbohydrate: 225.3 [169.9 – 293.2]	$P = 0.10$
		Not Reported	Not Reported	Breakfast skippers were negatively associated with carbohydrate intake (g/d) ET breakfast skippers had higher intake (β-coefficient = -0.19)	$P < 0.05$
		Fiber: 16.0 [10.2 – 21.8] g/d	Fiber: 15.8 [10.9 – 22.1] g/d	Fiber: 15.6 [10.6 – 21.1] g/d	$P = 0.93$
(Baron et al. 2011)	7-day food logs	Not Reported	49 ± 7.9 E%[k]	49 ± 7.8 E%[l]	$P > 0.05$
(Baron et al. 2013)	7-day food logs	Not Reported	237 ± 81 g/d [k] 49±7.9 E%[k]	260 ± 72 g/d[l] 49±7.8 E%[l]	$P > 0.05$
Total daily protein intake					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	87.4 ± 27.1 g/d[a]	Not Reported	87.7 ± 27.6 g/d[b]	*P-Trend* = 0.97
(Sato-Mito et al. 2011)	Dietary history questionnaire	13.5 ± 0.1 E%[c]	13.6 ± 0.1 E%[d] 13.5 ± 0.1 E%[e] 13.3 ± 0.1 E%[f]	13.2 ± 0.1 E%[g]	*P-Trend* <0.01
(Vera et al. 2018)	Single 24-hour recalls	83.01 ± 1.11 g/d	Not Reported	82.34 ± 1.15 g/d	$P = 0.68$ *P-Trend* = 0.94

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Yoshizaki et al. 2018)	A semi-quantitative food frequency questionnaire	66.0 ± 1.2 g/d[h]	64.1 ± 1.1 g/d[i]	63.4 ± 1.0 g/d[j]	*P-Trend* = 0.08
(Lucassen et al. 2013)	Three-day food recall diary	No significant difference in total intakes before and after 20:00			*P* = 0.89
(Mota et al. 2016)	3- day self-administered food diary	Not Reported	Not Reported	Chronotype score was negatively associated with protein intake. (g/kg/d) ET had a higher intake (β - coefficient = −0.23)	*P* = 0.04
(Maukonen et al. 2017)	48-hour dietary recalls	17.3 (0.3) E% on weekdays	17.4 (0.3) E% on weekdays	16.4 E% (0.3) on weekdays	*P* = 0.02
(Teixeira et al. 2018)	24-hour food recall	71.9 [55.0 – 97.2] g/d	79.3 [60.0 – 100.2] g/d	75.6 [57.3 – 105.8] g/d	*P* = 0.16
(Baron et al. 2011)	7-day food logs	Not Reported	14 ± 2.7 E%[k]	15 ± 2.0 E%[l]	*P* > 0.05
(Baron et al. 2013)	7-day food logs	Not Reported	69 ± 21 g/d (14%)[k]	84 ± 26 g/d (15%)[l]	*P* > 0.05
Total daily fat intake					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	Fat: 84.4 ± 31.6 g/d[a]	Not Reported	Fat: 85.6 ± 30.0 g/d[b]	*P-Trend* = 0.43
		Saturated fat: 28.2 ± 11.3 g/d[a]	Not Reported	Saturated fat: 28.8 ± 11.3 g/d[b]	*P-Trend* = 0.50
		Poly-unsaturated fat: 18.5 ± 7.7 g/d[a]	Not Reported	Poly-unsaturated fat: 18.5 ± 7.3 g/d[b]	*P-Trend* = 0.85
		Mono-unsaturated fat: 30.4 ± 12.3 g/d[a]	Not Reported	Mono-unsaturated fat: 31.0 ± 11.3 g/d[b]	*P-Trend* = 0.24
		Cholesterol: 304.3 ± 139.6 g/d[a]	Not Reported	Cholesterol: 308.0 ± 147.9 g/d[b]	*P-Trend* = 0.73
(Sato-	Dietary history	Fat: 28.9 ± 0.2 E%[c]	Fat: 29.3 ± 0.2 E%[d]	Fat: 30.1 ± 0.2 E%[g]	*P-Trend* < 0.01

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Mito, Sasaki et al. 2011)	questionnaire		29.7 ± 0.2 E%[e] 29.9 ± 0.21E%[f]		
		Cholesterol: 168 ± 3 mg/1000 kcal [c]	Cholesterol: 169 ± 2 mg/1000 kcal[d] 165 ± 2 mg/1000 kcal[e] 161 ± 2 mg/1000 kcal[f]	Cholesterol: 162 ± 3 mg/1000 kcal[g]	*P-Trend* < 0.05
(Vera et al. 2018)	Single 24-hour recalls	93.79 ± 1.500 g/d	Not Reported	93.03 ± 1.54 g/d	*P-Trend* = 0.73 *P-Trend* = 0.49
(Yoshizaki et al. 2018)	A semi-quantitative food frequency questionnaire	65.8 ± 1.3 g/d[h]	66.0 ± 1.2 g/d[i]	66.3 ± 1.10 g/d[j]	*P-Trend* = 0.88
(Lucassen et al. 2013)	Three-day food recall diary	NS difference in total intakes before and after 20:00			*P* = 0.14
(Mota et al. 2016)	3- day self-administered food diary	Not Reported	Not Reported	Chronotype score was negatively associated with cholesterol intake (mg/d) ET had a higher intake (β - coefficient = −0.24)	*P* = 0.04
(Maukonen et al. 2017)	48-hour dietary recalls	Fat: 31.7 (0.6) E% on weekdays	Fat: 32.1 (0.6) E% on weekdays	Fat: 32.3 (0.7) E% on weekdays	*P* = 0.81
		Fat: 31.1 (0.7) E% on weekends	Fat: 32.0 (0.7) E% on weekends	Fat: 33.1 (0.9) E% on weekends	*P* = 0.05
				Fat: Inversely associated Higher intake on weekends	*P-Trend* < 0.05
		SFAs: 11.6 (0.3) E% on weekdays	SFAs: 11.9 (0.3) E% on weekdays	SFAs: 11.8 (0.3) E% on weekdays	*P* = 1.00
		SFAs: 11.2 (0.4) E% on weekends	SFAs: 11.7 (0.4) on weekends	SFAs: 12.2 (0.5) E% on weekends	*P* = 0.06
				ME score was inversely associated on weekends ET was associated with higher intake of SFAs	*P-Trend* < 0.05

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Maukonen et al. 2016)	Food frequency questionnaire; Baltic Sea diet score	Fat: 32 E% in men	Fat: 32 E% in men	Fat: 32 E% in men	*P-Trend* = 0.67
		Fat: 30 E% in women	Fat: 31 E% in women	Fat: higher intake of 31 E% in women	*P-Trend* = 0.02
(Teixeira et al. 2018)	24-hour food recall	Fat: 45.2 [33.9 – 69.2] g/d	Fat: 53.7 [38.6 – 69.8] g/d	Fat: 53.6 [34.7 – 74.5] g/d	*P* = 0.10
				Breakfast skippers were negatively associated with fat intake (g/d) ET breakfast skippers had higher intake β-coefficient -0.18)	*P* < 0.05
		Cholesterol: 180.5 [102.7 – 278.9] mg/d	Cholesterol: 208.0 [142.1 – 296.6] mg/d	Cholesterol: 193.5 [127.6 - 297] mg/d	*P* = 0.18
(Baron et al. 2011)	7-d food logs	Not Reported	38 ± 7.2 E%[k]	35 ± 7.7 E%[l]	*P* > 0.05
(Baron et al. 2013)	7-day food logs	Not Reported	78 ± 23 g/d (38%)[k]	82 ± 24 g/d (35%)[l]	*P* > 0.05
Total daily micronutrient intake					
(Sato-Mito et al. 2011)	Dietary history questionnaire	Potassium: 1094 ± 12 mg/1000 kcal[c]	Potassium: 1101 ± 10 g/1000 kcal[d] 1084 ± 11 mg/1000 kcal[e] 1083 ± 10 mg/1000 kcal[f]	Potassium: 1046 ± 11 mg/1000 kcal[g]	*P-Trend* < 0.05
		Magnesium: 120 ± 1 mg/1000 kcal[c]	Magnesium: 121 ± 1 mg/1000 kcal[d] 120 ± 1 mg/1000 kcal[e] 119 ± 1 mg/1000 kcal[f]	Magnesium: 115 ± 1 mg/1000 kcal[g]	*P-Trend* < 0.05
		Iron: 3.73 ± 0.04 mg/1000 kcal[c]	Iron: 3.72 ± 0.03 mg/1000 kcal[d] 3.70 ± 0.03 mg/1000 kcal[e] 3.70 ± 0.03 mg/1000 kcal[f]	Iron: 3.59 ± 0.04 mg/1000 kcal[g]	*P-Trend* < 0.05
		Zinc: 4.12 ± 0.02 mg/1000 kcal[c]	Zinc: 4.14 ± 0.02 mg/1000 kcal[d] 4.11 ± 0.02 mg/1000 kcal[e]	Zinc: 4.04 ± 0.02 mg/1000 kcal[g]	*P-Trend* < 0.05

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
			4.07 ± 0.02 mg/1000 kcal[f]		
		Vitamin A: 308 ± 10 µg/1000 kcal[c]	Vitamin A: 294 ± 9 µg/1000 kcal[d] 287 ± 9 µg/1000 kcal[e] 297 ± 9 µg/1000 kcal[f]	Vitamin A: 271 ± 10 µg/1000 kcal[g]	*P-Trend* < 0.05
		Vitamin D: 3.7 ± 0.1 μg/1000 kcal[c]	Vitamin D: 3.7 ± 0.1 µg/1000 kcal[d] 3.6 ± 0.1 µg/1000 kcal[e] 3.5 ± 0.1 µg/1000 kcal[f]	Vitamin D: 3.4 ± 0.1 µg/1000 kcal[g]	*P-Trend* < 0.01
		Pyridoxin: 0.53 ± 0.01 mg/1000 kcal[c]	Vit B6: 0.54 ± 0.01 mg/1000 kcal[d] 0.53 ± 0.01 mg/1000 kcal[e] 0.52 ± 0.01 mg/1000 kcal[f]	Pyridoxin: 0.51 ± 0.01 mg/1000 kcal[g]	*P-Trend* < 0.01
		Riboflavin: 0.70 ± 0.01 mg/1000 kcal[c]	Vit B2: 0.69 ± 0.01 mg/1000 kcal[d] 0.69 ± 0.01 mg/1000 kcal[e] 0.69 ± 0.01 mg/1000 kcal[f]	Riboflavin: 0.67 ± 0.01 mg/1000 kcal[g]	*P-Trend* < 0.01
		Thiamine: 0.41 ± 0.003 mg /1000 kcal[c]	Vit B1: 0.42 ± 0.003 mg/1000 kcal[d] 0.41 ± 0.003 mg/1000 kcal[e] 0.41 ± 0.003 mg/1000 kcal[f]	Thiamine: 0.40 ± 0.004 mg/1000 kcal[g]	*P-Trend* < 0.01
		Folate: 156 ± 2 µg/1000 kcal[c]	Folate: 155 ± 2 µg/1000 kcal[d] 153 ± 2 µg/1000 kcal[e] 155 ± 2 µg/1000 kcal[f]	Folate: 145 ± 2 µg/1000 kcal[g]	*P-Trend* < 0.01
		Calcium: 275 ± 4 mg/1000 kcal[c]	Calcium: 273 ± 4 mg/1000 kcal [d] 269 ± 4 mg/1000 kcal[e] 266 ± 4 mg/1000 kcal[f]	Calcium: 251 ± 4 mg/1000 kcal[g]	*P-Trend* < 0.001
Total daily intake of food groups					
(Sato-Mito et al. 2011)	Dietary history questionnaire	Alcohol: 0.19 ± 0.05 E%[c]	Alcohol: 0.13 ± 0.04 E% 0.24 ± 0.05 E% 0.29 ± 0.04 E%	Alcohol: 0.44 ± 0.05 E% [b]	*P-Trend* < 0.01

Reference	Method of assessment	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) and other analysis**
		Rice: 171.4 ± 2.9 g/1000 kcal[c]	Rice: 167.7 ± 2.5 g/1000 kcal 158.0 ± 2.6 g/1000 kcal 153.6 ± 2.5 g/1000 kcal	Rice: 150.0 ± 2.8 g/1000 kcal [b]	*P-Trend* <0.001
		Vegetables: 126.7 ± 3.1 g/1000 kcal[c]	Vegetables: 127.5 ± 2.6 g/1000 kcal 121.9 ± 2.8 g/1000 kcal 121.3 ± 2.6 g/1000 kcal	Vegetables: 109.8 ± 2.9 g/1000 kcal [b]	*P-Trend* <0.001
		Pulses: 26 .6 ± 0.8 g/1000 kcal[c]	Pulses: 25.8 ± 0.7 g/1000 kcal 25.7 ± 0.7 g/1000 kcal 24.7 ± 0.7 g/1000 kcal	Pulses: 22.5 ± 0.8 g/1000 kcal [b]	*P-Trend* <0.001
		Eggs: 19.3 ± 0.6 g/1000 kcal[c]	Eggs: 19.4 ± 0.5 g/1000 kcal 18.1 ± 0.5 g/1000 kcal 17.1 ± 0.5 g/1000 kcal	Eggs: 17.4 ± 0.6 g/1000 kcal [b]	*P-Trend* <0.001
		Noodles: 28.8 ± 1.4 g/1000 kcal[c]	Noodles: 33.6 ± 1.2 g/1000 kcal 36. ± 1.23 g/1000 kcal 38.5 ± 1.2 g/1000 kcal	Noodles: 46.4 ± 1.3 g/1000 kcal [b]	*P-Trend* <0.001
		Dairy: 77.4 ± 3.1 g/1000 kcal[c]	Dairy: 76.5 ± 2.6 g/1000 kcal 74.3 ± 2.8 g/1000 kcal 71.4 ± 2.6 g/1000 kcal	Dairy: 65.6 ± 2.9 g/1000 kcal [b]	*P-Trend* < 0.05
		Confections: 42.5 ± 1.0 g/1000 kcal[c]	Confections: 41.8 ± 0.8 g/1000 kcal 44.8 ± 0.9 g/1000 kcal 46.8 ± 0.9 g/1000 kcal	Confections: 46.7 ± 1.0 g/1000 kcal [b]	*P-Trend* < 0.05
		Meat: 33.1 ± 0.7 g/1000 kcal[c]	Meat: 34.0 ± 0.6 g/1000 kcal 34.8 ± 0.6 g/1000 kcal 33.4 ± 0.6 g/1000 kcal	Meat: 35.7 ± 0.7 g/1000 kcal [b]	*P-Trend* < 0.05

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Vera et al. 2018)	Single 24-hour recalls	Not Reported	Not Reported	Lower intake of cereals	$P < 0.05$ *Other analysis:* ET have 1.3 times higher odds for alcohol (OR: 1.52; CI 95%: 1.25, 1.86; $P < 0.001$)
(Najem et al. 2020)	The Yale Food Addiction Scale (YFAS)	Not Reported	Not Reported	Not Reported	*Other analysis:* ME score negatively correlated with number of units of caffeine containing beverages/d ET was associated with higher intake of units of caffeine beverages/d (r = -0.14, $P = 0.00$)
(Lázár et al. 2012)	Medical Question-naire	Alcohol: reported lower intake	Not Reported	Not Reported	$P < 0.001$
		Daily caffeine intake was associated with diurnal preference; MTs reported lower intake	Not Reported	Not Reported	$P < 0.05$
(Yoshizaki et al. 2018)	A semi-quantitative food frequency questionnaire	Potatoes and starches intake: higher intake of 36.4 ± 1.7 g/d[h]	Potatoes and starches: 32.7 ± 1.6 g/d[i]	Potatoes and starches: 30.9 ± 1.5 g/d[j]	*P-Trend* = 0.04
		Green/yellow vegetables: higher intake of 76.2 ± 2.2 g/d[h]	Green/yellow vegetables: 67.1 ± 2.1 g/d[i]	Green/yellow vegetables: 65.4 ± 2.0 g/d[j]	*P-Trend* < 0.001 *Other analysis:* MT[h] were associated with a higher intake (β = 0.15, $P < 0.001$)
		White vegetables: higher intake of 123 ± 3.7 g/d[h]	White vegetables: 112 ± 3.4 g/d[i]	White vegetables: 112± 3.3 g/d[j]	*P-Trend* = 0.01 *Other analysis:* White vegetables: associated with high chronotype score MT[h] were associated with a higher intake (β = 0.11, $P < 0.001$)

Reference	Method of assessment	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) and other analysis**
		Fruits: higher intake of 81.9 ± 3.8 g/d[h]	Fruits: 72.7 ± 3.5 g/d[i]	Fruits: 59.9 ± 3.4 g/d[j]	*P-Trend* < 0.001 *Other analysis:* Fruits: associated with high chronotype score MT[h] were associated with a higher intake (β = 0.11, *P* < 0.001)
		Algae: higher intake of 4.6 ± 0.2 g/d[h]	Algae: 4.3 ± 0.2 g/d[i]	Algae: 4.1 ± 0.2 g/d[j]	*P-Trend* = 0.02 *Other analysis:* Algae: associated with high chronotype score MT[h] were associated with a higher intake (β = 0.10, *P* < 0.001)
		Confectioneries / savory snacks: 80.7 ± 2.9 g/d[h]	Confectioneries / savory snacks: 89.2 ± 2.7 g/d[i]	Confectioneries / savory snacks: 94.9 ± 2.6 g/d[j]	*P-Trend* = 0.001 *Other analysis:* Confectioneries/savory snacks: negatively associated with high chronotype score ET[j] were associated with a higher intake (β = -0.10, *P* < 0.001)
		Sugar-sweetened beverages: 42.7 ± 5.4 g/d[h]	Sugar-sweetened beverages: 43.8 ± 5.0 g/d[i]	Sugar-sweetened beverages: 60.8 ± 4.9 g/d[j]	*P-Trend* = 0.01 *Other analysis:* Sugar-sweetened beverages: negatively associated with high chronotype score ET[j] were associated with a higher intake (β = -0.13, *P* < 0.001)
(Silva et al. 2016)	Food frequency questionnaire	Not Reported	Not Reported	Meat: ET associated with a higher intake (β = 0.21)	*P* = 0.003
(Mota et al. 2016)	3- day self-administered food diary	Not Reported	Not Reported	Chronotype score was negatively associated with: - intake of sweets (servings/d) ET had a higher intake (β coefficient = −0.27) - vegetable intake (servings/d)	*P* = 0.03

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
				ET had a higher intake (β - coefficient = −0.26)	*P* = 0.04
		Chronotype score was positively associated with oil and fat intake (servings/d) MT had a higher intake (β - coefficient = 0.27)	Not Reported	Not Reported	*P* = 0.03
(Maukonen et al. 2017)	48-hour dietary recalls	Alcohol: 4.6 (1.5) g on weekdays	Alcohol: 4.3 (1.5) g on weekdays	Alcohol: 9.7 (1.9) g on weekdays Alcohol: Intakes increased with lower ME scores (ET) on weekdays	*P* = 0.57 *P-Trend* = 0.04
(Maukonen et al. 2016)	Food frequency questionnaire & Baltic Sea diet score	Cereals: women 85 g/d & men 89 g/d	Cereals: women: 79 g/d & men: 84 g/d	Cereals: women 74 g/d & men 78 g/d	*P-Trend* <0.001
		Fish: men 55 g/d	Fish: men 53 g/d	Fish: Men consumed less 49 g/d	*P-Trend* <0.05
		Alcohol: women 3.6 g/d & men 10.6 g/d	Alcohol: women: 4.4 g/d & men: 11.8 g/d	Alcohol: Consumed more. Women 5.1 g/d & men 13.3 g/d	women: *P-Trend* <0.001 men: *P-Trend* = 0.003
(Li et al. 2018)	Sugary beverage consumption – number of bottles or tins consumed per day last week.	Not Reported	Not Reported	Not Reported	*Other analysis:* Negative direct effects were found between chronotype and sugary beverage consumption MT had lower intake (β = − 0.15, SE = 0.03, *P* < 0.01)
(Culnan et al. 2013)	The Gray-Donald Eating Patterns Question-naire	Alcohol: Baseline: No difference in alcohol intake			*P* > 0.05
		Caffeine no difference between chronotypes at baseline			*P* > 0.05
		Not Reported	Not Reported	At follow-up: more ETs reported drinking alcohol [$x^2(1, n = 54) = 5.94$]	*P* < 0.05
				At follow-up: ET not more likely to change alcohol drinking status throughout study [x^2 (1, *n* = 54) = 3.19]	*P* > 0.05

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Baron et al. 2011)	7-day food logs	Not Reported	Fruits and vegetables: 3.4 ± 1.8 servings / d[k]	Fruits and vegetables: lower intake of 1.9 ± 1.1 servings / d[l]	$P < 0.01$ *Other analysis:* Fruits and vegetable intakes were negatively correlated with sleep timing (r = -0.49, $P < 0.01$) IT[k] were associated with higher intakes
			Fast food meals: (3.0 ± 1.8) servings/week[k]	Fast food meals/week: higher intake of (5.2 ± 3.8) servings/week[l]	$P < 0.05$
			Full-calorie sodas: (1.3 ± 2.5) servings/week[k]	Full-calorie sodas: higher intake of (4.5 ± 6.5) servings/week[l]	$P < 0.05$
		Not Reported	Caffeinated drinks: (7.3 ± 6.5) servings/week[k]	Caffeinated drinks: trend for higher intake (13.0 ±12.6) servings/week[l]	$P < 0.10$
(Muscogiuri et al. 2020)	PREDIMED questionnaire	Not Reported	Not Reported	Not Reported	*Other analysis:* - Food intake negatively associated to chronotype score. - ET associated with a higher OR for: red/processed meat <1/d (OR: 1.05; 95% CI: 1.02–1.08; P <0.001); butter, cream, margarine <1/d (OR: 1.05; 95% CI: 1.02–1.08;1.05, P = 0.001); commercial sweets / confectionery ≤2/week (OR: 1.04; 95% CI: 1.01–1.06; P = 0.007); soda drinks <1/d (OR: 1.04; 95% CI: 1.01–1.07; P = 0.001) - Food intake positively associated to chronotype score. MT were associated with a higher OR for:

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
					EVOO >4tbs (OR: 1.03; 95% CI: 1.00–1.06; *P* = 0.01); vegetable ≥2s/d (OR: 1.05; 95% CI: 1.02–1. 07; *P* < 0.001); fruit ≥ 3s/d (OR: 1.07; 95% CI: 1.04–1.10; *P* < 0.001); fish/seafood ≥3/w (OR: 1.037; 95% CI: 1.00–1.06; *P* = 0.02); poultry more than> red meats (OR: 1.05; 95% CI: 1.03–1.08; *P* < 0.001); tree nuts ≥3/w (OR: 1.03; 95% CI: 1.00–1.06; *P* = 0.01); wine glasses ≥7/w (OR: 1.05; 95% CI: 1.01–1.09; *P* = 0.004) - Most predictive factor of chronotype score among single contributing PREDIMED food items and score: Both MT (R^2 = 0.18, *P* < 0.001) and ET (R^2 = 0.23, *P* = 0.02) most influenced by PREDIMED score and IT most influenced by butter, cream and margarine <1/d (R^2 = 0.09, *P* = 0.04).
(Maukonen et al. 2017)	48-hour dietary recalls	Alcohol: 1.8 (0.7) g after 20:00 on weekdays	Alcohol: 1.9 (0.7) g after 20:00 on weekdays	Alcohol: 4.0 (0.9) g after 20:00 on weekdays	*P* = 0.09
		Not Reported	Not Reported	Alcohol: Intake increased with lower ME score values (ET) after 20:00 on weekdays	*P-Trend* <0.05
Daily energy distribution					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	Not Reported	Not Reported	Not Reported	*Other analysis:* Higher energy intake in morning window (within 2 hours after getting out of bed) associated with lower OR

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
					for overweight/obese in MT[a] (OR: 0.32; 95% CI: 0.16, 0.66; *P-Trend* = 0.0006)
		Not Reported	Not Reported	Not Reported	*Other analysis:* Higher energy intake at nighttime window (within 2 hours before bedtime), associated with higher OR for overweight/obese in ET[b] (OR: 4.94; 95% CI: 1.61, 15.1; *P-Trend* = 0.01)
(Muñoz et al. 2020)	Hypocaloric dietary treatment according to the Spanish Federation of Nutrition, Food and Dietetics guidelines	Breakfast 30%, midmorning 10%, lunch 35%; mid-afternoon 5% and dinner 20%	Not Reported	Breakfast 20%, mid-morning 5%, lunch 35%; mid-afternoon 10% and dinner 30%	Not Reported
(Maukonen et al. 2017)	48-hour dietary recalls	99% of MTs had energy intake >0 kJ on weekday mornings by 10:00	Not Reported	80% of ET had energy intake >0 kJ on weekday mornings by 10:00	Not Reported
		Not Reported	Not Reported	Weekday mornings: 350 kJ (4% TEI) lower energy intake as compared with MT	$P < 0.001$
		Not Reported	Not Reported	Weekend mornings by 10:00: 380 kJ lower energy than MT	$P = 0.004$
		81% of MTs had energy intake >0 kJ on weekday evenings by 20:00	Not Reported	94% of ET had energy intake >0 kJ on weekday evenings by 20:00	Not Reported
		Not Reported	Not Reported	Weekday evenings by 20:00: 430 kJ (6% TEI) more energy than MT	$P < 0.001$
		Not Reported	Not Reported	Weekend evenings by 20:00: 590 kJ (7% TEI) more energy than MT	$P < 0.001$

Reference	Method of assessment	Differences between types			
		Morning type	**Intermediate type**	**Evening type**	***P*-value (ET vs IT/MT) and other analysis**
		Not Reported	Not Reported	Cumulative energy intake of ET: Weekdays: lower from the beginning of the day until 22:00 Weekends: lower from the beginning of the day until 01:00	Not Reported
		Weekends: three peaks of energy intake of the same height at 08:00, 00:00 and 17:00	Not Reported	Weekdays: energy intake peaks on weekdays are an hour later than MT Weekends: six peaks of energy intake Highest peak at 19:00	Not Reported
		Sucrose: 12.5 (1.2) E% after 20:00 on weekdays	Sucrose: 13.4 (1.2) E% after20:00 on weekdays	Sucrose: 1.1 E% units more after 20:00 on weekdays 13.6 (1.5) E%	P <0.05
		Sucrose: 10.2 (1.9) E% after 20:00 on weekends	Sucrose: 13.8 (1.9) after 20:00 on weekends	Sucrose: 3.1 E% units more by 20:00 on weekends 13.3 (2.5) E%	P <0.05
(Maukonen et al. 2019) (Maukonen et al. 2019)	48-h dietary recalls covering two previous consecutive days	1596 (41%) kJ in the morning	Not Reported	340 kJ less energy in the morning - 1252 (90%) kJ	P < 0.01
		953 (43%) kJ in the evening	Not Reported	450 kJ more in the evening - 1402 (97%) kJ	P < 0.001
		Not Reported	Not Reported	Not Reported	*Other analysis:* %TEI in the morning and obesity risk had a significant interaction between %TEI in the morning and chronotype on increase in weight (≥ 5%) (P = 0.025) and increase in BMI (≥ 5%) (P = 0.012)

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Baron et al. 2011)	7- day food logs	Not Reported	Caloric intake after 20:00: 376 ± 237 kcal/d[k]	Caloric intake after 20:00: 754 ± 373 kcal/d[l]	$P < 0.001$ *Other analysis:* ET was associated with more calories consumed after 20:00 ($\beta = 0.45$, $r2_{\Delta} = 0.18$, $P = 0.001$)[k]
		Not Reported	Caloric intake at breakfast: 355 ± 133 kcal/d[k]	Caloric intake at breakfast: 285 ± 143 kcal/d[l]	$P > 0.05$
		Not Reported	Caloric intake at lunch: 528 ± 188 kcal/d[k]	Caloric intake at lunch: 503 ± 378 kcal/d[l]	$P > 0.05$
		Not Reported	Caloric intake for snacks: 405 ± 284 kcal/d [k]	Caloric intake for snacks: 536 ± 323 kcal/d[l]	$P > 0.05$
		Not Reported	Caloric intake at dinner: 630 ± 198 kcal/d [k]	Caloric intake at dinner: 825 ± 352 kcal/d[l]	$P < 0.05$
		Not Reported	Caloric intake after dinner: 150 ± 151 kcal/d [k]	Caloric intake after dinner: 208 ± 166 kcal/d[l]	$P > 0.05$
		Not Reported	Not Reported	Cumulative energy intake across the day; 1h increments Fewer calories at 9:00[l]	$P < 0.001$
		Not Reported	Not Reported	Fewer calories at 10:00, 11.00 and 12:00[l]	$P = 0.001$
		Not Reported	Not Reported	Afternoon: intake increased steeply, and caloric intake matched and began to exceed normal sleepers around average dinner time[l]	Not Reported
		Not Reported	IT reached a plateau as early as 21:00[k]	Caloric intake of late sleepers continued to rise after 23:00[l]	Not Reported
(Lucassen et al. 2013)	Three-day food recall	Working days: 299 ± 354 kcal after 20:00	Not Reported	Consumed more calories after 20:00 on working days 677 ± 460 kcal	$P < 0.001$
		Non-working days: 327 ± 354 kcal after 20:00	Not Reported	Consumed more calories after 20:00 on non-working days 537 ± 480 kcal/d	$P = 0.03$

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) and other analysis
		Morning type	Intermediate type	Evening type	
(Zerón-Rugerio et al. 2020)	6- day food logs & Quality Index Food Consumption Patter	Breakfast: 24.8 (10.4) % of kcal[m]	Breakfast: 26.9 (10.4) % of kcal[n] 26.5 (6.9) % of kcal[o]	Breakfast: 22.8 (8.3) % of kcal[p]	*P* = 0.26
		Lunch: 31.3 (7.5) % of kcal[m]	Lunch: 29.5 (10.2) % of kcal[n] 33.7 (10.5) % of kcal[o]	Lunch: 30.9 (9.6) % of kcal[p]	*P* = 0.36
		Dinner: 18.0 (10.4) % of kcal[m]	Dinner: 18.6 (9.8) % of kcal[n] 20.7 (9.1) % of kcal[o]	Dinner: 23.5 (11.3) % of kcal[p]	*P-Trend* = 0.02
Daily carbohydrate distribution					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	Not Reported	Not Reported	Not Reported	*Other analysis:* -In MT[a], the highest quintile of % carbohydrate intake in the morning (within 2h after getting out of bed) is associated with 80% decrease in risk for being overweight/obese (OR: 0.2; 95% CI: 0.10, 0.42; *P-Trend* <0.0001) -In ET[b], the highest quintile of % carbohydrate intakes during the evening (within 2 h before bedtime) is associated with an increase in OR for being overweight/obese (OR: 4.48; 95% CI: 1.64, 12.2; *P-Trend* = 0.01) -In ET[b], the highest quintile of % sugar intake at night (within 2h before bedtime) is associated with a 3-fold increase in OR for being overweight/obese (OR: 3.11; 95% CI:1.17; 8.22; *P-Trend* = 0.02) -In MT[a], the highest quintile of % sugar intakes during the morning (within 2 h after getting out of bed) (OR: 0.23; 95% CI: 0.11; 0.49; (*P-Trend* = 0.0003), % fiber (OR: 0.31;

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
					95% CI: 0.15; 0.65); (*P-Trend* = 0.0008) was associated with a decrease in OR for being overweight / obese
(Maukonen et al. 2017)	48-hour dietary recalls	Intake by 10:00 on weekdays: 52.8 (1.3) E%	Intake by 10:00 on weekdays: 50.5 (1.3) E%	Intake by 10:00 on weekdays: 47.1 (1.6) E%	$P < 0.001$ *P-Trend* < 0.001
		Intake after 20:00: 48.8 (2.0) E% on weekdays	Intake after 20:00: 51.3 (2.0) E% on weekdays	Intake after 20:00: 51.2 (2.4) E% on weekdays	*P-Trend* = 0.01
		Not Reported	Not Reported	CHO intakes increased with lower ME score values (ET) after 20:00 on weekdays	*P-Trend* < 0.05
		Intake by 10:00 on weekends: 52.6 (2.6) E%	Intake by 10:00 on weekends: 48.3 (2.4) E%	Intake by 10:00 on weekends: 48.5 (3.1) E%	*P-Trend* = 0.003
		Intake after 20:00: 46.3 (3.4) on weekends	Intake after 20:00: 50.3 (3.4) on weekends	Intake after 20:00: 49.8 (4.4) on weekends	$P = 1.00$
(Baron et al. 2013)	7- day Food logs	Not Reported	After 20:00: 47 ± 31 g (19%)[k]	After 20:00: higher intake 87 ± 39 g (33%)[l]	$P < 0.01$ *Other analysis:* - After 20:00: Moderate positive correlation with midpoint of sleep. - ET[l] were associated with higher intake (r = 0.52, $P < 0.001$)
Daily protein distribution					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	Not Reported	Not Reported	Not Reported	- In MT[a], the highest % protein intakes during the morning (within 2 h after

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
					getting out of bed) was associated with a 61% decrease in OR: 0.39 (CI 95%: 0.19, 0.81; (*P-Trend* = 0.03) for being overweight / obese - In ET[b], the highest % protein intake consumed at night (2 h before bedtime) is associated with 3.7-fold increase in OR for being overweight/obese (OR: 3.74; CI 95%: 1.33, 10.5; *P-Trend* = 0.02)
(Maukonen et al. 2017)	48-hour dietary recalls	Protein intake after 20:00 on weekdays: 12.4 (0.8) E%	Protein intake after 20:00 on weekdays: 13.1 (0.8) E%	Protein intake after 20:00 on weekdays: 13.4 (0.9) E%	*P-Trend* = 0.04
		Not Reported	Not Reported	Protein intakes increased with lower ME score values (ET) after 20:00 on weekdays	*P-Trend* < 0.05
		Protein intake after 20:00 on weekends: 11.6 (1.3) %	Protein intake after 20:00 on weekends: 12.7 (1.3) E%	Protein intake after 20:00 on weekends: 14.2 (1.7) E%	*P* = 0.25
		Intake by 10:00 on weekdays:14.8 (0.9) E%	Intake by 10:00 on weekends: 13.6 (0.9) E%	Intake by 10:00 on weekdays: 13.6 (0.9) E%	*P* < 0.001 *P-Trend* < 0.001
		Intake by 10:00 on weekends:14.8 (0.9) E%	Intake by 10:00 on weekends: 13.6 (0.9) E%	Intake by 10:00 on weekends:11.4 (1.2) E%	*P* < 0.003 *P-Trend* < 0.001
(Baron et al. 2013)	7-day food logs	Not Reported	After 20:00: 15 ± 12 g (21%)[c]	More protein at dinner [d]	*P* < 0.01
				After 20:00: 32 ± 16 g (37%)[d]	*P* > 0.01 *Other analysis:* - After 20:00: Moderate positive correlation with midpoint of sleep - ET[d] were associated with higher intake (r = 0.53 *P* < 0.001)[d]

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) and other analysis
		Morning type	Intermediate type	Evening type	
Daily fat distribution					
(Xiao et al. 2019)	24-Hour Dietary Assessment Tool	No association between timing of fat intake and BMI[a,b]			*Other analysis:* -No association between total fat intake during the morning (within 2 h after getting out of bed) (*P-Trend* = 0.47), cholesterol (*P-Trend* = 0.35), saturated fat (*P-Trend* = 0.90) and monounsaturated fat (*P-Trend* = 0.42) and OR of being overweight / obese in MT[a] - No association between total fat intake during night (2 h before bedtime) (*P-Trend* = 0.30), cholesterol (*P-Trend* = 0.06), saturated fat (*P-Trend* = 0.34) and monounsaturated fat (*P-Trend* = 0.31), polyunsaturated fat (*P-Trend* = 0.08) and OR of being overweight/obese in ET[b].
(Maukonen et al. 2017)	48-hour dietary recalls	Fat: 23.8 (1.0) E% by 10:00 on weekdays	Fat: 23.3 (1.0) E% by 10:00 on weekdays	Fat: 19.6 (1.2) E% by 10:00 on weekdays	*P* < 0.001 *P-Trend* = 0.002
		Fat: 22.6 (1.6) E% by 10:00 on weekends	Fat: 20.3 (1.5) E% by 10:00 on weekends	Fat: 18.8 (2.0) E% by 10:00 on weekends	*P -Trend* = 0.001
		Fat: 21.5 (1.2) E% after 20:00 on weekdays	Fat: 23.4 (1.2) E% after 20:00 on weekdays	Fat: 26.1 E% after 20:00 on weekdays (26.1 (1.5) E%)	*P* = 0.0025 *P-Trend* = <0.001
		Fat: 17.3 (2.0) E% after 20:00 on weekends	Fat: 20.0 (2.0) E% after 20:00 on weekends	Fat: on weekends after 20:00 (26.0 (2.6) E%)	*P* < 0.001
		SFAs: 9.0 (0.5) E% by 10:00 on weekdays	SFAs: 9.5 (0.5) E% by 10:00 on weekdays	SFAs: 7.3 (0.6) E% by 10:00 on weekdays	*P* = 0.002 *P -Trend* = 0.02
		SFAs: 8.3 (0.7) E% by 10:00 on weekends	SFAs: 7.1 (0.7) E% by 10:00 on weekends	SFAs: 6.4 (0.9) E% by 10:00 on weekends	*P* < 0.05
		SFAs: 8.8 (0.6) E% after 20:00 on weekdays	SFAs: 9.7 (0.6) E% after 20:00 on weekdays	SFAs: 10.3 (0.7) E% after 20:00 on weekdays	*P* <0.03

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
		SFAs: 6.8 (1.0) E% after 20:00 on weekends	SFAs: 7.9 (1.0) E% after 20:00 on weekends	SFAs: on weekends after 20:00 (10.3 (1.2) E%)	*P* <0.003
(Baron et al. 2013)	7- day food logs	Not Reported	After 20:00: 16 ± 12g (19 %)[k]	After 20:00: 30 ± 17 g (35%)[l]	*P* < 0.05
		Not Reported	4 h before sleep: 11 ± 9 g (16 %)[k]	Consumed less fat in the 4 h before sleep 10 ± 12 g (12 %)[l]	*P* < 0.01 *Other analysis:* - After 20:00: Moderate positive correlation with midpoint of sleep, - ETs[l] were associated with higher intake (r = 0.48, *P* < 0.01)
Adherence to guidelines					
(Najem et al. 2020)	The Yale Food Addiction Scale	Not Reported	Not Reported	Chronotype scores were negatively correlated with YFAS scores (r = - 0.10) ET was associated with a higher YFAS score	*P* = 0.10
(Zeron-Rugerio et al. 2019)	The MD Quality Index for children and adolescents	Not Reported	Not Reported	Lower adherence to the MD (β = 0.019)	*P* = 0.06
(Maukonen et al. 2016)	Food frequency questionnaire & Baltic Sea diet score	Not Reported	Not Reported	Lower adherence to the Baltic Sea diet score	*P-Trend* < 0.05
(De Amicis et al. 2020)	14-item adherence to traditional MD questionnaire	Higher adherence (7 ± 2)	Not Reported	Lower adherence (6 ± 2)	*P* < 0.05
(Culnan et al. 2013)	The Gray-Donald Eating Patterns Questionnaire	Not Reported	Junk food consumption did not vary by chronotype at baseline		*P* > 0.05
			After 8-weeks chronotype was not associated with change in scores on the Junk Food subscale		*P* > 0.05
	PREDIMED (Prevención con	PREDIMED score: 8.8 ± 1.9	PREDIMED score: 7.0 ± 1.5	PREDIMED score: 5.1 ± 1.8 (lowest score)	*P* < 0.001 *Other analysis:*

Reference	Method of assessment	Differences between types			
		Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) and other analysis
(Muscogiuri et al. 2020)	Dieta Mediterránea) questionnaire				- Chronotype score was positively associated to PREDIMED score - MT were associated with a higher PREDIMED score (r = 0.59, *P* < 0.001)
		Low adherence to MD: 3(3.0%) subjects	Low adherence to MD: 6 (12.0%) subjects	Low adherence to MD: 12 (54.5%) subjects	*P* < 0.001
		Average adherence to MD: 58 (58.0%) subjects	Average adherence to MD: 42 (84.0%)	Average adherence to MD: 10 (45.5%)	*P* = 0.001
		High adherence to MD: 9 (39.0%) subjects	High adherence to MD: 2 (4.0%) subjects	High adherence to MD: 0 (0%)	*P* < 0.001
(Zerón-Rugerio et al. 2020)	6- day food logs & Quality Index Food Consumption Pattern	Diet quality: 57.9 ± 6.8[m]	Diet quality: 60.7 ± 8.1[n] Diet quality: 64.0 ± 9.8[o]	Diet quality: 67.3 ± 9.4[p]	*P* < 0.001 or *P -Trend* < 0.001

[1] BMI, Body mass index, PREDIMED, Prevención con Dieta Mediterránea, P-Trend refers to the continuous association between the MEQ or MCTQ score and exposures of interest, MD, Mediterranean Diet, MSFsc, Mid-sleep midsleep corrected for sleep duration on free days, TDM, Elapsed time between dinner and the midpoint of sleep, TFEQ, Three-Factor Eating Questionnaire, %TWL, total body weight loss, Values reported as mean and standard deviation (SD) unless stated otherwise, YFAS, Yale Food Addiction Scale.

[2] [a]Early sleep was defined as a chronotype earlier than the median (3:04 AM), [b]Late sleep was defined as a chronotype later than the median, [c]Based on earliest midpoint of sleep quintiles, [d]Based on midpoint of sleep quintiles 2, [e]Based on midpoint of sleep quintiles 3, [f]Based on midpoint of sleep quintiles 4, [g]Based on latest midpoint of sleep quintiles, [h]Based on MEQ score tertile 1: 34–53, [i]Based on MEQ score tertile 2: 54–59, [j]Based on MEQ score tertile 3: 60–76, [k]Based on normal sleep timing (midpoint 4:08 am), [l]Based on late sleep timing (midpoint of sleep 7:15 am), [m]Based on wakeup time <7:52 h and early-bedtime <23:48 h and defined as early-bedtime/early-rise (EE), [n] Based on early-bedtime (<23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as early-bedtime/late-rise (EL), [o] Based on late-bedtime (≥23:48 h) and wakeup time (<7:52 h) and defined as late-bedtime/early-rise (LE),[p] Based on late-bedtime (≥23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as late-bedtime/late-rise (LL).

4.3.4 Differences between chronotype & eating behaviour

The eating behaviour that was most investigated among chronotypes was meal timing (clock hours for meals), meal skipping and portion sizes (**Table 4.4**):

Meal timing, skipping and intervals between meals and bedtime. Compared with other types, ET were more likely to display undesirable eating behaviour, for example, reporting later clock times for main meals (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019). As to be expected, the ETs had later clock times for breakfast than MT (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019), or even skipped breakfast altogether (Sato-Mito et al. 2011, Silva et al. 2016, Teixeira et al. 2018). Based on their individual preferences, ET also had later habitual clock times for lunch (Baron et al. 2011, Sato-Mito et al. 2011, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019), and dinner (Baron et al. 2011, Sato-Mito et al. 2011, Vera et al. 2018, Xiao et al. 2019) than MT. Across the studies ET had breakfast between 14 min to 2 h 44 min‡ later, lunch between 2 min to 1 h and 23 min‡ later, and dinner between 3 min to 1 h ‡ later (Sato-Mito et al. 2011, Lucassen et al. 2013, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019). Interestingly, ET had a longer time interval between the last meal or snack of the day (dinner) and bedtime (average of 316 min‡) compared with IT/MT (average of 231 minutes‡) (Baron et al. 2011, Xiao et al. 2019).

Portion sizes, number of servings and eating occasions. ET were 1.4 times (OR: 1.44; CI 95%: 1.16, 1.78; $P = 0.001$) more likely to consume larger portion sizes and 1.3 times (OR: 1.27; CI 95%: 1.04, 1.56; $P < 0.019$) more likely to have second servings (Vera et al. 2018); as well as watch television while eating (Sato-Mito et al. 2011), compared to MT and IT. There were no differences or associations between the number of eating occasions per day (Lucassen et al. 2013, Teixeira et al. 2018).

Eating behaviour scores. Four studies (Lázár et al. 2012, Lai & Say 2013, Vera et al. 2018, Beaulieu et al. 2020) investigated the associations and differences between chronotypes and the Three Factor Eating Questionnaire (TFEQ) scores (restraint which is the conscious restriction of food intake to control body weight and shape, disinhibition which is the loss of control of food intake that leads to overconsumption of food and hunger which is feelings and subjective perceptions of hunger that leads to food intake) (Stunkard & Messick 1985) and their subcategories (flexible and rigid control, habitual, emotional and situational disinhibition, internal and external locus for hunger). A higher score indicates a higher level of said eating behaviour. Vera et al. observed that ET had a higher total eating behaviour score (1.93 ± 0.26) (higher scores = more deleterious eating behaviours) and emotional eating score (12.40 ± 0.19) (<12, non-emotional; ≥12, emotional) than the MT scores of 0.01 ± 0.25 and 11.85 ± 0.19 respectively (*P*-value <0.001 and *P*-value < 0.046 respectively) (2018). The ET also felt less in control over their diet (OR: 1.33; 95% CI: 1.10, 1.60; $P = 0.003$), experienced more stress-related eating (OR: 1.27; 95%

CI: 1.04, 1.55; *P* = 0.019), food cravings (OR: 1.20; 95% CI: 0.99, 1.45; *P* = 0.063), and had greater problems controlling the amount of food consumed (OR: 1.31; 95% CI: 1.03, 1.58; *P* = 0.006) (Vera et al. 2018).

Table 4.4 *- Differences between chronotype & eating behaviour*

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
Meal timing					
(Xiao et al. 2019)	The Automated Self-Administered 24-Hour Dietary Assessment Tool (ASA24)	Breakfast: 7.6 ± 1.0 h[a]	Not Reported	8.6 ± 0.9 h[b]	*P-Trend* <0.001
		Lunch: 12.6 ± 1.1 h[a]	Not Reported	12.8 ± 1.3 h[b]	*P* <0.004
		Dinner: 18.2 ± 0.8 h[a]	Not Reported	18.5 ± 1.1 h[b]	*P-Trend* <000.1
(Sato-Mito et al. 2011)	Dietary history questionnaire	Breakfast: 6:35 ± 0:02 h:min[c]	Breakfast: 7:01 ± 0:02 h:min[d] 7:23 ± 0:02 h:min[e] 7:52 ± 0:02 h:min[f]	Breakfast: 9:19 ± 0:02 h: min[g]	*P-Trend* <0.0001 *P* <0.001
		Lunch: 12:20 ± 0:02 h:min[c]	Lunch: 12:20 ± 0:02 h:min[d] 12:22 ± 0:02 h:min[e] 12:23 ± 0:02 h:min[f]	Lunch: 12:42 ± 0:02 h:min[g]	*P* <0.0001 *P* <0.001
		Dinner: 18:51 ± 0:06 h:min[c]	Dinner: 18:55 ± 0:05 h:min[d] 19:05 ± 0:05 h:min[e] 19:17 ± 0:05 h:min[f]	Dinner: 19:19 ± 0:05 h:min[g]	*P* <0.0001 *P* <0.01
(Vera et al. 2018)	Single 24-hour recalls	Breakfast: 8.34 ± 0.03 h	Not Reported	Breakfast: 8.65 ± 0.035 h	*P* < 0.05
		Lunch: 14.55 ± 0.02 h	Not Reported	Lunch: 14.59 ± 0.02 h	
		Dinner: 21.08 ± 0.06 h	Not Reported	Dinner: 21.39 ± 0.67 h	
		Midpoint of food intake: 14.80 ± 0.02 h	Not Reported	Later midpoint of food intake: 15.06 ± 0.02 h	*P* < 0.001

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) & other analysis
		Morning type	Intermediate type	Evening type	
(Silva et al. 2016)	Preliminary questionnaire	Not Reported	Not Reported	Not Reported	*Other analysis:* - Weak positive correlations between MSF score and breakfast time (r = 0.24, *P* < 0.001) ET were associated with a later breakfast time - Weak positive correlations between MSF score and lunch time ET associated with later lunch times (r = 0.19, *P* < 0.01)
(Lucassen et al. 2013)	Three-day food records	Breakfast on working days: 1 h and 20 min earlier than ET (7:17 ± 1:31) h: min	Not Reported	Breakfast on working days: (8:38 ± 1:52) h: min	*P* < 0.001
(Teixeira et al. 2018)	24-hour food recall (24h-FR) and questionnaire	Breakfast: 07:20 h: min	Breakfast: 07:45 h: min	Breakfast: 08:00 h: min	*P* < 0.001
		Lunch: 12:13 h: min	Lunch: 12:39 h: min	Lunch: 12:39 h: min	*P* = 0.02
(Baron et al. 2011)	7-day food logs	Not Reported	Breakfast: 9:07 h[c]	Breakfast: 11:53 h[d]	*P* < 0.001
		Not Reported	Lunch 13:07 h[c]	Lunch: 14:36 h[d]	P < 0.01
		Not Reported	Dinner: 19:07 h[c]	Dinner: 20:13 h[d]	*P* < 0.05
		Not Reported	Last meal/snack of the day: 20:25 h[c]	Last meal/snack of the day: 22:17 h[d]	*P* < 0.001
Time interval between meals & bedtime					
(Xiao et al. 2019)	The Automated Self-Administered 24-Hour Dietary Assessment Tool (ASA24)	Duration between dinner and bedtime (258± 58.6) min[a]	Not Reported	Duration between dinner and bedtime (313 ± 70.7) min[b]	*P* < 0.001

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) & other analysis
		Morning type	Intermediate type	Evening type	
(Baron et al. 2011)	7-day food logs	Not Reported	Time between breakfast and lunch: 4 h:01min [c]	Time between breakfast and lunch: 3h:15 min [d]	$P < 0.01$
		Not Reported	Time interval between last meal or snack, and sleep onset: 3 h:53 min [c]	Time interval between last meal or snack, and sleep onset: 5h:19 min [d]	$P < 0.05$
(Zerón-Rugerio et al. 2020)	6- day food logs & Quality Index Food Consumption Patter	Not Reported	Not Reported	Had on average, the shortest time between dinner and the midpoint of sleep (5.8 (0.9) h)[p]	$P > 0.001$
Eating duration					
(Sato-Mito et al. 2011)	Dietary history questionnaire	Breakfast: 17.38 ± 0.21 min:sec[c]	Breakfast: 17.11 ± 0.17 min:sec[d] 17.19 ± 0.19 min:sec[e] 16.38 ± 0.20 min:sec[f]	Breakfast: 19.03 ± 0.18 min:sec[g]	*P-Trend* = 0.0002 $P < 0.01$
		Lunch: 21.50 ± 0.23 min:sec[c]	Lunch: 22.07 ± 0.19 min:sec[d] 23.21 ± 0.20 min:sec[e] 22.41 ± 0.22 min:sec[f]	Lunch: 25.29 ± 0.19 min:sec[g]	$P < 0.0001$ $P < 0.001$
		Dinner: 28.45 ± 0.31 min:sec[c]	Dinner: 29.26 ± 0.26 min:sec[d] 30.20 ± 0.28 min:sec[e] 29.36 ± 0.30 min:sec[f]	Dinner: 32.29 ± 0.26 min:sec[g]	*P-Trend* <0.0001 $P < 0.001$

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
Skipped meals					
(Sato-Mito et al. 2011)	Dietary history questionnaire	Less skipped breakfast 0.66 ± 0.07 per week [c]	Breakfast: 0.57 ± 0.06 times/week[d] 0.91 ± 0.06 times/ week[e] 1.05 ± 0.06 times/ week[f]	More skipped breakfast: 1.91 ± 0.07 per week[g]	*P-Trend* <0.001
		Lunch: 0.15 ± 0.03 times per week[c]	Lunch: 0.16 ± 0.03 times per week 0.20 ± 0.03 times per week[e] 0.22 ± 0.03 times per week[f]	Lunch: 0.29 ± 0.03 times per week[g]	*P-Trend* = 0.0002
		Dinner: 0.26 ± 0.05 times per week[c]	Dinner: 0.29 ± 0.04 times per week[d] 0.29 ± 0.04 times per week[e] 0.33 ± 0.04 times per week[f]	Dinner: 0.42 ± 0.04 times per week[g]	*P-Trend* = 0.01
(Silva et al. 2016)	Preliminary questionnaire	MSF: 5.28	-	MSF: 6.19	*P* = 0.02 Breakfast skippers had higher MSF values (towards ET)
(Teixeira et al. 2018)	24-hour food recall (24h-FR) and questionnaire	Frequency of breakfast skippers: 10.0 (15%)	Frequency of breakfast skippers: 14.1 (63%)	Frequency of breakfast skippers: 21.8 (27%)	*P* = 0.02
				1.7 times more likely to skip breakfast (OR: 1.7; 95% CI: 1.1–2.9)	*P* = 0.02
				Breakfast skippers (ET) were associated with later lunch time (β−0.23, r^2 = 0.06)	*p* < 0.01

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
				Breakfast skippers (ET) were associated with later dinner time (β−0.17, $r^2 = 0.04$)	$p < 0.04$
TV watching during meals					
(Sato-Mito et al. 2011)	Dietary history questionnaire	Frequency per week during breakfast: 3.27 ± 0.08[c]	Frequency per week during breakfast: 3.52 ± 0.07[d] 3.50 ± 0.07[e] 3.59 ± 0.08[f]	Frequency / week during breakfast: 3.55 ± 0.07[g]	*P-Trend* = 0.03 *P* < 0.05
		Frequency per week during lunch: 1.25 ± 0.08[c]	Frequency per week during lunch: 1.50 ± 0.07 [d] 1.89 ± 0.08 [e] 2.14 ± 0.08 [f]	Frequency / week during lunch: 3.24 ± 0.07[g]	*P-Trend* < 0.0001 *P* < 0.001
		Frequency per week during dinner: 3.63 ± 0.07[c]	Frequency / week during dinner: 3.87 ± 0.06 [d] 3.87 ± 0.07 [e] 3.97 ± 0.07 [f]	Frequency / week during dinner: 3.90 ± 0.06 [g]	*P-Trend* = 0.02 *P* < 0.05
(Vera et al. 2018)	Barriers to Weight-Loss checklist, The Emotional Eating Questionnaire & a 24-hour recall	Not Reported	Not Reported	Not Reported	*P* = 0.07 *Other analysis:* 1.2 times more likely OR: 1.23; CI 95%: [0.99;1.52]
Eating behaviour score & sub-categories					
(Vera et al. 2018)	Barriers to Weight-Loss	Total eating behaviour score 0.01 ± 0.25	Not Reported	Total eating behaviour score score: 1.93 ± 0.26	*P* < 0.001

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
	checklist, The Emotional Eating Questionnaire & a 24-hour recall				
(Lázár et al. 2012)	The English version of the Dutch Eating Behavior Questionnaire	Reported better restrained eating	Not Reported	Not Reported	*P* < 0 .044
		Reported better external eating behaviour	Not Reported	Not Reported	*P* < 0 .001
(Beaulieu et al. 2020)	TFEQ	Chronotype score not associated with TFEQ			P ≥ 0.117
Stress-related eating, control over intake & food cravings					
(Vera et al. 2018)	Barriers to Weight-Loss checklist, The Emotional Eating Questionnaire & single 24-hour recall	Lower emotional eating score: 11.85 ± 0.19	Not Reported	Higher emotional eating score 12.40 ± 0.19	*P* = 0.046
			Not Reported	Not Reported	*Other analysis:* Prone to stress-related eating OR: 1.27; CI 95%:1.04, 1.55; *P* = 0.02
			Not Reported	Not Reported	Problems controlling amounts of certain types of food OR: 1.31; CI 95%:1.08, 1,58; *P* = 0.01
			Not Reported	Not Reported	Feel less in control over diet when tired OR: 1.33; CI 95%: 1.10, 1,60; *P* = 0.003
			Not Reported	Not Reported	Experience specific food cravings OR: 1.20; CI 95%: 0.99, 1,45; *P* = 0.06
(Lázár et al. 2012)	The English version of the Dutch Eating Behavior Questionnaire	Not Reported	Not Reported	Emotional eating was associated with diurnal preference	*P* <0.05

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) & other analysis
		Morning type	Intermediate type	Evening type	
(Lai & Say 2013)	Craving of high-calorie foods questionnaire	Not Reported	Not Reported	Not Reported	*Other analysis:* High-calorie food craving was not correlated with ME score (r = 0.003; *P* = 0.92)
Portion sizes, number of servings & number of eating occasions					
(Vera et al. 2018)	Barriers to Weight-Loss checklist, The Emotional Eating Questionnaire & single 24-hour recall	Not Reported	Not Reported	Not Reported	*Other analysis:* - 1.4 times more likely to have larger portion sizes portion sizes OR: 1.44; CI 95%:1.18,1.77; *P* < 0.001 - 1.3 times more likely to have second servings OR: 1.27; CI 95%:1.04,1.56; *P* < 0.019
(Lucassen et al. 2013)		Eat more frequently during working days (4.9 ± 1.5 occasions)	Not Reported	Eat less frequently during working days (4.4 ± 1.5 occasions)	*P* = 0.18
		Number of eating occasions during non-working days (4.2 ± 1.2 occasions)	Not Reported	Eat less frequently during non-working days (4.3 ± 1.6 occasions)	*P* = 0.57
		Portion sizes during working days (461 ± 177 kcal)	Not Reported	Portion sizes during working (545± 219 kcal)	*P* = 0.07

Reference	Method of assessment	Differences between types			*P*-value (ET vs IT/MT) & other analysis
		Morning type	**Intermediate type**	**Evening type**	
		Portion sizes during non-working days (599 ± 273 kcal)	Not Reported	Portion sizes during non-working days (622 ± 380 kcal)	$P = 0.75$
		Lower food intake after 20:00 on working days: 299 ± 354 kcal (50% fewer calories in fewer eating occasions than ET)	Not Reported	Higher food intake after 20:00 on working days: 677 ± 460 kcal	$P < 0.001$
		Food intake after 20:00 on non-working days: 327 ± 354 kcal	Not Reported	Food intake after 20:00 on non-working days: 537 ± 480 kcal	$P = 0.028$
(Teixeira et al. 2018)	24-hour food recall (24h-FR) and questionnaire	4.7 ± 1.2 meals/d	4.5 ± 1.1 meals/d	4.6 ± 1.1 meals/d	$P = 0.44$
Junk, energy rich & high- fat foods					
(Vera et al. 2018)	Barriers to Weight-Loss checklist, The Emotional Eating Questionnaire & single 24-hour recall	Not Reported	Not Reported	Not Reported	*Other analysis:* 1.4 times more likely to have energy-rich foods OR:1.44; CI 95%:1.16, 1.78; $P = 0.001$
(Beaulieu et al. 2020)	Appetite ratings and food reward (validated diurnal Leeds Food Preference Questionnaire) were measured in	No differences between chronotypes with regards to liking for high-fat relative to low fat foods			$P = 0.66$ *Other analysis:* - No interaction between chronotype and meal timing (AM vs PM) with regards to liking for high-fat relative to low fat foods
					$P > 0.05$

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
	response to a standardised test meal	No differences between chronotypes with regards to wanting/desire for high-fat relative to low fat foods			*Other analysis:* - No relationship between MEQ score and liking for high-fat food ($r = -0.15$, $P > 0.05$) - Inverse association between MEQ score and desire for high-fat food. ET associated with greater wanting/desire ($P = 0.01$, $r = -0.42$)
		No differences between chronotype with regards to desire for high-fat relative to low fat foods			$P > 0.05$ *Other analysis:* No interaction between chronotype and meal timing regarding desire for high-fat relative to low fat foods
Other					
(Vera et al. 2018)	Barriers to Weight-Loss checklist, The Emotional Eating Questionnaire & single 24-hour recall	Not Reported	Not Reported	Not Reported	*Other analysis:* 1.3 times more likely to eat in places other than the kitchen/dining room OR: 1.32; CI 95%: 0.99; 1.52; $P = 0.02$
		Not Reported	Not Reported	Not Reported	1.3 times more likely to eat directly from packets and containers OR: 1.31; CI 95%:1.06; 1.62; $P = 0.01$
(Beaulieu et al. 2020)	Appetite ratings and food reward (validated diurnal Leeds Food Preference Questionnaire) were measured in response to a	No differences in ratings of sweetness or pleasantness between the different meal timings (AM vs PM) and chronotype			$P \geq 0.157$ *Other analysis:* No interactions between meal timing (AM vs PM) and chronotype with regards to ratings of sweetness or pleasantness
		No differences between chronotype and prospective test meal consumption			*Other analysis:*

Reference	Method of assessment	Differences between types Morning type	Intermediate type	Evening type	*P*-value (ET vs IT/MT) & other analysis
	standardised test meal.				No interaction between meal timing (AM vs PM) and chronotype with regards to prospective test meal consumption
		No differences between chronotype and savory perception of test meal and no interaction between meal timing (AM vs PM) and chronotype with regards to savory perception			$P \geq 0.26$ *Other analysis:* No interactions among meal timing (AM vs PM), appetite ratings, chronotype and timepoint for test meal
		No differences between chronotype and appetite ratings			$P > 0.05$
		No interactions among meal timing (AM vs PM), chronotype, appetite ratings and timepoint (of test meal session)			*Other analysis:* No interaction between meal timing (AM vs PM), chronotype and perceived test meal fillingness
		Perceived test meal fillingness was higher	Not Reported	Not Reported	$P \leq 0.04$

[1] MD, Mediterranean Diet, MSFsc, Mid-sleep midsleep corrected for sleep duration on free days, NC, Neck circumference, PREDIMED, Prevención con Dieta Mediterránea, P-Trend refers to the continuous association between the MEQ or MCTQ score and exposures of interest, TDM, Elapsed time between dinner and the midpoint of sleep, TFEQ, Three-Factor Eating Questionnaire, Values reported as mean and standard deviation (SD) unless stated otherwise.

[2] [a]Early sleep was defined as a chronotype earlier than the median (3:04 AM), [b]late sleep was defined as a chronotype later than the median, [c]Based on earliest midpoint of sleep quintiles, [d]Based on midpoint of sleep quintiles 2, [e]Based on midpoint of sleep quintiles 3, [f]Based on midpoint of sleep quintiles 4, [g]Based on latest midpoint of sleep quintiles, [h]Based on MEQ score tertial 1: 34–53, [i]Based on MEQ score tertial 2: 54–59, [j]Based on MEQ score tertial 3: 60–76, [k]Based on normal sleep timing (midpoint 4:08 am), [l]Based on late sleep timing (midpoint of sleep 7:15 am), [m]Based on wakeup time <7:52 h and early-bedtime <23:48 h and defined as early-bedtime/early-rise (EE), [n] based on early-bedtime (<23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as early-bedtime/late-rise (EL), [o] based on late-bedtime (≥23:48 h) and wakeup time (<7:52 h) and defined as late-bedtime/early-rise (LE),[p] based on late-bedtime (≥23:48 h) and late-rise (wakeup time ≥ 7:12 h) and defined as late-bedtime/late-rise (LL).

4.4 Discussion

The aim of this project was to systematically review the existing evidence that chronotype impacts on body composition, and biomarker outcomes, by also considering behavioural factors such dietary intake, eating habits/behaviour in healthy adults. In this scoping systematic review, we consistently found that ET when compared to MT were more likely to be overweight/obese. This finding may be linked to their irregular eating patterns and unhealthy eating behaviours that could lead to circadian misalignment. Both MT and ET had very similar dietary intakes (energy and macronutrients), however, clear differences were apparent regarding the distribution of food intake throughout the 24-hour day, skipping and timing of meals as well as diet quality (micronutrients, food groups and types), which may lead to body composition changes. Furthermore, ET displayed higher risk of metabolic disease (see **Figure 4.2** depicting the main outcomes).

Most of the studies that explored meal timing found that individuals tend to consume food based on preferences according to their chronotype (Vera et al. 2018, Mazri et al. 2020, Lotti et al. 2021, Phoi et al. 2021). In ET, most of their energy and macronutrient intake were distributed towards the biological night (Qin 2003) and clock times for meals were later than those of MT. The mechanisms of this chronotype-body composition relationship are yet to be fully explained; however, it may be hypothesised and in part supported from data of this systematic review that several interconnected mechanisms, including mistimed food intake, lower diet quality and eating behaviours that favour weight gain and metabolic alterations have an influence.

Meal skipping, and especially breakfast skipping, was also prevalent in ET. These irregular eating patterns, including later timing of food intake and skipping of meals, seen in ET may be explained by their later preferred sleep and wake timing (Baron et al. 2011, Sato-Mito et al. 2011, Kanerva et al. 2012, Reutrakul et al. 2014, Muñoz et al. 2017), and are often in conflict with work time obligations or social demands (Dashti et al. 2015). Those extreme ET may experience significant misalignment between their internal circadian rhythms and their work hours as well as social demands. For example, during the week, ET have to wake up early for work and subsequently go to sleep earlier, in contrast with their internal timing, but during the weekends, they stay up longer and wake up later. This difference between sleep and wake times during the week and the weekend has been termed ‘social jetlag’ (Wittmann et al. 2006) and may result in adverse health outcomes such as greater risks for obesity and adverse metabolic health outcomes (Covassin et al. 2016).

Such results are exacerbated by too short sleep durations during the week as it often occurs in ET since they are more prone to accumulate sleep debt throughout their work week and consequently attempt to resolve this by altering their sleep schedules over the weekend, resulting in a higher social jetlag, and altered circadian rhythms (Wittmann et al. 2006, Roenneberg et al. 2012). Forced early wake times (for

school or work), as often found in ET, especially in teenagers and young adults, may then lead to redistribution or "catch-up" of the skipped meals to later in the day, because `normal' breakfast times would still be closer to their biological night, which is supported similar to the findings from this review. This may support the popular breakfast skipping theory, which pose those individuals who omit breakfast tend to be hungrier later in the day, leading to an overcompensation of energy intake, especially during the evening (Stanton Jr 1989). This occurs despite ET and MT still consuming the same amount of food within 24 hours (Meule et al. 2012, Dashti et al. 2015). According to Manoogian and Panda (2017), the external cues of feeding and fasting can impact metabolic processes. If these cues are disrupted, it can lead to increased risk of disease. Since ET eat closer to their bedtime, their fasting period is shortened, which may be more detrimental to their health, potentially delaying digestion. In time restricted eating (TRE) studies it was demonstrated that longer fasting periods are more beneficial for health than shorter ones (Manoogian & Panda 2017).

Metabolically, ingested calories are optimally used during the morning, possibly due to the higher thermic effect of food in comparison with the evening. When healthy individuals omit breakfast, they are in a fasted state at the beginning of their biological day (Yoshida et al. 2012). Consequently, the overnight fasting period is prolonged and an increase in postprandial insulin concentrations and fat oxidation is seen (Nas et al. 2017). Ultimately low-grade inflammation and impaired glucose metabolism may result in the development of metabolic inflexibility and weight gain (Nas et al. 2017). This review found that MT were more likely to have more regular eating patterns, and this have been linked by another researcher to higher postprandial thermogenesis and lower fasting and total LDL-cholesterol (Scheer et al. 2009). This highlights the endogenous circadian control of metabolic responses and the importance of meal timing. Glucose tolerance changes during the day, peaking during the day light hours when food is normally eaten, and troughs during the night when fasting occurs. Therefore, if ET shifts their eating to later in the day than MT, they may develop poor glycaemic control (Henry, et al. 2020). In comparison, this review found that ET were at higher risk of adverse metabolic health as shown by their lower HDL-cholesterol and higher triglycerides concentrations, higher metabolic syndrome scores (Vera et al. 2018), urinary epinephrine concentrations (Lucassen et al. 2013, Vera et al. 2018), insulin concentrations and HOMA-IR (Lázár et al. 2012), similar to the findings of Lotti et al. (2021). Other studies have also linked hormonal alterations, altered glucose metabolism, in conjunction with misaligned eating, as further mechanisms by which ET are more likely to become overweight/obese and are also at higher risk for pre-clinical states of diabetes (Deyoung, et al. 2007, Hogben et al. 2007, Schubert & Randler 2008, Broms et al. 2011).

These findings align well with previous studies (not chronotype focussed) that have linked aspects of mistimed food intake in humans such as breakfast skipping, late lunch eating and higher energy intake

at dinner with indicators of obesity (Aronoff et al. 2001, Horikawa et al. 2011, Bo 2014, Kutsuma et al. 2014, Wang et al. 2014).

Energy requirements and the oxidation of macronutrients varies across the 24-hour light-dark cycle (Rácz et al. 2018), consequently the timing of food intake has different effects on energy utilisation and as a result may change weight loss effectiveness and body composition (Garaulet et al. 2013, Jakubowicz et al. 2017). In this systematic review only one study found that consuming a higher amount of energy and proportion of carbohydrates and protein in the morning and during the early part of the day, seemed to be protective against developing obesity in MT. Consuming more energy, protein and carbohydrates towards the evening in ET was found to favour weight gain and obesity (Xiao et al. 2019). This reinforced previous studies that showed the detrimental effect of late eating (Garaulet et al. 2013). Generally, it seems that the timing of eating in alignment with one's chronotype, could be an important and beneficial factor when considering body composition outcomes (Muñoz et al. 2020). When participants of the latter study were following chronotype-adjusted diets, they had greater weight loss success compared with the traditional, hypocaloric control diet. The weight loss between MT and ET was similar, suggesting that this may be an effective approach for any chronotype (Muñoz et al. 2020). Besides chronotype, there are other factors which also target the circadian metabolic functions (including effective weight loss and reducing fat mass in obese adults) for example time restricted eating (TRE), which has also been widely known for its beneficial effects on cardiometabolic health (including lipid profiles and glucose concentrations) in humans (Adafer et al. 2020).

Eating behaviours may alter energy intake by influencing the types and amounts of foods eaten, timing of food intake and where food intake occurs, ultimately affecting body weight (French et al. 2012). The ET often displayed unhealthy eating behaviours such as consuming larger portion sizes, second servings, experiencing more food cravings, emotional- and stress-related eating, and presenting TFEQ scores that reflect unhealthy eating behaviours (Lázár et al. 2012, Vera et al. 2018). Studies have found that emotional eaters consumed snacks more often than non-emotional eaters. This suggests that there is a link between diet and body weight that may be mediated in part by dieting behaviour. In comparison, MT showed greater control of their eating behaviours (Lázár et al. 2012). MT had higher restraint scores (Lázár et al. 2012), which has been linked by other researchers to a higher consumption of "healthy food" such as green vegetables and fewer energy dense foods such as fats (Fleurbaix Laventie Ville Sante Study Group 2004), which was also seen in this review.

Other studies showed that insufficient sleep (shorter sleep duration) and being an ET impaired the appetite-regulating hormone leptin (Knutson 2007) as well as increase insulin concentrations (Nguyen & Wright 2010), which may lead to insulin resistance. Therefore, chronic misalignment of the circadian clocks lead to elevated leptin levels during the day and night, possibly due to over-secretion and leptin

resistance, which is a vicious circle because in turn, it can contribute again to overeating (Nguyen & Wright 2010).

These often-unhealthy eating behaviours among ET are further exaggerated by the higher intake of unhealthy, stimulating and energy-dense foods (Baron et al. 2011, Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Vera et al. 2018, Xiao et al. 2019, Najem et al. 2020, Muscogiuri et al. 2020). Exploring food group intake differences between chronotypes gives an idea of the micronutrient intakes. The fruit and vegetables intakes of ET found in this review is far below the World Health Organization guidelines of five servings per day (WHO 2020) and may account for the lower micronutrient intake reported by Sato-Mito et al. (2011). The ET also consumed more caffeine and alcohol. The stimulating effects of these items may account for the higher intake (Peuhkuri et al. 2012). In comparison, MT reported healthier, nutrient-dense food choices (Sato-Mito et al. 2011, Maukonen et al. 2017, Yoshizaki et al. 2018). They also displayed more control over their eating behaviour, which may account for the higher prevalence of a normal BMI in MT.

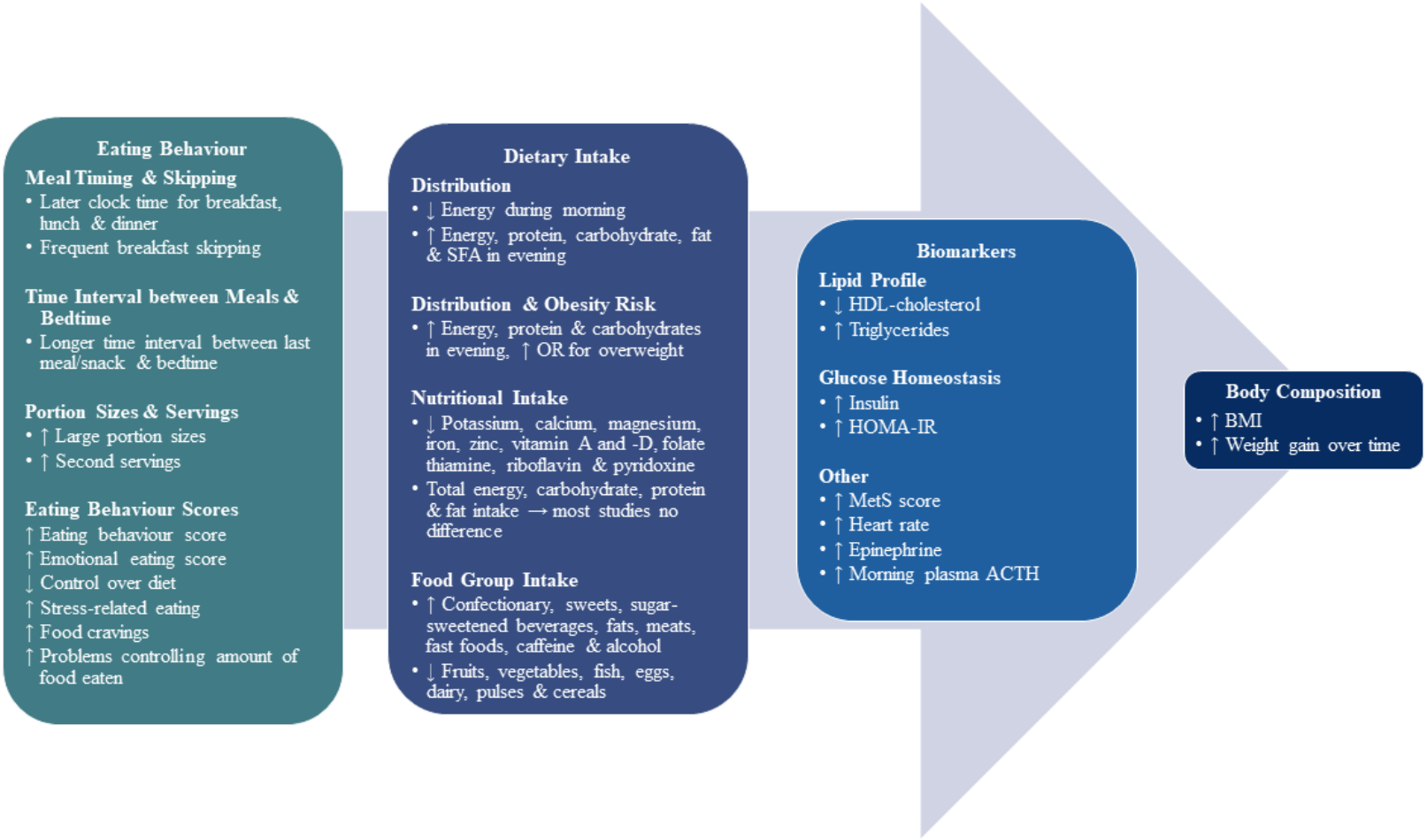

Figure 4.2 *- Main outcomes - late chronotypes (evening types) in comparison to early chronotypes (morning types)*

(ACTH) adrenocorticotropic hormone; (BMI) Body mass index; (HDL-Cholesterol) High Density Lipoprotein Cholesterol; (HOMA-IR) Homeostatic Model Assessment – Insulin Resistance; (MetS) Metabolic Syndrome; (OR) Odds Ratio; (SFA) Saturated Fat Acids.

4.5 Strengths & limitations

To our knowledge, this is the most comprehensive review of different dietary elements, including nutrients and energy, but also taking into account food intake, eating patterns, timing and composition of meals that compare extreme chronotypes with different body composition. This review has several strengths: Firstly, we included not only dietary intake (energy and macronutrient intakes) and body composition profiles but also eating and behavioural aspects and biomarkers as outcomes for different chronotypes. Secondly, the formatting of the data tables to compare the different chronotypes allowed a more comprehensive review of the literature, rather than only listing the specific outcomes. Another strength of this systematic review is that it did not include studies that recruited participants with acute, pre-existing and chronic diseases, which may have influenced chronotype.

One limitation of this systematic review was that included studies varied considerably in their classification method of chronotypes, the statistical analysis approach and study design, which made comparisons between chronotypes challenging. Another limitation was that not all the included studies were designed to assess differences between body composition groups as their main comparator.

4.6 Recommendations for research

Further studies are needed to explore interindividual and personalized optimal meal timing and the distribution of macronutrients at eating occasions with regards to weight management. Such optimization could be alleviated by assessment of the person's chronotype. In the same vein, the pathogenesis of obesity is not yet fully understood despite decades of research dedicated to the investigation of the underlying mechanisms and the development of successful interventions and treatments. Thus, it is crucial to add emerging evidence that consideration of extreme chronotypes and circadian misalignment is a contributing factor. More research is required to establish whether food intake that is in "misalignment with one's chronotype" is an important factor to consider (and potentially correct) in weight management. It should also be explored whether food intake should be adapted to chronotype-related wake and sleep-wake timing or whether food intake should simply be prioritized to "day" hours and limited during "night" hours.

4.7 Conclusions

This systematic review showed that ET were more likely to be overweight/obese and have poorer metabolic health in comparison with MT (and IT). It also highlighted key areas for clarification; firstly, this review found limited evidence of detailed assessment of diet quality, micronutrient intakes, food choices and quantities consumed between chronotypes, as well as inadequate focus on timing of intake, or investigating both in conjunction with eating and other eating behaviours. Such data could inform

strategies (for example, eating in alignment with internal body clocks; improvement of sleep timing and quality; adjusting mealtimes to improve the eating and fasting windows, e.g., time restricted eating) around healthy weight management in the future. This systematic review supports the assumptions that chronotype have an impact on body composition through interconnected mechanisms including mistimed food intake; eating behaviours and food choices that favour weight gain, as well as metabolic alterations.

The systematic review provided vital background knowledge and identified the research gaps that guided the statistical analysis of the PROMIsE data and the direction of the subsequent chapters.

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5 Chapter 5: Chronotype differences in timing of dietary intake, body composition & metabolic biomarkers

Abstract

Background and aims: Meal timing and composition are important zeitgebers (time cues) for circadian timing which may be influenced by chronotype. Eating at clock times misaligned with internal body clocks may result in poor metabolic health outcomes. This study aimed to investigate if chronotype is associated with body composition, eating patterns (meal timing and composition) and metabolic biomarkers among NZ European and Pacific women.

Methods: Healthy women (*n* = 287; 130 Pacific and 157 New Zealand (NZ) European, 18-45 years) were included in the final analysis. Height and weight were measured to calculate BMI. Whole body fat percentage (BF%), android and gynoid fat percentage were assessed using dual-energy x-ray absorptiometry. Chronotype was assessed using the Munich Chronotype Questionnaire. Dietary intake (energy, macro- and micronutrients) was assessed using five-day estimated food records. Fasting venous blood samples were collected to assess metabolic biomarkers (lipid profile, glucose- and hormonal control).

Results: Most participants (54 %) were classified as intermediate chronotypes (IT; n = 155), 34 % (n = 97) as evening types (ET), and 12 % as morning types (MT; n = 35). The ET versus earlier chronotypes (MT-IT) had a higher body composition profile: higher BMI (31.4 vs 26.1 kg/m^2), BF% (36 vs 34 %), and android to gynoid fat percentage (AG) ratio (0.98 vs 0.87). The ET had a poorer lipid profile and glucose homeostasis. Daily energy intakes were higher in ET (8745 kJ) vs MT-IT (8532 kJ, *P* = 0.03). Distinct differences were evident in the distribution of dietary intakes across the day: MT-IT vs ET had higher intakes of energy, protein, carbohydrate, and fat in the morning by 10:00, while ET had a higher energy, protein, carbohydrate, and fat intakes in the evening after 20:00 (P <0.05). Being an ET in the high BF% and high AG ratio group were more likely to have lower energy (β = -513 and β = -327,), protein (β = -4.48, and β = -3.14) and carbohydrate (β = -10.7 and β = -9.38) intakes by 10:00 (*P*<0.05), and higher energy (β = 603 and β = 684), carbohydrate (β = 12.9 and β = 16.3) and fat (β = 7.25 and β = 7.50) intakes after 20:00.

Conclusion: The results from this study showed that chronotypes consume similar energy and macronutrient intakes. Lower diet quality was seen the ET, who consumed lower micronutrient containing food in comparison with earlier types. The ET had a lower dietary intake in the morning versus higher evening and night intakes, with an inverse pattern seen among MT-IT. The high dietary intake in the evening or late at night-time was linked to higher BF% and AG ratio and unhealthy metabolic profile in ET.

Key words: Obesity; Adiposity; Circadian; Biomarkers; Metabolic Health; Morningness, Eveningness; Meal Timing; Macronutrient Distribution

5.1 Introduction

Humans show circadian preferences for the timing of their sleep and wakefulness, within the normal 24-hour day. The term chronotype (Roenneberg et al. 2003) was coined to describe the preferred sleep-wake timing of an individual relative to the light-dark cycle that influences the timing of their diurnal preferences and the modulation of physiological functions and behaviour (Ehret 1974, Horne 1976, Adan et al 2012). Different chronotypes have different phases angles of entrainment with regards to their sleep and wake timing relative to external clock times (Roenneberg et al. 2007). The sleep and wake times in early chronotypes (=morning type; MT) is many hours advanced in comparison with a late chronotype (=evening type; ET) (Lack et al. 2009). Chronotype therefore drives not only sleep and wake times (Duffy et al. 2001), but likely also the timing of food intake (fasting or eating). Not surprisingly, extreme chronotypes may experience marked misalignment between their internal body clocks and daytime work and social demands (Wittmann et al. 2006) that may result in adverse health outcomes (Covassin et al. 2016), including weight gain and obesity (Merikanto et al. 2013, Covassin et al. 2016).

Obesity is a complex disease, with many biological, environmental and socio-economic factors (Blüher 2019) involved in its aetiology. Globally the prevalence of obesity has tripled since 1975 (World Health Organization 2021) and in NZ one in every four adults are affected. There is a strong relationship between obesity and ethnicity, with 87% of Pacific peoples being affected in comparison with 65% of the rest of the population (Ministry of Health 2019, Ministry of Health 2020). The aetiology of obesity is not fully understood despite decades of research dedicated to the investigation of underlying mechanisms and the development of successful interventions (Taubes et al. 2013, Schwartz et al. 2017). A growing number of studies have reported a link between the circadian timing system and especially the influence of chronotype in obesity and dietary outcomes (Sato-Mito et al 2011, Kanerva et al. 2012, Maukonen et al. 2016, Maukonen et al. 2017, Muñoz et al. 2017, Maukonen et al. 2019). It has been shown that most metabolic processes such as appetite, nutrient metabolism, digestion, hormonal regulation, as well as various behavioural aspects including food intake, sleep and wake timing (Bass 2012, Jiang & Turek 2017), are under circadian control. The internal time keeping system requires an external zeitgeber ('time cue') to entrain the circadian clocks with the external environment (Aschoff 1965, Moore 1973, Roenneberg et al. 2003, Ko & Takahashi 2006, Bass 2010, Garaulet & Madrid 2010, Huang et al. 2011). While light is the primary zeitgeber for the central circadian clock, other external cues such as the timing and composition of food intake are also capable of setting the rhythms in the peripheral clocks (Bass 2012, Youngstedt et al. 2016, Jiang & Turek 2017). Although a zeitgeber can aid in resetting the circadian clock, it can also become a 'desynchroniser', when experienced at the incorrect time of the daily cycle (Boivin & Czeisler 1998, Reppert & Weaver 2002, Salgado-Delgado

et al. 2010 , Bass 2012, Youngstedt et al. 2016, Jiang & Turek 2017, Nelson & Chbeir 2018, Challet 2019, Garaulet 2020).

In recent years, more studies have examined the relationship between chronotype and dietary intake, with a main focus only on absolute food intake (Sato-Mito et al 2011, Sato-Mito et al 2011, Kanerva et al. 2012, Maukonen et al. 2016, Mota 2016, Li et al. 2018, Teixeira et al. 2018, Yoshizaki 2018, Zeron-Rugerio et al. 2019, Najem et al. 2020) and to a lesser extent on distributions of energy (Baron et al. 2011, Lucassen et al. 2013, Maukonen et al. 2017, Muñoz et al. 2017, Maukonen et al. 2019, Zerón-Rugerio et al. 2020) and macronutrient intake (Baron et al. 2013, Maukonen et al. 2017, Muñoz et al. 2017, Xiao et al. 2019) throughout the day. There is clear evidence that macronutrient metabolism and energy expenditure vary across the 24-hour light-dark cycle (Roberts 1964, Takahashi et al. 1968, Van Cauter et al. 1997, Saad et al. 2012, Cipolla-Neto et al. 2014, Rácz et al. 2018) as evident by various metabolic processes and endocrine regulation that exhibit a marked morning-evening difference (Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Covassin et al. 2016). Indeed, our recent systematic review found that clock times for main meals as well as the energy and macronutrient distribution in the morning and evening varied between chronotypes (van der Merwe et al. 2022). However, differences in energy and macronutrient distribution were reported by a limited number of included studies (Xiao et al. 2019). Consequently, not only the timing of food intake but also differences in energy and macronutrient distributions throughout the day have different effects on energy utilisation (Garaulet et al. 2013, Jakubowicz et al. 2013, Jakubowicz et al. 2017). Together this may affect body composition and metabolic health outcomes differently between chronotypes.

A limited number of studies have explored the effect of energy and macronutrient distribution on body composition between chronotypes and those that did, have yielded contrasting results (Muñoz et al. 2017, Xiao et al. 2019). These studies primarily used BMI as a measure of body composition (Muñoz et al. 2017, Xiao et al. 2019). Although BMI is a useful adiposity measure in population-based studies (De Lorenzo et al. 2016), it does not reliably reflect body composition, metabolic- and fat mass disease risk on an individual level (De Lorenzo et al. 2016). Especially in a group with a high fat free mass (FFM), such as in Pacific women which is 2.9 kg higher than in NZ European women (Rush et al. 2007). It is therefore suggested that in conjunction with BMI, BF% should be measured to assess adiposity (De Lorenzo et al. 2016, von Hurst et al. 2016). Furthermore, in addition to total BF%, assessment of android, gynoid and visceral fat percentage should be considered to provide an indication of metabolic health. For example, higher central obesity is associated with higher metabolic- and cardiovascular disease risk (Sun et al. 2010). These findings can further be linked with associated biomarkers, for example among chronotypes in a healthy population (Lázár et al. 2012, Lucassen et al. 2013, Vera et al. 2018, Muñoz et al. 2020) associations between ET and a poor lipid profile (including higher triglycerides and lower HDL-cholesterol concentrations) (Muñoz et al. 2017, Vera et al. 2018)

and glucose homeostasis (Vera et al. 2018) were reported, suggesting a link between chronotype and metabolic health.

Since MT may be advanced (earlier) in terms of their sleep and wake timing and ET delayed (later) relative to IT, (Roenneberg et al. 2007) optimal meal timing may differ among individuals based on their chronotype (Xiao et al. 2019). It is also possible that food intake should simply be prioritised to "day" hours and limited during "night" hours, because ingested nutrients and energy are better metabolised during the day (Roberts 1964, Takahashi et al. 1968, Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Saad et al. 2012, Cipolla-Neto et al. 2014, Covassin et al. 2016 , Rácz et al. 2018). A clear link between chronotype, the diet, body composition and biomarkers are yet to be established. Therefore, the aim of this study was to investigate if chronotype is associated with body composition, eating patterns (nutrient composition; intake timing) and metabolic biomarkers in NZ European and Pacific women. Objectives were to investigate (1) differences in body composition, biomarkers and total dietary intake between chronotypes, (2) differences in the energy and macronutrient distribution throughout the 24-hour day between chronotypes, (3) possible associations between energy and macronutrient distribution and chronotype within body composition groups (BF% and the distribution of adiposity), and (4) associations between chronotype, metabolic biomarkers, body composition parameters and timing of energy intake.

5.2 Methods

5.2.1 Study design, participants & procedure

This cross-sectional study recruited 304 healthy women through web-based advertisements, at cultural festivals, university and work partnerships and email lists from Auckland, NZ. Participants were selected to be of Pacific (n = 162) and New Zealand (NZ) European (n = 142) ethnicity, between the ages of 18 and 45 years and had to have a BMI <25 or ≥30 kg/m^2. During screening, participants' self-reported height, weight and BMI were used for recruitment. However, during the study, measured height and weight were used to determine actual BMI groups stratified as low <25 (normal BMI) and high ≥25 kg/m^2 (overweight and obese BMI). Participants were excluded if they were pregnant or lactating; diagnosed with chronic illness (e.g., diabetes and cardiovascular disease); had undergone bariatric surgery; had severe food allergies; used medication that could interfere with appetite or the immune system (e.g., appetite suppressants and corticosteroids); were current smokers or had severe dietary restrictions or avoidances (e.g., vegan). Participants visited the Massey University Human Nutrition Research Unit in Auckland, NZ, between July 2016 and September 2017 for data collection. Written informed consent was obtained from all participants. Ethics approval was obtained from the Southern Health and Disability Ethics Committee (16/STH/32) (Kindleysides et al. 2019). The participants attended two visits, up to 14 days apart; visit one included blood sampling (metabolic

biomarkers), body composition measurements (height, weight, BF analysis measured by bioelectrical impedance analysis (BIA)), completion of questionnaires (MCTQ, retrospective seven-day, sleep, and work questionnaire) as well as an interview for demographic information. At-home data collection between study visits included a five-day food record (5DFR) and visit two included FR returns, body composition measurements (weight, WC, HC, DXA measurements) and a one-on-one food record review interview with a dietitian. Full details on study procedures have been reported elsewhere (Kindleysides et al. 2019). Procedures related to this analysis are briefly described below.

5.2.2 Demographic information & deprivation index

Demographic information, including health information (disease conditions and medication use), age and work patterns was collected during a one-by-one interview with a researcher during the first visit. The NZ Deprivation Index 2013 (NZDep2013) was used as a measure of socio-economic status. The NZDep2013 is determined by combining census information such as home ownership, housing, qualifications, employment, income, access to transport, communications, and family structure. The deprivation scores are grouped into deciles, where decile one represents the geographical area with the least deprived scores and decile 10 the areas with the most deprived scores (Crampton et al. 2020).

5.2.3 Anthropometrics measurements

All anthropometrics measurements were conducted according to the International Society for the Advancement of Kinanthropometry (ISAK) protocols by level one ISAK trained research staff (Stewart et al. 2011). Height and weight were used in the Quetelet index (weight/height^2) to determine BMI. Waist- and hip circumferences were measured with a Lufkin W600PM flexible steel tape with the participant in a relaxed standing position with their arms folded across their chest and used to determine waist-to-hip (WHR) and waist-to-height ratio (WHtR) respectively. Dual-energy x-ray absorptiometry (DXA; Hologic QDR Discovery A, Hologic Inc with APEX V. 3.2 software) was used to assess lean body mass (kg), whole body total fat mass (kg), percentage android-, gynoid- and visceral fat (calculated by DXA software and reviewed by two trained researchers who modified the regions if required), and android to gynoid percentage fat (AG) ratio (De Lorenzo et al. 2016). Fat mass (FM) and fat free mass (FFM) was measured by DXA, and Bioelectrical impedance analysis and used as FM:FFM ratio. Participants with a larger body frame that did not fit within the scanning parameters of the DXA, had two scans taken. The first scan was set up to include the whole body with one arm outside the range, and the second scan was then taken to include the arm that was incomplete on the first scan. The lean and fat mass regions that were calculated by the DXA software for each of the two scans were manually consolidated using standard procedures.

5.2.4 Chronotype, sleep & work schedules

A retrospective seven-day, sleep and work questionnaire was used to collect information on sleep and work schedules to identify the number of hours worked per day and whether shifts were worked (day or night). Participants were excluded from the analyses if they reported night shift work (n = 17). The Munich Chronotype Questionnaire (MCTQ) that was developed by Roenneberg et.al. was used to subjectively assess an individual's chronotype using self-reported sleep timing (bed- and wake times and estimated time to fall asleep) on workdays and work-free days within the last four weeks (Roenneberg et al. 2007). The questionnaire was completed online (using SurveyMonkey) during the first visit. From sleep times, midsleep (which is the midpoint between sleep onset and wake time) was calculated separately for workdays (MSW) and work-free days (MSF) and weighted to obtain midsleep, corrected for sleep duration on free days (MSFsc). The MSFsc was used as a proxy for chronotype, where MSFsc scores greater than five (i.e., midsleep occurs after 05:00) reflect late chronotypes (ET) and scores smaller than three (i.e., midsleep occurs before 03:00) reflect earlier chronotypes (MT) (Roenneberg et al. 2003).

5.2.5 Dietary intake

Dietary intake for energy, macro- and micronutrient intakes, and distribution of food intake throughout the day (clock times) were assessed by using written estimated five-day food records (5DFR) (non-consecutive) that included two weekend- and three weekdays. Participants received training for estimating and documenting their portion sizes. The 5DFR was reviewed by a NZ registered dietitian followed by a one-on-one, in-depth discussion with the participant to clarify portions, cooking methods, and brands of food products reported. A visual portion book aid, household measures (e.g., metric cups and spoons), and web-based tools were used to confirm specific portion sizes and brands consumed (Kindleysides et al. 2019). Data were entered manually into the computer software, FoodWorks 10 (Xyris Software (Australia) Pty Ltd, Queensland, Australia) and the NZ food composition database (NZ FOODFiles 2016) was used to analyse the nutrient (energy, macro- and micronutrient) content of the 5DFR (Kindleysides et al. 2019). Due to the multitude of food items consumed, if no match for any item could be found in the NZ databases, food composition was sought from AusFoods 2017 (AUSNUT 2011-13 food composition files), developed by Food Standards Australia New Zealand.

5.2.6 Blood samples

Following an overnight fast of at least 10 hours, fasting venous blood samples were collected by a trained phlebotomist during the first study visit. Metabolic biomarkers related to lipid profiles, glucose and hormonal control were assessed. Collection and processing of the blood samples has been reported in detail elsewhere (Kindleysides et al. 2019). Homeostasis model assessment (HOMA-IR) index for

insulin resistance was calculated (fasting blood glucose [mmol/L] X fasting plasma insulin [μU/mL]/22.5).

5.2.7 Data processing & statistical data analysis

Analysis was conducted using IBM SPSS Statistics for Windows version 28 (Armonk, NY: IBM Corp) and a *P*-value of < 0.05 was accepted as statistically significant. Night shift workers (n = 17) were excluded from the analysis because these individuals have altered sleep-wake and fasting-feeding cycles due to work obligations and which are not necessarily representative of their chronotype. A total of 287 were included in the final analysis of which 157 were NZ European and 130 were Pacific women. To assess the association between chronotype, timing of dietary intake, body composition and biomarkers, the study participants were grouped as ET (MSFsc >5), IT (MSFsc = 3 - 5) and MT (MSFsc < 3). The MT and IT were merged into a MT-IT group for analysis due to the small sample size for MT. The study population was stratified according to low and high body composition groups; BF% into normal BF <35 % or high BF ≥35 % and AG ratio into normal < 0.8 or a high ≥0.8 AG ratio.

The distribution of the different variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests, box plots, and histograms. Normally distributed data were presented as means and standard deviations, non-normal distributed data as medians and 25th and 75th percentiles and categorical data as frequency summary statistics. Log transformed data values were back transformed to the original scale and reported as geometric mean [95 % confidence intervals (CI)]. The Welche's T-tests were used for normally distributed data and Mann-Whitney U tests for non-normal data when comparing two groups. When comparing more than two groups ANOVA tests (with Tukey HSD as post-hoc tests) were used for normally distributed data and Kruskal-Wallis tests (with multiple comparison rank test, adjusted by Bonferroni correction) for non-normally distributed data.

The clock time for the first and last eating occasion of the day was determined for each chronotype. An eating occasion was defined as 210 kJ that was at least fifteen minutes apart from the next eating occasion. This energy caption excluded low energy beverage-only eating occasions such as tea with milk. The first eating occasion was defined as the first 210 kJ consumed from wake time and the last eating occasion was defined as the last 210 kJ consumed before sleep onset (Leech et al. 2015, Kant 2018). From this information the overnight fasting period was determined for each chronotype (**Appendix C.1**).

The energy distribution across the 24-hour day was analysed by calculating the average hourly intakes for chronotype groups and statistical significance was determined with multiple comparison rank tests, adjusted by Bonferroni correction. Thereafter, four dietary windows were created (window 1: 03:00 – 09:59, window 2: 10:00 – 14:59, window 3: 15:00 – 19:59, window 4: 20:00 – 02:59) based on previous

studies (Baron et al 2011, Baron et al. 2013, Maukonen et al. 2017). Mean intakes were determined for each window. A two-way repeated measures ANCOVA was performed to compare the effect of chronotype on the distribution of mean energy and macronutrient intakes across the four windows (degrees of freedom was corrected using Greenhouse-Geisser estimates of sphericity). Analysis was adjusted for ethnicity, age, and deprivation index. Within windows 1 and 4, there were significant differences between chronotype groups and thereafter, all analyses included only those two windows as a morning intake by 10:00 and an evening intake after 20:00.

The association between chronotype and energy and macronutrient intakes in the morning by 10:00 and evening after 20:00 were assessed by multiple linear regression models (enter method) within normal (<35 %) and high BF% (≥35 %) groups, and then within normal (<0.8) and high (≥0.8) AG Ratio groups to reflect high body fat areas. The intake for each nutrient per time window was entered as the dependent variable and chronotypes (ET vs. MT-IT) as the independent variable. Further, Pearson's correlation analysis was conducted to assess the associations between MCTQ (individual midsleep times), energy intake in the morning by 10:00 and evening after 20:00, different biomarkers and body composition markers.

5.3 Results

5.3.1 Study participants

Table 5.1 depicts the descriptive statistics for each of the three chronotype groups independently as well as the combined MT-IT group. The majority of ET were of Pacific ethnicity (n = 83, 64 %, *P* <0.001), and the majority of the NZ Europeans were IT (n = 115, 73.2 %, *P* <0.01). The ET in comparison with MT-IT were younger (median of 23 vs 30 years, *P* <0.01), from higher socioeconomic deprived areas (8 (6,10) vs 5 (3,7), *P* <0.01), and had higher median body composition markers (*P* <0.05), including BMI (31.4 vs 26.1 kg/m^2), WC (88.8 vs 80.2 cm), HC (114 vs 107 cm), android fat % 37 vs 33.5 %) and visceral fat % (34.8 vs 31.4 %).

Table 5.1 *- Study participants characteristics*

Variable	Morning Type (n = 35)	Intermediate Type (n = 155)	Evening Type (n = 97)	*P*-value †	MT- IT† (n = 190)	*P*-value ᶻ
Demographics						
Age (years)	35 (29, 38)	29 (24, 37) [a]	23 (21, 26) [a, b]	**0.03**	30.0 (28.9, 31.0)	**<0.01**
NZ European n (%)	28 (17.8)	115 (73.2)	14 (8.9)	**<0.01**	143 (49.8)	**<0.01**
Pacific n (%)	7 (5.4)	40 (30.8)	83 (63.8)		47 (16.4)	
Deprivation score	5 (2, 6)	5 (3, 7)	8 (6, 10) [a, b]	**<0.01**	5 (3, 7)	**<0.01**
Physical activity	28.0 (15.8, 44.6)	28.4 (17.8, 40.3)	20.5 (11.0, 38.2) [b]	**0.04**	28.2 (17.6, 41.2)	**0.01**
Body composition						
BMI (kg/m^2)	24.2 (21.7, 31.4)	27.3 (22.7, 33.1)	31.4 (24.6, 36.8) [a, b]	**<0.01**	26.1 (22.7, 32.9)	**<0.01**
BMI Categories, n (%)						
<25 kg/m^2	19 (54)	66 (43)	26 (27)	**0.01**	85 (45)	**<0.01**
≥25 kg/m^2	16 (46)	89 (57)	71 (73)		105 (55)	
BF%	32 ± 9	34 ± 7	36 ± 7	**0.02** [a ¥]	34 ± 7	**0.02** [r]
BF% Categories, n (%)						
< 35%	23 (66)	69 (45)	45 (46)	0.07	92 (48)	0.75
≥ 35%	12 (34)	86 (55)	52 (54)		98 (52)	
WC (cm)	78.2 (72.1, 96.9)	81.5 (73.8, 96.1)	88.8 (78.0, 102) [a, b]	**0.01**	80.2 (73.2, 96.1)	**<0.01**
HC (cm)	102 (95.2, 117)	108 (99.7, 119)	114 (104, 124) [a, b]	**<0.01**	107 (99.1, 119)	**<0.01**
Android Fat (%)	29.5 (19.9, 39.4)	34.3 (25.5, 39.8)	37.0 (29.2, 42.7) [a, b]	**0.01**	33.5 (24.3, 39.6)	**<0.01**
Gynoid Fat (%)	35.7 ± 6.5	37.9 ± 5.7	38.8 ± 5.2 [a]	**0.02** ¥	37.5 ± 5.9	0.05[r]
Visceral Fat (%)	27.1 (17.1, 37.3)	32.2 (22.2, 38.3)	34.8 (27.6, 41.2) [a, b]	**<0.01**	31.4 (21.5, 38.1)	**<0.01**
AG Ratio	0.80 (0.69, 0.96)	0.87 (0.77, 0.98)	0.98 (0.81, 1.06) [a, b]	**<0.01**	0.87 (0.74, 0.98)	**<0.01**
AG Ratio groups n (%)						
<0.8	18 (51.4)	54 (34.8)	23 (23.7)	**<0.01**	72 (37.9)	**0.02**
≥ 0.8	17 (48.6)	101 (65.2)	74 (76.3)		118 (62.1)	

(MT-IT) Morning- and intermediate types combined group; (NZ) New Zealand European; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (WC) Waist Circumference, (HC) Hip Circumference, (AG) Android to Gynoid Fat Percentage Ratio.

Values are reported as Median (25th ,75th percentiles), means ±SD, or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale. Missing deprivation index (n = 3), †Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data [a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, ᶻMann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data; Chi-square test for categorical data; ¥One-way ANOVA for comparing three groups continuous, normally distributed data with Tukey HSD as post hoc test; [r] Independent T-test for comparing two continuous, normally distributed data.

In terms of metabolic biomarkers, the ET also had higher median concentrations of triglycerides (1.0 vs 0.8 mmol/L), leptin (15678 vs 10304 pg/mL, insulin (15.7 vs 9.5 uU/ml), and higher mean $HbA1_C$ (33.0 vs 31.5 mmol/L) than the MT-IT (P <0.05) (**Table 5.2**). On the other hand, the MT-IT had higher mean total cholesterol (5.0 vs 4.56 mmol/L), higher median HDL-cholesterol (1.60 vs 1.40 mmol/L), LDL-cholesterol (2.8 vs 3.01 mmol/L) and ghrelin (48.9 vs 28.1 pg/mL) concentrations in comparison with ET (P<0.05).

With regards to dietary intake, there was no difference in daily macronutrient intakes between the MT-IT and ET. However, ET had a 213 kJ higher daily mean energy intake vs MT-IT (8745 vs 8532 kJ, respectively, P = 0.03). In contrast, the MT-IT had higher median intakes of alcohol, fibre, caffeine, PUFA, vitamin A, riboflavin, folate, potassium, magnesium, calcium, iodine, and higher mean vitamin E.

***Table 5.2** - Metabolic biomarkers and dietary intake differences between chronotype groups*

Variable	Morning type (n = 35)	Intermediate type (n = 155)	Evening type (n = 97)	*P*-value †	MT-IT† (n = 190)	*P*-value ‡
Metabolic biomarkers						
Cholesterol (mmol/L)	5.07 [4.68, 5.50]	4.93 [4.79, 5.07]	4.56 [4.43, 4.70] [a, b]	**<0.01** [Ɣ]	5.0 ± 1.0	**<0.01**[r]
LDL-Cholesterol (mmol/L)	3.0 (2.4, 3.4)	3.1 (2.5, 3.6)	2.8 (2.4, 3.3)	0.06	3.0 (2.5, 3.6)	**0.02**
HDL-Cholesterol (mmol/L)	1.7 (1.4, 2.0)	1.6 (1.3, 1.9)	1.4 (1.2, 1.7) [a, b]	**<0.01**	1.6 (1.3, 1.9)	**<0.001**
Triglycerides (mmol/L)	0.8 (0.7, 1.2)	0.9 (0.6, 1.1)	1.0 (0.8, 1.2) [b]	**0.01**	0.8 (0.7, 1.1)	**<0.01**
HbA1c (mmol/L)	31.8 ± 2.7	31.6 ± 2.9	33.0 ± 3.1[b]	**0.01** [Ɣ]	31.5 [31.1, 31.9]	**<0.01**[r]
Insulin (uU/ml)	8.4 (6.0, 10.1)	10.1 (6.7, 14.9)	15.7 (10.2, 26.3) [a, b]	**<0.01**	9.5 (6.7, 14.7)	**<0.01**
Glucose (mmol/L)	5.2 (5.1, 5.3)	5.3 (5.3, 5.4)	5.4 (5.3, 5.5)	0.15	5.3 (5.0, 5.6)	0.16
Ghrelin (pg/mL)	74.8 (45.4, 120.8)	46.3 (22.1, 74.2) [a]	28.1 (17.3, 58.7) [a, b]	**<0.01**	48.9 (24.5, 83.6)	**<0.01**
Peptide YY (pg/mL)	120 (97, 148)	136 (114, 168)	135 (104, 168)	0.20	133 (111, 167)	0.78
Leptin (pg/mL)	7210 (2716, 14683)	11291 (5726, 20459)	15678 (7683, 23196) [a]	**0.01**	10304 (4895, 19708)	**0.01**
C-reactive protein (mg/L)	0.9 (0.3, 2.2)	1.2 (0.5, 2.8)	0.8 (0.3, 2.9) [b]	0.06	1.1 (0.5, 2.7)	0.10
Dietary intake						
Energy (kJ)	8553 ± 1702	8527 ± 2056	8745 ± 2577	0.74[Ɣ]	8532 ± 1992	**0.03**[r]
Protein (g)	85.6 ± 20.3	86.5 ± 21.1	82.4 ± 24.4	0.36 [Ɣ]	86.3 ± 20.9	0.17[r]
Fat (g)	89.8 ± 26.7	93.6 ± 30.6	89.7 ± 28.5	0.55 [Ɣ]	92.9 ± 29.9	0.31[r]
Carbohydrate (g)	196.9 ± 50.5	191.7 ± 63.9	223.1 ± 76.5 [b]	**<0.01** [Ɣ]	192.7 ± 61.6	0.05[r]
Sugar (g)	90.6 (65.8, 98.6)	77.5 (59.4, 97.7)	81.5 (62.5, 116)	0.28	79.0 (60.2, 98.3)	0.26
Fibre (g)	25.6 (17.3, 33.9)	22.4 (18.8, 27.2)	17.4 (14.2, 21.1) [a, b]	**<0.01**	22.5 (18.6, 28.1)	**<0.01**
MUFA (g)	31.3 (23.3, 42.1)	34.9 (25.9, 43.3)	32.7 (26.6, 40.1)	0.48	34.3 (25.6, 43.0)	0.54
PUFA (g)	1.6 (8.82, 19.1)	12.1 (9.52, 14.9)	11.3 (8.96, 13.5) [a]	0.05	12.2 (9.39, 15.5)	**0.02**
SFA (g)	30.9 (24.5, 42.6)	34.5 (26.8, 44.6)	33.6 (28.0, 43.6)	0.65	34.0 (26.2, 44.0)	0.96
Cholesterol (mg)	269 [220, 329]	293 [268, 320]	262 [238, 288]	0.24	289 [266, 313]	0.12[r]
Caffeine (mg)	117 (45.8, 282)	107 (37.9, 232)	54.4 (21.5, 120) [a, b]	**<0.01**	108 (38.6, 234)	**<0.01**
Alcohol (g)	0.32 (0.01, 11.0)	0.09 (0.00, 7.64)	0.03 (0.00, 0.32) [a]	**0.01**	0.09 (0.00, 7.64)	**0.01**
Vitamin A equivalents (µg)	793 (587, 1003)	819 (587, 1008)	511 (323, 783) [a, b]	**<0.01**	806 (587, 1007)	**<0.01**

Variable	Morning type (n = 35)	Intermediate type (n = 155)	Evening type (n = 97)	*P*-value [†]	MT-IT[†] (n = 190)	*P*-value [ǂ]
Thiamine (mg)	1.3 (1.0, 1.6)	1.2 (0.9, 1.5)	1.2 (0.9, 1.6)	0.62	1.2 (1.0 ,1.6)	0.93
Riboflavin (mg)	1.7 (1.5, 2.0)	1.8 (1.4, 2.2)	1.6 (1.2, 2.1) [b]	**0.04**	1.8 (1.4, 2.2)	**0.01**
Niacin equivalents (mg)	36.1 (30.0, 43.5)	35.1 (29.7, 39.9)	34.5 (27.2, 42.0)	0.72	35.2 (29.8, 40.2)	0.58
Vitamin B6 (mg)	2.0 (1.6, 2.6)	2.2 (1.7, 2.7)	2.0 (1.5, 2.8)	0.68	2.2 (1.6, 2.7)	0.45
Folate (µg)	330 (260, 416)	334 (256, 427)	256 (180, 351) [a, b]	**<0.01**	332 (256, 420)	**<0.01**
Vitamin B12 (µg)	3.7 (2.7, 4.3)	3.5 (2.7, 4.7)	3.8 (2.8, 4.8)	0.62	3.5 (2.7, 4.7)	0.35
Vitamin C (mg)	63.7 [50.0, 81.4]	62.4 [56.2, 69.3]	52.9 [44.7, 62.6]	0.18 [Ɣ]	62.7 [57.0, 69.0]	0.83[r]
Vitamin E (mg)	10.3 [9.03, 11.8]	9.27 [8.68, 9.90]	7.94 [7.30, 8.64] [a, b]	**<0.01** [Ɣ]	9.3 [7.6, 11.9]	**<0.01**
Sodium (mg)	2526 [2310, 2761]	2524 [2386, 2670]	2721 [2512, 2947]	0.25 [Ɣ]	2524 [2405, 2649]	0.06[r]
Potassium (mg)	3170 ± 809	2987 ± 762	2615 ± 832 [a, b]	**<0.01** [Ɣ]	3021 ± 772	**<0.01**[r]
Magnesium (mg)	354 (271, 442)	310 (265, 383)	262 (217, 301) [a, b]	**<0.01**	312 (265, 387)	**<0.01**
Calcium (mg)	810 (669, 1050)	809 (628, 980)	607 (491, 861)	**<0.01**	809 (635, 980)	**<0.01**
Iron (mg)	12.8 [11.6, 14.0]	11.1 [10.5, 11.7]	10.9 [10.2, 11,7] [a]	0.05 [Ɣ]	12.0 ± 3.8	0.29 [r]
Zinc (mg)	10.9 (9.0, 12.9)	10.1 (8.6, 11.8)	9.6 (8.3, 12.0)	0.27	10.2 (8.8, 11.8)	0.19
Selenium (µg)	56.3 [49.1, 64.5]	60.7 [56.7, 65,0]	53.7 [48.8, 59.1]	0.09 [Ɣ]	59.9 [56.3, 63.6]	0.06[r]
Iodine (µg)	110 (85.0, 133)	93.8 (73.7, 115)	82.7 (58.6, 114) [a, b]	**<0.01**	95.4 (74.4, 126)	**<0.01**

(MT-IT) Morning- and intermediate types combined group; (HbA1c) Haemoglobin A1C; (LDL) Low-Density Lipoprotein; (HDL) High-Density Lipoprotein; (HbA1c) Haemoglobin A1C; (LDL) Low-Density Lipoprotein, (HDL) High-Density Lipoprotein; (SFA) Saturated Fatty Acids; (PUFA) Poly-Unsaturated Fatty Acids; (MUFA) Mono-Unsaturated Fatty Acids.

Values are reported as Median (25th ,75th percentiles), means ±SD, or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale. [†]Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data [a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, [ǂ]Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data; Chi-square test for categorical data; [Ɣ]One-way ANOVA for comparing three groups continuous, normally distributed data with Tukey HSD as post hoc test; [r]Independent T-test for comparing two continuous, normally distributed data.

As per definition of an ET, they had later median wake time (09:48 vs 07:48) and sleep onset (00:48 vs 22:54) time as well as greater social jetlag (1.75 vs 0.88 hours). A positive number indicates later bed- and wake times on work free days. The median sleep duration was similar between ET and MT & IT (8.8 vs 8.9 hrs, respectively, $P = 0.63$, **Appendix C.2**).

5.3.2 Energy distribution across the 24-hour day

By investigating the average intake across all days, both the MT-IT and ET had three higher energy intake hours across the 24-hour day (in the morning, afternoon, and evening) (**Figure 5.1**). From visual inspection, the highest energy intake hours per chronotype group was approximately at the same clock hour during the afternoon and evening, with the exception of the first energy intake of the day, which was for ET approximately three hours later (at 10:00) than the MT-IT (at 07:00).The afternoon and evening highest energy intake hours differed in size with the MT-IT consuming more energy in the morning from 06:00 – 09:00 in comparison with the ET (1382 kJ vs 601 kJ, $P < 0.05$). The inverse was found at night from 21:00 – 02:00 where the ET consumed more energy than the MT-IT (1350 kJ vs 402 kJ, $P < 0.05$). However, during the afternoon between lunch and dinner time, the ET consumed more energy, balancing out the lunch and dinner time highs with MT. The distinct energy intake differences between MT-IT and ET are clear for early morning before 10:00 and later evening after 20:00. See **Appendix C.3-5** for the average hourly protein, carbohydrate and fat intakes.

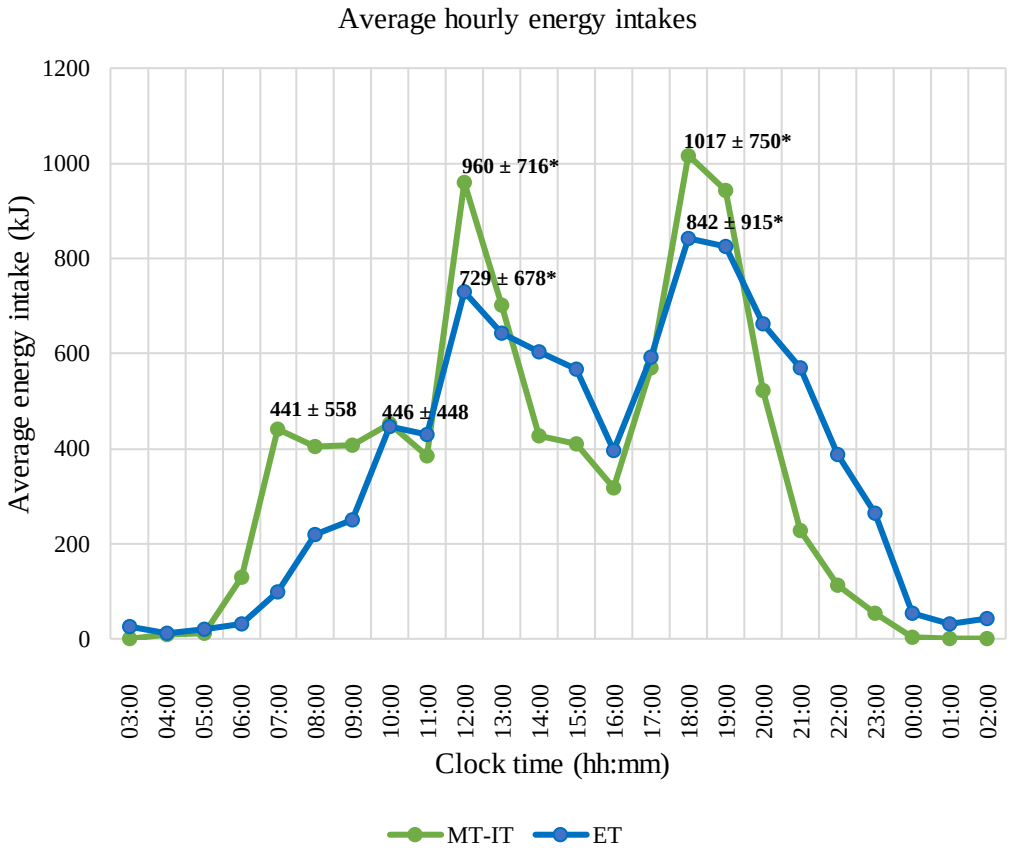

Figure 5.1 *- Average hourly energy intake between chronotypes*

Blue lines indicate ET and green lines indicate MT-IT,
Values are reported as means ±SD for each highest energy intake hours,
** Statistically significant differences between highest energy intake hours, with multiple comparison rank test, adjusted by Bonferroni correction*

5.3.3 Dietary intake time windows

A two-way repeated measures ANCOVA was performed to compare the effect of chronotype on the distribution of macronutrient intakes across four dietary intake windows. There was a significant main effect of chronotype on energy intake windows ($F(2.79, 787) = 17.2$, $P < 0.01$, figure 5.2), protein ($F(2.69, 757) = 19.6$, $P < 0.01$, figure 5.3), carbohydrate ($F(2.87, 810) = 11.2$, $P < 0.01$, figure 5.4) and fat ($F(2.85, 803.6) = 12.3$, $P < 0.01$, figure 5.5). Furthermore, a significant interaction effect between chronotype and the energy ($F(2.79,787) = 7.12$, $P < 0.001$), protein ($F(2.69,757) = 5.39$, $P < 0.01$), carbohydrate ($F(2.87,810) = 6.13$, $P < 0.01$) and fat ($F(2.85,804) = 5.94$, $P < 0.01$) intake windows was found.

Figure 5.2

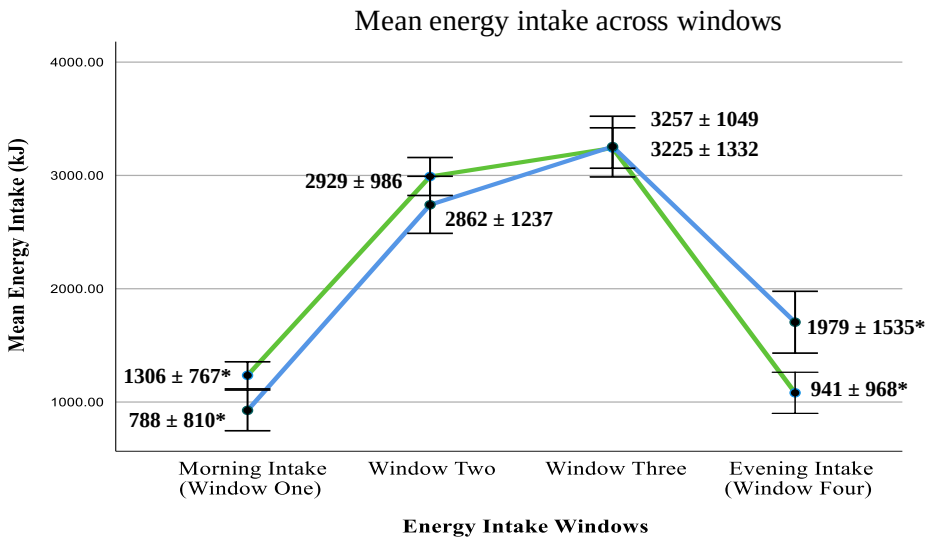

Figure 5.3

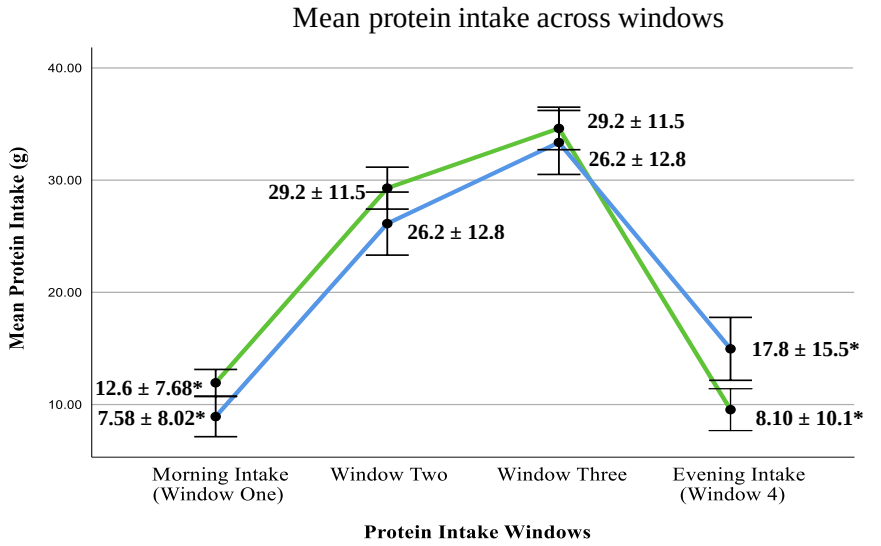

Figure 5.4

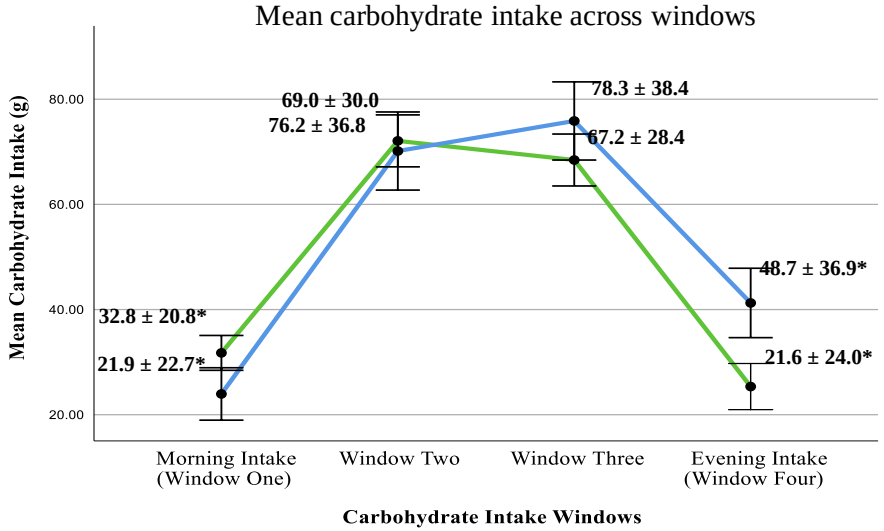

Figure 5.5

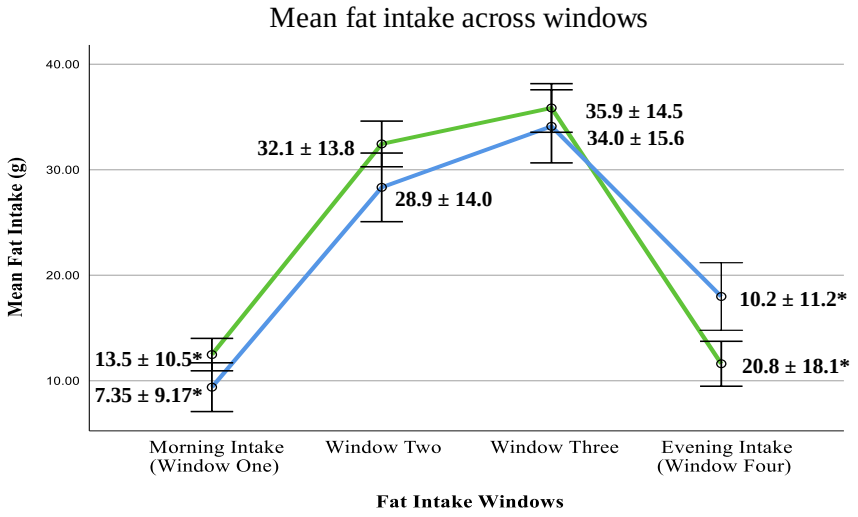

Figure 5.2 - 5.5 *- Two way repeated ANCOVA as windows of dietary intake*

Morning Intake (Window 1: 03:00 – 9:59), Window 2: 10:00 – 14:59, Window 3: 15:00 – 19:59, Evening Intake (Window 4: 20:00 – 02:59),
Blue lines indicate ET and green lines indicate MT-IT,
Values are reported as means ±SD,
** Statistically significant differences between chronotype groups in the specific window*

Further analysis showed that the MT-IT had higher nutrient and caffeine intakes in comparison with the ET in the morning window (1), while the ET had higher intakes during the evening window (4, **Table 5.3**). No statistically significant differences were found between chronotype groups and energy and macronutrient intakes in windows two and three (**Appendix C.6**).

***Table 5.3** - Dietary intake according to morning (time window 1; before 10:00) and evening intakes (time window 4; after 20:00)*

Dietary intake	Morning & intermediate type (n = 190)	Evening type (n = 97)	*P*-value [r]	Morning & intermediate type (n = 190)	Evening type (n = 97)	*P*-value[r]
Dietary window	**Morning intake (time window 1)**			**Evening intake (time window 4)**		
Energy (kJ)	1306 ± 767	788 ± 810	<**0.01**	941 ± 968	1979 ± 1535	<**0.01**
Protein (g)	12.6 ± 7.68	7.58 ± 8.02	<**0.01**	8.10 ± 10.1	17.8 ± 15.5	<**0.01**
Carbohydrate (g)	32.8 ± 20.8	21.9 ± 22.7	<**0.01**	21.6 ± 24.0	48.7 ± 36.9	<**0.01**
Fat (g)	13.5 ± 10.5	7.35 ± 9.17	<**0.01**	10.2 ± 11.2	20.8 ± 18.1	<**0.01**
Sugar (g)	16.0 ± 10.8	8.90 ± 12.5	<**0.01**	10.7 ± 11.6	20.9 ± 18.7	<**0.01**
Fibre (g)	4.89 ± 3.56	1.74 ± 2.00	<**0.01**	2.06 ± 2.45	3.84 ± 3.59	<**0.01**
MUFA (g)	5.03 ± 3.86	2.00 ± 2.42	<**0.01**	3.64 ± 4.28	7.96 ± 7.46	<**0.01**
PUFA (g)	2.37 ± 2.36	0.85 ± 1.28	<**0.01**	1.25 ± 1.64	2.51 ± 2.38	<**0.01**
SFA (g)	5.58 ± 4.75	2.46 ± 2.83	<**0.01**	4.20 ± 4.55	8.54 ± 8.52	<**0.01**
Cholesterol (mg)	68.7 ± 92.1	28.5 ± 59.1	<**0.01**	25.1 ± 33.8	57.5 ± 55.0	<**0.01**
Caffeine (mg)	68.9 ± 84.1	17.2 ± 33.2	<**0.01**	8.47 ± 15.6	14.0 ± 21.3	**0.03**
Alcohol (g)	0.00 ± 0.01	0.00 ± 0.00	0.21	1.64 ± 4.90	2.04 ± 6.72	0.60

(SFA) Saturated Fatty Acids, (PUFA) Poly-Unsaturated Fatty Acids, (MUFA) Mono-Unsaturated Fatty Acid. Values are reported as means ±SD,
[r]Independent T-test for comparing two groups continuous, normally distributed data,
Window 1: 03:00 – 9:59, Window 2: 10:00 – 14:59, Window 3: 15:00 – 19:59, Window 4: 20:00 – 02:59.

5.3.4 Associations between chronotypes, dietary intake in the morning by 10:00 (window 1) and evening after 20:00 (window 4) within body composition group

Table 5.4 shows chronotype as the predictor of dietary intake by 10:00 in the morning (window 1) and dietary intake by 20:00 in the evening (window 4) by total BF% and AG ratio groups (see **Appendix C.7** for analysis stratified by BMI groups). Among participants in the high BF% group, ET predicted lower energy (β = -513, *P* = 0.01), protein (β = -4.48, *P* <0.01) and carbohydrate (β = -10.7, *P* = 0.02) intake in the morning compared with MT-IT. In contrast, ET predicted higher energy (β = 603, *P* = 0.02), carbohydrate (β = 12.9, *P* = 0.04) and fat (β = 7.25, *P* = 0.02) intake in the evening compared with MT and IT. Among participants with a normal BF%, ET also predicted a higher energy (β = 650, *P* = 0.02), protein (β = 6.12, *P* = 0.02) and carbohydrate (β = 19.9, *P* = <0.01) intake in the evening. When stratifying by AG ratio the ET in the high AG ratio group predicted lower energy (β = -327, *P* = 0.02) protein (β = -3.14, *P* = 0.02), and carbohydrate (β = -9.38, *P* = 0.02) in the morning window. In

contrast, the ET in the high AG ratio group, predicted higher energy (β = 684, P <0.01) protein (β = 6.01, P = 0.01), carbohydrate (β = 16.3, P <0.01) and fat (β = 7.50, P = 0.01) intake in the evening after 08:00 compared with MT-IT.

Table 5.4 *- The association between chronotype (ET vs MT & IT) and nutrient intake according to morning stratified by BF% & AG ratio groups*

Nutrient	**Normal BF% (<35 %) n = 137**				**High BF% (≥35 %) n = 150**				**Normal AG Ratio (<0.8) n = 95**				**High AG Ratio (≥0.8) n = 192**			
	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*
Morning intake (Window 1)																
Energy intake (kJ)	185	-188	-554; 178	0.31	155	-513	-720; -106	**0.01**	240	-188	-665; 289	0.44	138	-327	-600, -54.5	**0.02**
	Adjusted R^2 = 0.13, $P < 0.01^{\delta}$				Adjusted R^2 = 0.11, $P < 0.01$				Adjusted R^2 = 0.08, $P = 0.02$				Adjusted R^2 = 0.11, $p < 0.001^{\delta}$			
Protein intake (g)	1.90	-1.38	-5.14; 2.38	0.47	1.49	-4.48	-7.39; -1.51	**<0.01**	2.52	-2.07	-7.07; 2.93	0.41	1.33	-3.14	-5.77, -0.51	**0.02**
	Adjusted R^2 = 0.09, $P < 0.01^{\delta}$				Adjusted R^2 = 0.15, $P < 0.01$				Adjusted R^2 = 0.03, $P = 0.14$				Adjusted R^2 =0.13, $p < 0.001^{\delta}$			
Fat intake (g)	2.45	-2.22	-7.06; 2.61	0.37	1.97	-3.91	-7.79; -0.18	0.05	3.11	-2.99	-9.16; 3.18	0.34	1.77	-2.88	-6.38; 0.62	0.11
	Adjusted R^2 = 0.09, $P < 0.01$ $^{\delta}$				Adjusted R^2 = 0.11, $P < 0.01$ $^{\delta}$				Adjusted R^2 = 0.09, $P = 0.04$				Adjusted R^2 =0.07, $p < 0.001^{\delta}$			
Carbohydrate intake (g)	4.90	-4.22	-13.9; 5.47	0.40	4.49	-10.7	-19.6; -1.85	**0.02**	6.75	-1.44	-14.9; 12.0	0.83	3.83	-9.38	-16.9, -1.83	**0.02**
	Adjusted R^2 = 0.10, $P < 0.01^{\delta}$				Adjusted R^2 = 0.06, $P < 0.01$				Adjusted R^2 = 0.04, $P = 0.11$				Adjusted R^2 =0.06, p =0.003			
Evening intake (Window 4)																
Energy intake (kJ)	273	650	110; 1190	**0.02**	249	603	111; 1094	**0.02**	295	418	-169; 1004	0.16	228	684	233, 1135	**<0.01**
	Adjusted R^2 = 0.18, P <0.001$^{\delta}$				Adjusted R^2 = 0.17, $P < 0.01^{\delta}$				Adjusted R^2 = 0.20, $P < 0.01$ $^{\delta}$				Adjusted R^2 = 0.16, $P < 0.01$			
Protein intake (g)	2.63	6.12	0.92; 11.3	**0.02**	2.66	5.21	-0.57; 10.7	0.05	2.98	3.43	-2.48; 9.34	0.25	2.35	6.01	1.38, 10.6	**0.01**
	Adjusted R^2 = 0.13, $P < 0.01$				Adjusted R^2 = 0.17, $P < 0.01$ $^{\delta}$				Adjusted R^2 = 0.11, $P < 0.01$				Adjusted R^2 =0.16, $P < 0.01^{\delta}$			
Fat intake (g)	2.98	5.03	-0.87; 10.9	0.09	3.07	7.25	1.19; 13.3	**0.02**	3.35	2.69	-3.97; 9.35	0.42	2.70	7.50	2.18, 12.8	**0.01**
	Adjusted R^2 = 0.15, $P < 0.01^{\delta}$				Adjusted R^2 = 0.13, $P < 0.01^{\delta}$				Adjusted R^2 = 0.17, $P < 0.01^{\delta}$				Adjusted R^2 = 0.13, $P < 0.01^{\delta}$			
Carbohydrate intake (g)	6.55	19.9	6.94; 32.8	**<0.01**	6.06	12.9	0.94; 24.9	**0.04**	6.68	14.2	0.92; 27.5	**0.04**	5.62	16.3	5.22, 27.4	**<0.01**
	Adjusted R^2 = 0.24, $P < 0.01^{\delta}$				Adjusted R^2 = 0.18, $P < 0.01^{\delta}$				Adjusted R^2 = 0.27, $P < 0.01^{\delta}$				Adjusted R^2 =0.18, $P < 0.01^{\delta}$			

(BF%) Body Fat Percentage; (AG) Android to Gynoid Fat Percentage Ratio,
Multiple linear regression models (Enter method) repeated within normal (<35 %) and high (≥35 %) BF% groups and then within normal AG Ratio (<0.8) and high AG Ratio (≥0.8), dependant variable: Nutrient Intake, Independent variable: Chronotype, adjusted for age, deprivation index & ethnicity, $^{\delta}$Covariate ethnicity significant in the model, Missing deprivation index (n = 3).

5.3.5 Correlations between chronotype, biomarkers, body composition and morning energy intake (window 1) and evening energy intake after 20:00 (window 4)

Correlation analysis revealed that those with later chronotypes (towards ET) were positively correlated with a higher body composition, including muscle mass and lean body mass **Table 5.5**. Women with an earlier chronotype (towards MT) were negatively correlated to a higher total serum-, HDL- and LDL-cholesterol, $HbA1_c$ and ghrelin concentrations. While those with a later chronotype were positively correlated with higher insulin, leptin, and triglyceride concentrations. Those with an earlier chronotype were negatively correlated to a higher morning energy intake, while those with a later chronotype were positively correlated with a higher evening energy intake.

Higher morning energy intake was negatively correlated with a lower body composition (all markers except WHR and WHtR). While the inverse was true for higher evening energy intake that was positively correlated with a higher body weight, BMI, body fat mass measured by BIA, HC and muscle mass measured by BIA. Higher morning energy intake was positively correlated with higher HDL-cholesterol and ghrelin and lower leptin concentrations, while higher evening energy intake was positively correlated with higher triglycerides, HbA_{1c} and insulin concentrations, and negatively correlated with lower ghrelin concentrations.

Table 5.5 *- Correlations of chronotype with biomarkers, body composition and morning energy intake (window 1) & evening energy intake (window 4)*

Body composition & metabolic biomarkers	**MCTQ**	**Morning energy intake (window 1)**	**Evening energy intake (window 4)**
MCTQ	-	-0.36***	0.49***
Morning Energy Intake (Window 1)	-0.36***	-	-0.22***
Evening Energy Intake (Window 4)	0.49***	-0.22***	-
Body composition markers			
Weight (kg)	0.24***	-0.19**	0.16**
BMI	0.20***	-0.17*	0.13*
Body fat mass (BIA, kg)	0.22***	-0.19**	0.14*
Fat mass (DXA, kg)	0.21***	-0.18**	0.11
BF% (BIA)	0.17**	-0.18**	0.10
BF% (DXA)	0.14*	-0.16**	0.04
Visceral Fat %	0.21***	-0.20***	0.08
Android Fat %	0.20***	-0.19***	0.08
Gynoid Fat %	0.14*	-0.14*	0.31
Android to Gynoid fat Ratio	0.20***	-0.19***	0.10
FM to FFM Ratio (DXA)	0.13*	-0.15*	0.03
FM to FFM Ratio (BIA)	0.16**	-0.13*	0.10
Waist circumference	0.15*	-0.13*	0.08
Hip circumference	0.20***	-0.17**	0.15*
Waist-to-hip-ratio	0.002	-0.03	-0.06
Waist-to-height-ratio	0.11	-0.11	0.06
Muscle mass (BIA)	0.22***	-0.17**	0.15*
Lean body mass (DXA, kg)	0.13*	-0.15*	0.03
Biomarkers			
Cholesterol (mmol/L)	-0.22***	0.09	-0.07
LDL-Cholesterol (mmol/L)	-0.15**	0.01	-0.05
HDL-Cholesterol (mmol/L)	-0.23***	0.23***	-0.07
Triglycerides (mmol/L)	0.19**	-0.07	0.14*
HbA1c (mmol/L)	-0.23***	-0.16**	0.26***
Insulin liggins (uU/ml)	0.26***	-0.19***	0.21***
Glucose (mmol/L)	0.08	-0.06	0.10
Ghrelin (pg/mL)	-0.29***	0.20***	-0.21***
Leptin (pg/mL)	0.16**	-0.18**	0.05
Peptide YY (pg/mL)	0.01	-0.03	0.45
C-reactive protein (mg/L)	-0.03	-0.04	-0.09

*Pearson's Correlation Analysis, *P < 0.05, **P < 0.01, ***P ≤ 0.001,*
(BMI) Body Mass Index; (BF%) Body Fat Percentage; (HbA1c) Haemoglobin A1C; (LDL) Low-Density Lipoprotein; (HDL) High-Density Lipoprotein; (FM) Fat Mass, (FFM) Fat Free Mass; (BIA) Bioelectrical Impedance Analysis; (DXA) Dual-Energy X-Ray Absorptiometry;(MCTQ) Munich Chronotype Questionnaire

5.4 Discussion

In this study the chronotype-specific differences in dietary intake (nutritional composition and timing), body composition and metabolic biomarkers were investigated. We found similar total daily energy and macronutrient intakes for MT and ET, but lower micronutrient intakes in the ET. Energy and macronutrient distributions were specific to each chronotype group according to their preferred sleep-wake timing, which is later for ET than MT-IT. The chronotype-specific distribution of dietary intake influenced fat mass and -distribution, with the ET having a higher body composition. The ET had poorer glucose homeostasis, endocrine regulator markers and lipid profile outcomes in comparison with the earlier chronotypes.

The most studied body composition marker in chronotype research is BMI (Kasof 2001, Schubert & Randler 2008, Sato-Mito et al 2011, Kanerva et al. 2012, Meule et al. 2012, Lai & Say 2013, Reutrakul et al. 2013, Patterson et al. 2016, Muñoz et al. 2017, Li et al. 2018, Patterson et al. 2018, Zeron-Rugerio et al. 2019, Najem et al. 2020). This study investigated a range of body composition markers. We found that women with a later chronotype (ET) had higher BMI, BF% and AG ratio, despite having similar sleep durations and similar total energy and macronutrient intakes in comparison with earlier chronotypes, which corroborate findings from a recent systematic review (van der Merwe et al. 2022). In comparison with the earlier types, the ET had the lowest diet quality with low intakes of folate, vitamins A, -C, and -E, magnesium potassium, calcium, iron, and iodine, which is similar to findings from others (Sato-Mito et al 2011, Kanerva et al. 2012). The low micronutrient intakes seen in the ET, is indicative of low intake of micronutrient dense foods such as fruits, vegetables, wholegrain and dairy (Baron et al. 2011, Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020). These findings agree with others who have suggested that the role of the diet in obesity development is too simplistically described as energy intake that does not match with energy expenditure (Taubes et al. 2013), whereas it is rather that the timing of food intake is a crucial factor with regards to weight gain and adverse metabolic health outcomes (Arble et al. 2009, Garaulet et al. 2013, Garaulet 2014, Ruiz-Lozano et al. 2016).

The timing of dietary intake is important as there is clear evidence that macronutrient metabolism and energy expenditure vary across the 24-hour light-dark cycle (Roberts 1964, Takahashi et al. 1968, Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Saad et al. 2012, Cipolla-Neto et al. 2014, Covassin et al. 2016 , Rácz et al. 2018), having different effects on energy utilisation and ultimately body composition (Garaulet et al. 2013, Jakubowicz et al. 2013, Jakubowicz et al. 2017). Insulin sensitivity and glucose tolerance are optimal during daytime, coupled with increased energy expenditure, lipogenesis and adiponectin synthesis, making it the ideal phase of the 24-hour day for acquiring and storing energy through dietary intake (Cipolla-Neto et al. 2014). Conversely, upregulation of gluconeogenesis, glycogenolysis and lipolysis is prominent at night

(Cipolla-Neto et al. 2014), with resulting higher concentrations of circulating fatty acids, triglycerides and cholesterol concentrations (Takahashi et al. 1968, van Moorsel et al. 2016), making this the ideal phase for fasting. Therefore, when food is consumed at a later time during the 24-hour day it may have a 'desynchronising' effect on the circadian clocks, resulting in increased adiposity and poor metabolic health outcomes (Salgado-Delgado et al. 2010 , Fong et al. 2017, Nelson & Chbeir 2018, Challet 2019, Arredondo-Amador et al. 2020). The importance of the daytime-eating and night-time-fasting pattern has been shown by other researchers, who found that food intake later in the day affects body composition and weight loss success negatively (Garaulet et al. 2013, Jakubowicz et al. 2013).

Chronotype mediates both sleep and wake patterns (Duffy et al. 2001) as well as meal timing. Previous studies have shown that ET delay their meal timing with later clock times for main meals (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019). Studies that have explored the differences between chronotypes and the distribution of food intake throughout the day, have focused mainly on energy distribution (Baron et al. 2011, Lucassen et al. 2013, Maukonen et al. 2017, Muñoz et al. 2017, Maukonen et al. 2019, Zerón-Rugerio et al. 2020) and to a lesser extent on macronutrient distribution (Baron et al. 2013, Maukonen et al. 2017, Muñoz et al. 2017, Xiao et al. 2019). Later chronotypes typically consumed more energy and macronutrients during the evening and less energy and macronutrients during the morning in comparison with earlier types (Baron et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2017, Maukonen et al. 2019, Xiao et al. 2019, Zerón-Rugerio et al. 2020). In our study we saw a similar pattern with the ET consuming only 9.01 % (788 kJ) of their total daily energy intake in the mornings and two and a half times the quantity at night (22.6 % of total energy intake; 1979 kJ). In comparison, those with an earlier chronotype (MT-IT), consumed a higher proportion (15.3 %; 1306 kJ) of their total energy intake in the morning and one and a half of that during the evening (11 %, 941 kJ). With regards to the macronutrient distributions, the MT-IT had a higher morning protein, carbohydrate and fat intake in comparison with ET who had a higher evening protein, carbohydrate and fat intake. This reflects the ET's internal preference for structuring their sleep and wake timing (Roenneberg et al. 2007), including timing of dietary intake to later in the day, and the night respectively (van der Merwe et al. 2022). One theory for the higher ET- dietary intake at night, is the breakfast skipping theory, which proposes that when breakfast is skipped the individual tends to be hungrier later in the day, especially during the evening, resulting in an overconsumption of food (Stanton Jr 1989), thereby still consuming the same amount of food in the 24-hour day despite missing a meal.

Few studies have investigated the effect of chronotype-specific dietary distribution in relation to body composition outcomes (Muñoz et al. 2017, Xiao et al. 2019). Xiao et al. (2018), linked the lower morning and higher evening dietary intake pattern observed among ET with a higher BMI. They found that MT who consumed a higher proportion of their energy, carbohydrates, and protein in the morning

(within two hours after waking up) were less likely to be overweight/obese, while the ET that consumed a higher proportion of energy, carbohydrates, and protein in the evening (within two hours before bedtime) were more likely to be overweight/obese than the MT (Xiao et al. 2019). Similar to the results from Xiao et al. (2018), our study revealed that, among participants in the high BF% group, ET was associated with lower energy, protein, and carbohydrate intake in the morning compared with MT-IT, but higher energy, carbohydrate, and fat intakes at night when compared with MT-IT. To clarify these findings the distribution of the different body fat compartments, namely AG ratio was investigated. The AG ratio has been shown to be a more sensitive predictor of NCD and metabolic syndrome in healthy adults than BMI and fat mass (Kang et al. 2011, Fu et al. 2014, Okosun et al. 2015). The results were similar, the ET in the high AG ratio group (more central body fat) predicted lower energy, protein, and carbohydrate intakes in the morning, while predicting higher energy, protein, carbohydrate, and fat intakes in the evenings when compared with MT-IT, further emphasising the obesity promoting effects of late-night eating among ET. The importance of the overnight fasting period is also highlighted in time restricted eating studies which have demonstrated that longer fasting periods are more beneficial for health than shorter ones (Manoogian & Panda 2017) and since ET eat closer to their bedtime, their fasting period is shortened.

Later circadian timing of food intake has also been linked with poor metabolic health. A study by Garaulet et al. (2020) found that after one week of eating lunch later, lean women showed metabolic changes that are typically seen in obese women, such as impaired glucose tolerance, decreased resting energy expenditure, and decreased carbohydrate oxidation (Garaulet 2020). In this study, late eaters, i.e., those who consumed a higher proportion of energy during the evening, were positively correlated with a late chronotype (ET), higher body composition (including weight, BMI, HC, and body fat mass) as well as a poorer glucose homeostasis (higher HbA1c and insulin) and higher triglycerides concentrations. While, early eaters, i.e., those who consumed a higher proportion of energy during the morning, were correlated with an earlier chronotype (MT), a lower body composition and lower metabolic markers. In addition, the length of the overnight fasting period plays a role in metabolic health and weight gain. Time restricted eating (TRE) studies have shown that longer fasting periods are more beneficial for metabolic health than shorter ones (Manoogian & Panda 2017). In this study the ET women had a slightly shorter (although not significant) overnight fasting period of 14.3 hours vs the MT-IT who had an overnight fasting period of 14 hours. The duration between the last eating occasion of the day and the wake time for ET was significantly shorter than for the MT-IT (10 vs 11.6 hours). Furthermore, the ET women had a later clock time for their first eating occasion of the day in comparison with the MT-IT (10:54 vs 08:41) **(Appendix C.1)**, which may result in elevated postprandial insulin concentrations and enhanced fat oxidation, ultimately contributing to the development of metabolic inflexibility and weight gain (Nas et al. 2017). These results highlight the metabolic changes that occur when food is consumed at the inappropriate time of the day.

5.5 Strengths & limitations

To our knowledge this was the first study to explore body composition (including android and gynoid fat mass distribution)-, and metabolic biomarker differences in depth, between chronotypes in a healthy population. We explored the diet in depth with total energy, macronutrient but also micronutrient intakes, with a particular focus on the distribution of dietary intake in relation to body composition.

This study had some limitations. Firstly, it was cross sectional in design, making the investigation of dietary intake, metabolic biomarkers, and body composition changes over time not possible. The study could not differentiate between weekday and weekend days, due to the fact that the MCTQ reports usual wake- and sleep times for weekdays and weekend days that did not necessarily correspond with the mealtimes reported in the food records. Based on the age-dependent distribution of chronotypes, we had a small number of MT in this cohort of young women, which could not be controlled for as a random cross-sectional recruitment strategy was implemented. This study was not specifically designed to have equally balanced MT and ETs. Lastly, due to the small sample size of MT in the Pacific and NZ European groups, the analysis could not be further stratified by ethnicity, however, analyses controlled for ethnicity to mitigate this limitation.

5.6 Conclusion

In conclusion, chronotypes tend to consume similar amounts of total energy and macronutrient intakes. ET women are more likely to have a higher body composition and poorer metabolic biomarker profile, likely due to their higher nightly and lower morning food intake. This eating pattern predisposing ET to consume, digest and metabolise food during the normal fasting phase of the light/dark cycle. In addition, ET have a poor diet quality favouring high intakes of micronutrient poor foods. Earlier chronotype's habitual early wake-, sleep- and meal timing are possibly protective against a high body composition and unhealthy metabolic biomarker profiles.

The main focus areas of the chapter were the distribution of macronutrient intakes throughout the day and the effect of the distribution on body composition. In addition, the differences in metabolic biomarker profiles between the different chronotypes were investigated. The following chapter (six) will investigate dietary patterns and provide a whole diet picture beyond total dietary intake differences between chronotypes.

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6 Chapter 6: Nutrient & food pattern analysis among women with different chronotypes

Abstract

Background and aims: Food pattern analysis explores the whole diet in terms of the foods that are typically consumed together, to provide context on dietary combinations that may contribute to nutrition-related health outcomes. Nutrients are an important consideration alongside to interpret the food patterns, however, nutrient patterns are rarely explored. The aim of this study was to identify dietary patterns (food and nutrient patterns) and investigate if the patterns are associated with chronotype and body composition.

Methods: Overall, 287 healthy women aged 18-45 years (130 Pacific and 157 NZ European) were included in this analysis. Anthropometrical measurements included height, weight, body mass index (BMI), whole body fat percentage using dual-energy X-ray absorptiometry. The Munich Chronotype Questionnaire was used to assess different chronotypes, i.e., early chronotypes (=morning types; MT), intermediate chronotypes (IT) and late chronotypes (evening types; ET). Participants completed a five-day estimated food record, including two weekend- and three weekdays, from which food group-, energy-, macro- and micronutrient intakes were assessed. Both food - and nutrient patterns were extracted by principal component analysis. Dietary pattern scores were categorised into tertiles to investigate relationships with body composition and chronotype.

Results: Four food patterns as well as four nutrient patterns were extracted. Most of the earlier chronotypes (MT-IT, n =190) followed the *Healthy* and *Animal Products* food patterns and the *High Protein-Fat-Fibre* nutrient pattern. The *Healthy* food pattern was high in fruits, vegetables and wholegrains and was associated with lower BMI and BF%, NZ European ethnicity and lower deprivation score. Most ET women (n = 97) followed the *High Carbohydrate* nutrient pattern, which was high in carbohydrates, starch and sugar. The *High Carbohydrate* nutrient pattern was associated with Pacific ethnicity and higher deprivation scores.

Conclusion: The identified food- and nutrient patterns created a whole-diet picture of the typical nutrients and foods consumed together, explaining some of the variance seen in body composition differences between different chronotypes.

Key words: Morningness, Eveningness; Adiposity; Dietary Pattern Analysis; Food Combinations.

6.1 Introduction

Nutritionally sound diets are the foundation of health and wellbeing (Leech et al. 2015). Obtaining essential nutrients and dietary factors are vital for survival and can prevent and/or promote disease development. It is well-established that a poor diet contributes to adverse health outcomes and consequently increases the risk of diseases such as inflammatory diseases, hypertension, diabetes and obesity (McHill et al. 2011). In recent years, a gradual shift in diet-disease-risk research has occurred (Leech et al. 2015). Dietary assessment approaches have moved away from a simplistic single-nutrient-only approach to whole-diet assessments such as dietary pattern analysis (Krebs-Smith et al. 2015). This whole-diet approach recognises that individuals don't eat singular foods or nutrients, but rather consume a variety of foods/nutrients typically grouped together at different eating occasions (Kant 2018). Dietary pattern analysis, using methods such as principal component analysis (PCA), thus provide an overall perspective of the diet, illustrating the combinations of foods and inclusive nutrients consumed together (Frank 2002, Krebs-Smith et al. 2015, Liese et al. 2015).

Food pattern analysis has previously been used to explore the diet and the link with nutrition-related health outcomes, such as obesity (Paradis et al. 2009). It is well known that body composition is influenced by a range of interplaying factors including the types and combinations of foods consumed together (Miller et al. 1990, Satia-Abouta et al. 2002, Newby et al. 2003, Ahluwalia et al. 2009, Paradis et al. 2009, Austin et al. 2011, Suliga et al. 2015). It has been shown that the "Western dietary pattern", characterised by energy dense and micronutrient poor foods such as sugar sweetened beverages, fast foods, refined grains, red- and processed meats, and confectionery, is associated with weight gain and obesity as well as an elevated risk for the development of lifestyle diseases (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015). Conversely the "Prudent dietary pattern", high in whole grains, fruits, vegetables, legumes, and fish, has been linked with more healthy body composition including waist circumference and BMI and consequently a lower risk for disease development (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015). Several studies have demonstrated a marked difference in types of foods consumed among the early risers, morning- (MT) and late risers, evening types (ET). The ET tended to consume more unhealthy foods such as sweets and confectionery , sugar sweetened beverages (Baron et al. 2011, Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020), alcohol (Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Vera et al. 2018) and fatty (Mota et al. 2016, Muscogiuri et al. 2020) foods in comparison with earlier chronotypes (MT) who consumed more healthy foods such as fruits, vegetables (Baron et al. 2011, Sato-Mito et al. 2011, Yoshizaki et al. 2018), dairy (Sato-Mito et al. 2011) and fish (Maukonen et al. 2017). Differences in total independent food group intakes among chronotypes is one of the most explored dietary aspects in the chronotype literature

(Sato-Mito et al 2011, Kanerva et al. 2012, Patterson et al. 2016, Muñoz et al. 2017, Patterson et al. 2018, van der Merwe et al. 2022).

Nutrient pattern analysis is the combination of several nutrients that offer more information on the synergistic effects of nutrients as consumed from the food sources (Newby & Tucker 2004). These patterns are beneficial in creating nutritional profiles that are comparable between populations (Freisling et al. 2010). Studies have found inverse associations between weight gain and obesity and single nutrients such as protein and carbohydrate, while fat intake was positively associated with BMI (Miller et al. 1990, Satia-Abouta et al. 2002, Ahluwalia et al. 2009, Austin et al. 2011). Nutrient pattern analysis in relation to greater cancer risk has often been explored (Palli et al. 2001, Bosetti et al. 2013), and to lesser extent in regard to metabolic syndrome and obesity (Moskal et al. 2014, Vajdi et al. 2020). Principal component analysis (PCA) is a multivariate statistical method that compiles dietary patterns based on the extent to which food items/nutrients in a dataset correlate with each other (Moeller et al. 2007). A score for each study participant is derived from either the food item or nutrient data that reflects their intake of each dietary pattern.

The majority of studies discriminating between different chronotypes, demonstrated that although ET typically have a higher BMI, they typically have similar total energy and macronutrient intakes in comparison with MT (van der Merwe et al. 2022). Therefore, this study seeks to create a more comprehensive insight into the whole diet in terms of both food and nutrient combinations and aims to link the results with body adiposity among chronotype groups. The aim of this study was to identify dietary patterns (food and nutrient patterns) and investigate if the patterns are associated with chronotype and body composition. Furthermore, to describe the food patterns in terms of nutrient intake distributions and then describe nutrient patterns in terms of food intake distribution.

6.2 Methods

6.2.1 Study design, participants & procedure

The study participants formed part of the cross-sectional study, PRedictors linking Obesity and gut Microbiome: PROMIsE that was conducted between July 2016 and September 2017 at the Massey University Human Nutrition Research Unit in Auckland, New Zealand (NZ) (Kindleysides et al. 2019). Healthy women from both NZ European and Pacific ethnic groups, between the ages of 18 and 45 years were recruited based on healthy BMI (18.5 - 24.9 kg/m^2) and obese BMI ($\geq$ 30 kg/m^2), aiming for n = 68 from each group (272 in total). Participants were excluded if they were pregnant or lactating; diagnosed with chronic illness (e.g., diabetes and cardiovascular disease); underwent bariatric surgery; having severe food allergies; using medication impacting appetite or the immune system (e.g., appetite suppressants and corticosteroids); current smokers; using severe dietary restrictions /avoidances (e.g.,

vegan). For this analysis in particular, night shift workers (n = 17) were also excluded because these individuals exhibit altered sleep-wake times and fasting-feeding cycles due to work schedules and not necessarily because of their chronotype. The final sample size for this study was 287 participants of which 157 were NZ European and 130 were Pacific women. **Figure 6.1** depicts the recruitment aims, recruited and final sample size following exclusions. Ethics approval was obtained from the Southern Health and Disability Ethics Committee (16/STH/32), and written informed consent was obtained from all participants prior to data collection. Details of the study procedures and recruitment have been published elsewhere (Kindleysides et al. 2019). Methodology pertaining this sub-study are described below. The participants attended two visits (no more than 14 days apart). Visit one included body composition measurements (height, weight, completion of questionnaires (MCTQ; (Roenneberg et al. 2003), and retrospective seven-day, sleep and work questionnaire) as well as an interview for demographic information. The five-day food records (5DFR) were completed at home between study visits. Visit two included body composition measurements (weight, BF% measurements), 5DFR returns, and a one-on-one food record interview with a dietitian. Details on study procedure have been discussed in detail elsewhere (Kindleysides et al. 2019).

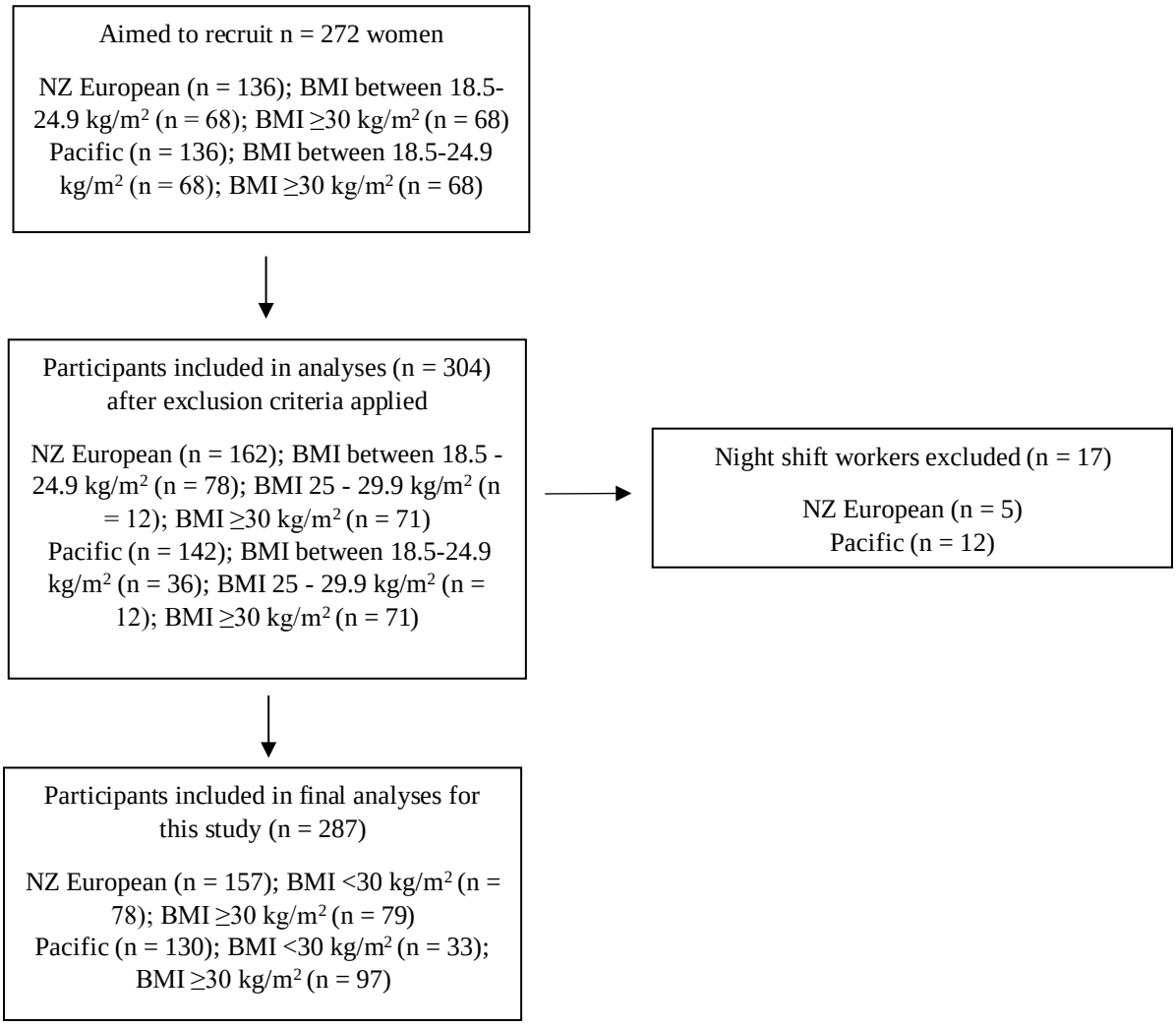


Figure 6.1 *- Flow chart of participants included in the analysis*

(NZ) New Zealand, (PROMIsE) PRedictors linking Obesity and gut Microbiome

6.2.2 Anthropometrical measurements

Anthropometrical measurements (height and weight) were conducted according to the International Society for the Advancement of Kinanthropometry (ISAK) protocols by level one ISAK trained research staff (Stewart et al. 2011). Body mass index was determined using the Quetelet index (weight (kg)/height $(m)^2$). Whole body composition measurements were performed using dual-energy x-ray absorptiometry (DXA; Hologic QDR Discovery A, Hologic Inc with APEX V. 3.2 software) to assess body fat percentage of total body as well as other body fat parameters. Recognising that BMI does not accurately reflect adiposity on an individual level, BF% was measured to create a comprehensive body composition profile (De Lorenzo et al. 2016).

6.2.3 Demographic information & deprivation index

Interviewer-administered questionnaires were used to collect health (including information on menstruation, disease conditions, medications, previous surgery, and supplement use) and demographic (age, housing, education, income) information. Socio-economic status was measured by using the NZ Deprivation Index 2013 (NZDep2013) which is calculated from census information such as home ownership, housing, qualifications, employment, income, access to transport, communications, and family structure. The deprivation scores were classified into deciles, where one represents the geographical area with the least and 10 the most deprived scores (Crampton et al. 2020).

6.2.4 Chronotype, sleep & work schedules

Chronotype, sleep and work schedules were assessed by using the Munich Chronotype Questionnaire (MCTQ). The MCTQ, developed by Roenneberg et al. assesses chronotype from averaged midsleep timing of the last four weeks (Roenneberg et al. 2003). Midsleep time (i.e., the midpoint between the time of subjectively estimated sleep onset and waketime) derives from midsleep times on weekdays (MSW) and on free days (MSF) and is corrected for sleep duration on free days (MSFsc) (Roenneberg et al. 2003). A greater number of the MSFsc (= later midsleep time) is an indicator for late chronotypes (ET; midsleep > 5) and vice versa for early chronotypes (MT; midsleep time < 3). A midsleep time between 3 and 5 indicates an intermediate chronotype (Roenneberg et al. 2003). Information on sleep and work schedules (number of hours worked per day as well as the type of shift work (day or night)) was collected via a retrospective seven-day, sleep and work questionnaire. Participants who reported night shift work were excluded from the analyses.

6.2.5 Dietary intake

Dietary intake for total energy (kJ), macro (g) - and micronutrient intakes (g, mg, or µg), were assessed using estimated 5DFR from two weekend- and three weekdays, which were reviewed by a NZ registered dietitian. Participants received training for estimating and documenting their food portions (10-minute instructional video and interviewer). Visual portion book aids, household measures (e.g., metric cups and spoons), and Web-based tools were provided to participants to confirm specific portion sizes and product brands consumed. Data were entered into the computer software, FoodWorks10 (Xyris Software (Australia) Pty Ltd, Queensland, Australia) and the NZ food composition database (NZ FOODFiles 2016) was used to analyse the nutrient (energy, macro- and micronutrient) content of the 5DFR. If an appropriate compositional match for an item was not found in the NZ food composition database, a food item from the Xyris brand name database AusFoods 2017 (based on the Australian food composition databases AUSNUT 2011-13, developed by Food Standards Australia New Zealand) was selected (Kindleysides et al. 2019).

Foods items from the 5DFR were categorised into food groups (g/day) based on a previous study conducted in this NZ population (Schrijvers et al. 2016). The foods were categorised into groups that were similar in nutritional composition and characteristics (**Appendix D.1**). For example, individual food items such as cream cheese was categorised in the "cheese" group and green banana was categorised in the "starchy vegetable" group. Mixed dishes such as "beef lasagne" were allocated as individual food items to the corresponding food groups - the "wheat pasta" was allocated to the "refined grains", "beef mince" to "meat", "canned tomatoes", and "fresh onion" to "non-starchy vegetables" groups. Originally 66 food groups were created, however some food groups were only used by a small number of participants, below the recommended 5 to 10 per food group (Kim and Mueller 1978). Therefore, the food groups with low number of participants were combined with similar groups, creating 33 food groups. For example, the "wholegrain mixed dishes" group that contain dishes such as tabbouleh was combined with the "wholegrains" group.

6.3 Data handling & statistical data analysis

Statistical analyses were conducted using IBM SPSS Statistics for Windows version 28 (Armonk, NY: IBM Corp). Two sets of dietary patterns were extracted by PCA method with orthogonal (varimax) rotation using the food weight consumed by each participant. Firstly, dietary patterns using the average food group intakes (g/day) were extracted to determine food patterns. Secondly, dietary patterns were extracted by using average nutrient intakes (g/mg/µg day) to determine nutrient patterns. The suitability of the data sets for PCA was determined with the Kaiser-Meyer-Olkin Measure of Sampling Adequacy (measures the proportion of variance in the variables that might be caused by underlying factors) which was 0.65 for the food pattern and 0.69 for the nutrient pattern (> 0.05 acceptable) (Kaiser 1974) and

Bartlett's test of sphericity (determines whether there is a significant difference in the variances), for which the *P*-value was <0.001 (<0.05 acceptable) (Field 2009). The factors from the PCA retained for both the food- and nutrient patterns were based on the break-even point of the scree plot and Eigenvalue cut-off > 1.0, the interpretable variance percentage and the factor loadings (Field 2009). Factor loadings of > 0.2 (positive relationship) or ≤ -0.2 (inverse relationship) were considered significant. Each of the factor loadings represent the correlations between the food patterns and the food groups, or the correlations between the nutrient patterns and the nutrients. Food groups and nutrients which cross-loaded on multiple dietary patterns were retained as they aided in explaining the specific dietary pattern. However, the highest loading which was considered to have the greatest impact is in bold. Participants were allocated a negative or positive score for each of the extracted food and nutrient patterns, that were further categorised into tertiles to differentiate women with higher scores (tertile three = T3) from those with lower scores (tertile one = T1).

The study participants were grouped by chronotypes, i.e.: ET (MSFsc >5), IT (MSFsc = 3 - 5) and MT (MSFsc <3). The MT and IT were combined as one group (MT-IT) for analysis due to the small MT sample size. The differences between study variables across the different dietary patterns' tertiles were determined using the following methods, distinguishing between normal and non-normally distributed data. Non-normally distributed data (presented as medians and 25^{th} and 75^{th} percentiles) was assessed by using Kruskal-Wallis tests with multiple comparison rank test, adjusted by Bonferroni correction. Normally distributed data (presented as means and standard deviations) was assessed by one-way ANOVA tests with Tukey HSD as post-hoc tests. Log transformed data values were back transformed to the original scale and reported as geometric mean [95 % CI]. Chi-square tests were used to describe differences between categorical data (e.g., ethnicity and chronotypes). A *P*-value of < 0.05 was considered as statistically significant.

Binary logistic regression (enter method) was used to assess the association between (1) chronotype groups (MT-IT vs ET) and the dietary patterns, (2) body composition groups (BMI and BF%) and the two types of dietary patterns (food- and nutrient patterns). The dependent variable was T1 vs T3, while the independent variable was either (1) the chronotype groups (MT-IT used as reference category) or (2) body composition groups (low body composition was used as reference category). The regression analyses were adjusted for age, deprivation index, total energy intake and ethnicity.

6.4 Results

6.4.1 Study participants

The women included in these analyses had a median (25^{th}, 75^{th} percentiles) age of 27 (22, 35) years, BMI of 28.6 (23.0, 34.1) kg/m^2 and BF% of 35 (29.0, 40.0) % (**Table 6.1**). The majority of the women

(n = 190; 66%) were classified as MT-IT followed by ET (n = 97; 34%). Average wake times on workdays were 07:30 (07:00, 09:00) and 09:00 (08:00, 10:00) on work free days. Average sleep onset times on workdays were 22:45 (22:13, 23:34) and 23:49 (23:04, 01:15) on work free days.

Table 6.1 *- Characteristic of study population (n = 287)*

Demographics	**N (%)**
Age (years) †	27 (22, 35)
Ethnicity Categories, n (%)	
NZ European	157 (54.7)
Pacific	130 (45.3)
Chronotype Groups, n (%)	
Morning & Intermediate Types	190 (66.2)
Evening Types	97 (33.8)
Deprivation score †	6 (3, 9)
BMI (kg/m^2) †	28.6 (23.0, 34.1)
BF%†	35 (29.0, 40.0)
Average wake time on workdays (hh:mm) †	07:30 (07:00, 09:00)
Average wake time on work free days (hh:mm) †	09:00 (08:00, 10:00)
Average sleep onset time workdays (hh:mm) †	22:45 (22:13, 23:34)
Average sleep onset time work-free days (hh:mm) †	23:49 (23:04, 01:15)

(NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, †Values reported as Median (25th – 75th percentiles)

6.4.2 Principal component analysis for food patterns

The principal component analysis (PCA) yielded four *food* patterns, which explained 27.4% of the variation in dietary intake among the study population (**Table 6.2**). The Kaiser-Meyer-Olkin Measure of Sampling Adequacy was 0.65 and Bartlett's test of sphericity was significant ($P < 0.001$), verifying that PCA was appropriate for this data set.

Food pattern one, the *Healthy* food pattern, explained 11% of the variance. This food pattern was characterised by positive high loadings on coloured vegetables, nuts & seeds, non-starchy vegetables, apple, banana, & orange, and moderate loadings on tea, unsaturated plant-based fats, shakes & protein powders, coffee, wholegrains, water, and meats (see **Table 6.2**). This food pattern had negative loadings on fast-foods, sugar- & artificially sweetened beverages, and savoury snack foods.

Food pattern two, the *Discretionary Food & Beverages* food pattern, explained 5.5% of the variance and had positive loadings on legumes (e.g., hummus), creamy dressings & sauces, discretionary snack breads, sweetened dairy & milk alternatives, alcoholic drinks, and moderate loadings on fish & seafood, other fruits & juices. This food pattern had negative loadings on meat, starchy vegetables, processed meats, and animal & coconut fats; thus, characterised as a preference for plant-based proteins, discretionary snack breads and high energy beverages.

The third food pattern, the *Animal Products* food pattern, explained 5.5% of variance. This pattern had positive loadings on processed meats, eggs, cheese, and dairy milk, with negative loadings on apple, banana, & orange, tea, discretionary snack breads, sweetened dairy & milk alternatives, cakes & puddings, fish & seafood, and other fruits & juices.

Food pattern four, the *Sugary, Fatty Foods* food pattern, explained 5.4% of variance and had positive loadings on sweet snack foods, sugar added to food & beverages & sweet spreads, and saturated animal-based & coconut fats. Negative loadings on wholegrains, dairy milk, breakfast cereals and refined grains were found.

***Table 6.2** - Rotated factor matrix of food patterns*

Food group	**Food pattern**			
	Healthy	**Discretionary Food & Beverages**	**Animal Products**	**Sugary, Fatty Foods**
Explanation of variation of food choices (in %; 27.5% total)	11.1	5.5	5.5	5.4
Coloured vegetables	**0.70**			
Fast Foods	**-0.68**			
Nuts & seeds	**0.65**			0.34
Sugar & Artificially Sweetened Beverages	**-0.60**			
Non-Starchy Vegetables	**0.57**			
Apple, Banana & Oranges	**0.51**		-0.28	
Tea	**0.43**	0.24	-0.12	
Unsaturated Plant-Based Fats	**0.38**		0.34	
Shakes & Protein Powders	**0.37**			
Coffee	**0.36**	0.29	0.31	
Wholegrains	**0.34**			-0.26
Water	**0.29**			
Savoury Snack Foods	**-0.26**			
Legumes	0.22	**0.60**		
Meat	**0.31**	-0.54		
Creamy Dressings & Sauces		**0.45**		
Discretionary Snack Breads		**0.40**		
Sweetened Dairy & Milk Alternatives		**0.30**	-0.29	
Alcoholic Drinks		**0.46**		
Starchy Vegetables		**-0.44**		
Processed Meats		-0.27	**0.40**	
Eggs	0.33		**0.40**	0.36
Cakes & Puddings			**-0.38**	
Cheese	0.31	0.24	**0.37**	0.27
Fish & Seafood		**0.22**	-0.29	-0.26
Other Fruit & Juices		**0.25**	-0.29	
Savoury Sauces Spreads & Condiments				
Sweet Snack Foods				**0.52**

Food group	Food pattern			
	Healthy	**Discretionary Food & Beverages**	**Animal Products**	**Sugary, Fatty Foods**
Sugar-Added to Food and Beverages & Sweet Spreads				**0.51**
Dairy Milk			**0.21**	-0.44
Saturated Animal-based & Coconut Fats	0.26			**0.43**
Breakfast Cereals			-0.21	**-0.31**
Refined Grains				**-0.26**

Patterns identified based on factor loadings >0.2; food groups with factor loadings <0.2 not shown, Food groups which cross-loaded on multiple food patterns are shown in the table, with the highest factor loading indicated in bold, and only used once across the four patterns.
Kaiser-Meyer-Olkin Measure of Sampling Adequacy = 0.65, Bartlett's test of sphericity is significant (P < 0.001).

6.4.2.1 Demographics, body composition & chronotype distributions across the food patterns tertiles

Women with the highest score (T3) on the *Healthy* food pattern had a lower deprivation score (5 (3, 7) vs 8 (6, 9)), BMI (23.9 (22.0, 32.8) vs 30.8 (25.1, 36.3) kg/m^2) and BF% (32 ± 8 vs 37 ± 6 %) compared with those with the lowest score (T1) ($P < 0.001$). A higher percentage of the MT-IT (44.4 %) and NZ Europeans (49.7 %) scored high on this pattern (T3), compared with fewer of the ET (11.3 %) and Pacific women (13.8 %) ($P < 0.001$) (see **Table 6.3**). For those consuming the *Discretionary Food & Beverages* food pattern, significantly more of the NZ European women (43.9 %) and MT-IT (38.1 %) and fewer of the Pacific women (20 %) and ET (23.7 %) scored high (T3). Furthermore, women who scored high vs those who scored low were mostly from less deprived areas ((5 (2, 7) vs 8 (5, 9)) and had a lower BMI (25.3 (22.7, 31.8) vs 31.5 (23.4, 37.1) kg/m^2) ($P < 0.001$). Similarly, for the third *Animal Products* food pattern, a higher percentage of the NZ Europeans (43.3 %) and MT-IT (39.5 %) scored high on the Animal Products food pattern (T3), while a lower percentage of the Pacific women (21.5%) and ET (21.6 %) scored high ($P <0.001$). A higher proportion of NZ European (40.1 %) scored high on the *Sugary, Fatty Foods* pattern and a lower proportion of Pacific (25.4 %) women scored high ($P = 0.01$). There was no statistically significant difference between the tertiles with regards to chronotypes for this pattern.

Table 6.3 *-Participant characteristics per food pattern tertiles*

	Healthy				**Discretionary Food & Beverages**				**Animal Products**				**Sugary, Fatty Foods**			
	T1	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]
Age (years)	28 (22, 37)	28 (22, 36)	25 (22, 34)	0.37	25 (22, 34)	29 (22, 36)	26 (23, 37)	0.24	27 (22, 36)	29 (23, 36)	25 (21, 34)	0.10	25 (21, 33)	29 (22, 36)	27 (23, 36)	0.15
NZ European n(%) n = 157	20 (12.7)	59 (37.6)	78 (49.7)	**<0.01**	34 (21.7)	54 (34.4)	69 (43.9)	**<0.01**	41 (26.1)	48 (30.6)	68 (43.3)	**<0.01**	43 (27.4)	51 (32.5)	63 (40.1)	**0.01**
Pacific n(%) n = 130	75 (57.7)	37 (28.5)	18 (13.8)		62 (47.7)	42 (32.3)	26 (20)		55 (42.3)	47 (36.2)	28 (21.5)		52 (40)	45 (34.6)	33 (25.4)	
MT-IT n(%)	37 (19.6)	68 (36)	84 (44.4)	**<0.01**	53 (28)	64 (33.9)	72 (38.1)	**0.02**	61 (32)	54 (28.4)	75 (39.5)	**<0.01**	56 (29.5)	68 (35.8)	66 (34.7)	0.60
ET n(%)	58 (59.8)	28 (28.7)	11 (11.3)		43 (44.3)	31 (32)	23 (23.7)		35 (36.1)	41 (42.3)	21 (21.6)		39 (40.2)	28 (28.9)	30 (30.9)	
Deprivation score	8 (6,9)	5 (3, 7)[a]	5 (3, 7)[a]	**<0.01**	8 (5,9)	5 (3, 8)[a]	5 (2, 7)[a]	**<0.01**	6 (4,9)	6 (3, 9)	5 (3, 8)	0.11	6 (4,9)	5 (3, 8)	5 (3, 8)	0.13
BMI (kg/m^2)	30.8 (25.1, 36.3)	29.5 (23.2, 33.5)	23.9 (22.0, 32.8)[a]	**<0.01**	31.5 (23.4, 37.1)	29.7 (23.1, 34.2)	25.3 (22.7, 31.8)[a]	**0.01**	29.2 (23.6, 33.7)	27.1 (23.0, 33.3)	29.2 (22.9, 35.8)	0.61	30.4 (23.0, 35.7)	26.8 (22.6, 32.7,)	29.7 (23.9, 34.3)	0.16
BF% [*]	37 ± 6	35 ± 7	32 ± 8[a,b]	**<0.01**[¥]	35 ± 7	35 ± 7	34 ± 7	0.20[¥]	35 ± 6	34 ± 7	34 ± 8	0.78[¥]	35 ± 7	33 ± 7	35 ± 7	0.07[¥]

NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (MT-IT) Morning Types & Intermediate Types, (ET) Evening Types,
Healthy pattern: MT-IT: n =189, ET = 97; Discretionary Food & Beverages pattern: MT-IT: n =189, ET = 97; Animal Products pattern: MT-IT: n =190, ET = 97; Sugary, Fatty Foods: MT-IT: n =189, *ET = 97,*
Values reported as Median (25th – 75th percentiles), [*]*Values are reported as means ±SD, P-value of < 0.05 was considered statistically significant*
[†]*Kruskall- Walis with multiple comparison rank tests were used to compare between three groups of continuous, non-normally distributed data,* [a] *compared against T1,* [b] *compared against T2 , adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167,*
[¥]*One-way ANOVA for continuous, normally distributed data with Tukey HSD as post hoc test,*
Chi-square test for categorical data, n(%).

6.4.2.2 Nutrient distribution between food pattern tertiles (T3 vs T1)

Women with the highest score for the *Healthy* food pattern (T3) had a significantly higher intake of total protein (11 g more) and lower intake of total carbohydrates (60 g less) in comparison with low scores (T1) **(Appendix D.2)**. This food pattern was also high in micronutrients, with high scoring women having significantly higher vitamin A, -C, -E, -B6, riboflavin, folate, potassium, magnesium, calcium, iron, iodine, and selenium intakes, while low in sodium, in comparison with women who had low scores for this food pattern. No statistically significant differences in energy and macronutrient intakes for the *Discretionary Foods & Beverages* food pattern were found, but higher vitamin -C, -E, folate, potassium, magnesium, calcium, and iodine intakes were seen among women with high scores for this food pattern in comparison with women with lower scores. For the *Animal Products* food pattern, women with the highest scores had significantly higher protein (10.9 g more, $P \leq 0.01$), and lower carbohydrate intakes (60.3 g less, $P \leq 0.01$) than those with lower scores. Furthermore, women with the highest score for the *Animal Products* food pattern had lower vitamin C intakes, while having higher vitamin A, riboflavin, folate, vitamin B12, sodium, potassium, iron, zinc, selenium, and iodine compared to women with lower scores. Women with higher scores for the *Sugary, Fatty Foods* pattern had significantly higher energy intake (1101 kJ more, $P < 0.01$), protein (9.4 g more, $P = 0.01$), fat (24 g more, $P \leq 0.01$), vitamin A, -C, -E, potassium, and magnesium intakes, with lower thiamine intakes compared to women with lower scores.

6.4.2.3 Associations between chronotype-, BMI- and BF% groups & food patterns tertiles

For chronotype groups, the odds ratio of scoring high (T3) on the *Healthy* food pattern was lower for ET women vs. earlier chronotypes (MT-IT), even after adjusting for age, deprivation index, total daily energy intake and ethnicity (OR = 0.26, 95% CI [0.11, 0.63]) (**Table 6.4**). The odds ratio for scoring high on the *Discretionary Food & Beverages and Animal Products* food patterns was also lower for ET women vs. earlier chronotypes (MT-IT), however, after adjustments, the association was no longer significant. For the BMI and BF% groups, the odds ratio of scoring high (T3) on the *Healthy* food pattern was lower for women in the high BMI (OR = 0.36, 95% CI [0.17, 0.77]) and high BF% (OR = 0.33, 95% CI [0.15, 0.69]) groups, even after adjustments. The odds ratio of scoring high on the *Sugary, Fatty Foods* pattern was higher for high BMI groups (OR = 2.09, 95% CI [1.05, 4.15], only after adjustment. *The Discretionary Food & Beverages* food and *Animal Products* food patterns were not associated with having a high BMI or BF% (adjusted or unadjusted).

Table 6.4 *- The association between chronotype-, BMI and BF% groups & food pattern tertiles 1 & 3*

a) Chronotype groups				**b) BMI groups**				**c) BF% groups**			
Univariate analysis		Multivariate analysis		Univariate analysis		Multivariate analysis		Univariate analysis		Multivariate analysis	
β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
Healthy											
-2.49	**0.08 [0.04, 0.18] ***	**-1.36**	**0.26 [0.11, 0.63] *$^{\delta}$**	**-0.35**	**0.26 [0.14, 0.48] ***	**-1.02**	**0.36 [0.17, 0.77] *$^{\delta}$**	**-1.01**	**0.37 [0.20, 0.66] ***	**-1.12**	**0.33 [0.15, 0.69] *$^{\delta}$**
Discretionary Food & Beverages											
-0.93	**0.39 [0.21, 0.73] ***	0.02	1.02 [0.42, 2.48]$^{\delta}$	-0.59	0.56 [0.31, 1.00]	-0.16	0.85 [0.44, 1.66]$^{\delta}$	-0.44	0.64 [0.36, 1.14]	-0.24	0.79 [0.42, 1.49]$^{\delta}$
Animal Products											
-0.72	**0.49 [0.26, 0.92] ***	0.01	1.01 [0.45, 2.27]$^{\delta}$	-0.18	0.84 [0.46, 1.51]	0.06	1.06 [0.56, 2.01]$^{\delta}$	-0.13	0.88 [0.50, 1.56]	-0.07	0.93 [0.50, 1.71]$^{\delta}$
Sugary, Fatty Foods											
0.04	1.04 [0.46, 2.36]	0.40	1.50 [0.73, 3.06]$^{\delta}$	0.34	1.40 [0.77, 2.55]	**0.74**	**2.09 [1.05, 4.15] *$^{\delta}$**	-0.07	0.94 [0.53, 1.66]	0.08	1.08 [0.58, 2.00]$^{\delta}$

**P-value < 0.05*

Binary Logistic regression (enter method), ***Model 1 (univariate)****: Dependent variable: Food pattern tertiles (T1 vs T3), Independent variable: a) chronotype groups of MT-IT and ET, b) normal BMI (<25 kg/m^2) and high BMI (≥25 kg/m^2) groups, c) normal (<35 %) and high (≥35 %) BF% groups,* ***Model 2 (multivariate)****: Dependent variable: Food pattern tertiles (T1 vs T3), Independent variable: a) chronotype groups of MT-IT and ET, b) normal BMI (<25 kg/m^2) and high BMI (≥25 kg/m^2) groups, c) normal (<35 %) and high (≥35 %) BF% groups, Adjustments for age, deprivation index, total daily energy intake & ethnicity. Reference category: MT-IT or normal BMI (<25 kg/m^2) or normal BF% (<35 %),*

Missing deprivation index (n = 3),

$^{\delta}$Covariate ethnicity significant in the model.

6.4.3 Principal component analysis for nutrient patterns

Next, PCA analysis was used in the same way to extract four nutrient patterns, which explained 56.3% of the variation in dietary intake among the entire study population. The Kaiser-Meyer-Olkin Measure of Sampling Adequacy was 0.69 and Bartlett's test of sphericity was significant ($P < 0.001$), verifying that the PCA was appropriate for this data set. **Table 6.5** shows the factor loadings of each nutrient, within the four nutrient patterns.

The first nutrient pattern, the *Micronutrient-Rich pattern,* explained 17.4% of the variance. This nutrient pattern was characterised by positive loadings on magnesium, potassium, vitamin E, folate, iron, vitamin A, calcium, vitamin C, and moderate thiamine, and iodine. **The second nutrient pattern, the *High Protein-Fat-Fibre,*** explained 15.4% of the variance. This nutrient pattern was characterised by positive loadings on total fat, mono-unsaturated fatty acids (MUFA), Poly-unsaturated fatty acids (PUFA), saturated fatty acids (SFA), protein, cholesterol, and fibre. **The third nutrient pattern, the *High Vitamin B,*** explained 14% of the variance. This nutrient pattern had positive loadings on niacin, vitamin B12, vitamin B6, riboflavin, zinc, sodium, and selenium. **The fourth nutrient pattern, the *High Carbohydrate,*** explained 9.4% of the variance and had positive loading on total carbohydrates, starch, and sugar, with negative loadings on cholesterol and vitamin B6.

***Table 6.5** - Rotated factor matrix of nutrient pattern*

	Nutrient Pattern:			
Nutrients	**Micronutrient Rich**	**High Protein-Fat-Fibre**	**High Vitamin B**	**High Carbohydrate**
Explanation of Variation of Nutrient Intakes (%)	17.4	15.4	14.0	9.4
Magnesium (mg)	**0.88**		0.22	
Potassium (mg)	**0.82**		0.35	
Vitamin E (mg)	**0.77**			
Folate (µg)	**0.75**		0.24	
Iron (mg)	**0.70**		0.37	
Vitamin A (µg)	**0.66**			
Calcium (mg)	**0.62**		0.29	
Vitamin C	**0.54**			
Thiamine (mg)	**0.39**		0.22	
Iodine (µg)	**0.21**			
Fat (g)		**0.93**		0.24
MUFA (g)		**0.90**		
Protein (g)		**0.78**		
SFA (g)		**0.76**		0.29
PUFA (g)		**0.73**		

	Nutrient Pattern:			
Nutrients	**Micronutrient Rich**	**High Protein-Fat-Fibre**	**High Vitamin B**	**High Carbohydrate**
Cholesterol (g)		**0.66**		-0.28
Fibre (g)		**0.53**		
Alcohol (g)				
Niacin (mg)	0.22		**0.85**	
Vitamin B12			**0.84**	
Vitamin B6 (mg)			**0.75**	
Riboflavin (mg)	0.34		**0.72**	
Zinc (mg)	0.42		**0.61**	
Sodium (mg)			**0.53**	
Selenium (µg)	0.34		**0.45**	
Carbohydrates (g)		0.26		**0.95**
Starch (g)		0.28		**0.82**
Sugars (g)				**0.82**

(SFA) Saturated Fatty Acids, (PUFA) Poly-Unsaturated Fatty Acids, (MUFA) Mono-Unsaturated Fatty Acids,
Patterns identified based on factor loadings >0.2; food groups with factor loadings <0.2 not shown,
Nutrients which cross-loaded on multiple food patterns are shown in the table, with the highest factor loading indicated in bold, and only used once across the four patterns.
Kaiser-Meyer-Olkin Measure of Sampling Adequacy = 0.69,
Bartlett's test of sphericity: P < 0.001.

6.4.4 Demographics, body composition, & chronotype distributions across the nutrient patterns tertiles

Women with the highest score (T3) on the *Micronutrient Rich* nutrient pattern had a lower BMI (25.5 (22.5, 32.0) vs 32.2 (25.5, 35.8) kg/m^2) compared with those with the lowest score (T1) ($P < 0.01$). A higher proportion of the MT-IT (37.9 %) scored high on the *High Protein-Fat-Fibre* nutrient pattern in comparison with the proportion of ET (27.7 %) although not significant ($P = 0.05$). For the High Vitamin B nutrient pattern, women with the highest score had a higher BMI (30.5 (23.7, 36.6) vs 26.3 (22.8, 32.8)) in comparison with those with the lowest score ($P = 0.03$). Significantly more of the Pacific women (45.4 %), ET (43.3 %) and those with highest deprivation index score (T3) 7 (4, 9) and fewer of the NZ European women (24.2 %) and MT-IT (28.9 %) scored high on the High Carbohydrate nutrient pattern ($P < 0.01$, **Table 6.6**).

Table 6.6 *- Participant characteristics per nutrient pattern tertiles*

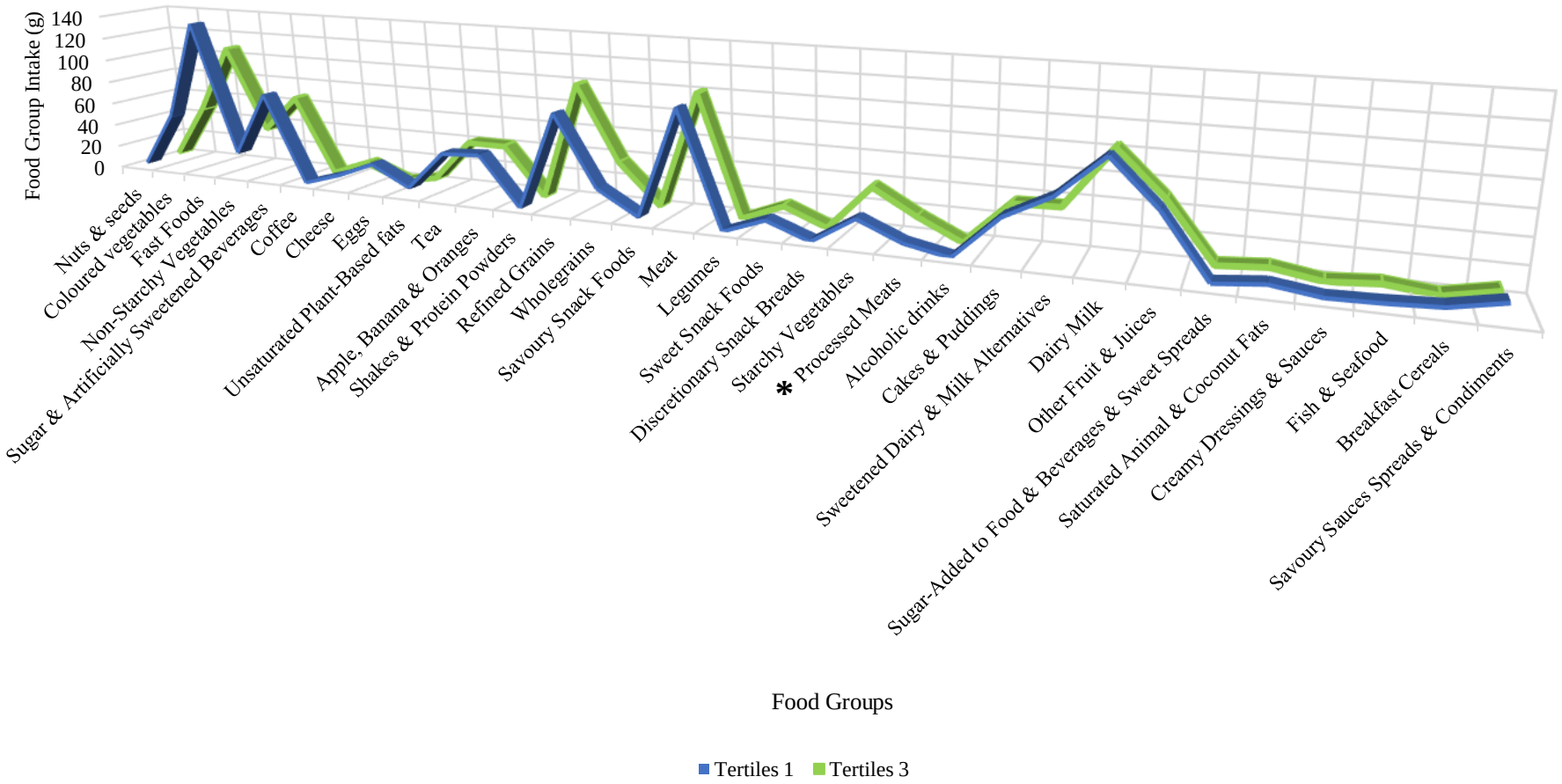
	Micronutrient Rich				**High Protein-Fat-Fibre**				**High Vitamin B**				**High Carbohydrate**			
	T1	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]	**T1**	**T2**	**T3**	*P*-value[†]
Age (years)	25 (22, 35)	29 (23, 34)	28 (22, 36)	0.39	28 (22, 34)	29 (23, 37)	25 (22, 35)	0.25	27 (22, 36)	26 (22, 34)	28 (22, 34)	0.58	26 (22, 34)	29 (23, 36)	26 (22, 35)	0.08
NZ European n (%) n = 157	48 (30.6)	49 (31.2)	60 (38.2)	0.28	46 (23.3)	54 (34.4)	57 (36.3)	0.11	50 (31.8)	50 (31.8)	57 (36.3)	0.53	68 (43.3)	51 (32.5)	38 (24.2)	**<0.01**
Pacific n (%) n = 130	46 (35.4)	46 (35.4)	38 (29.2)		50 (38.5)	41 (31.5)	39 (30)		45 (34.6)	46 (35.4)	39 (30)		28 (21.5)	43 (33.1)	59 (45.4)	
MT-IT n (%) n = 190	62 (32.6)	64 (33.7)	64 (33.7)	0.95	56 (29.5)	62 (32.6)	72 (37.9)	0.05	66 (34.7)	58 (30.5)	66 (34.7)	0.34	76 (40)	59 (31.1)	55 (28.9)	**<0.01**
ET n (%) n = 97	32 (33)	31 (32)	34 (35.1)		40 (41.2)	33 (34)	24 (24.7)		29 (29.9)	38 (39.2)	30 (30.9)		20 (20.6)	35 (36.1)	42 (43.3)	
Depriva-tion score	5 (3, 8)	6 (4, 9)	6 (3, 8)	0.36	6 (4, 9)	6 (3, 9)	5 (3, 8)	0.29	5 (3, 8)	6 (4, 8)	6 (3, 9)	0.47	5 (2, 8)	5 (3, 8)	7 (4, 9)[a]	**<0.01**
BMI (kg/m^2)	32.2 (25.5, 35.8)[b]	25.5 (22.8, 33.5)	25.5 (22.5, 32.0)[a]	**<0.01**	27.2 (23, 34)	28.9 (23.1, 34.3)	29.8 (23, 33.7)	0.86	26.3 (22.8, 32.8)	28 (23.1, 33.5)	30.5 (23.7, 36.6)[a]	**0.03**	26.7 (22.8, 32.2)	28.5 (23, 34.6)	30.7 (23.8, 35.8)	0.12
BF% [*]	34 ± 7	35 ± 7	35 ± 7	0.50[ɣ]	36 ± 8	34 ± 7	34 ± 7	0.16[ɣ]	34 ± 7	34 ± 7	35 ± 7	0.60[ɣ]	35 ± 8	35 ± 7	34 ± 7	0.35[§]

(NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (MT-IT) Morning Types & Intermediate Types, (ET) Evening Types,
Values reported as Median (25th – 75th percentiles),[*]Values are reported as means ±SD, P-value of < 0.05 was considered statistically significant,
[†]Kruskall- Walis with multiple comparison rank tests were used to compare between three groups of continuous, non-normally distributed data, [a] compared against T1, [b] compared against T2 , adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167,
[ɣ]One-way ANOVA for continuous, normally distributed data with Tukey HSD as post hoc test,
Chi-square test for categorical data, n(%)
Missing deprivation index (n = 3)

6.4.5 Food distribution across nutrient pattern tertiles

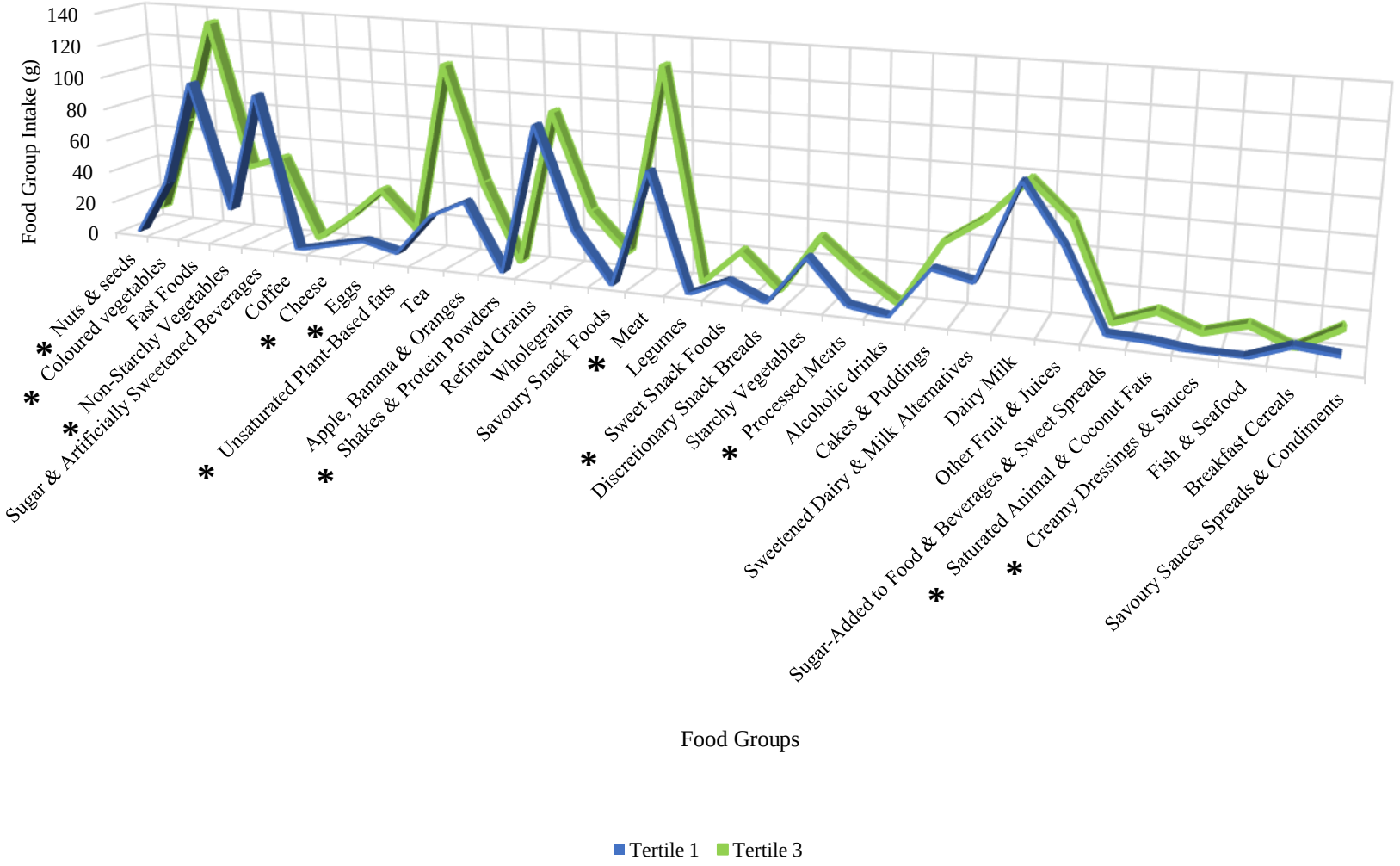
Women scoring high on the *Micronutrient Rich* nutrient pattern had significantly higher intakes of processed meats, as well as a high intake of starchy vegetables, coloured vegetables, wholegrain and meats, although intakes were not significantly different (**Figure 6.2**). For the *High Energy Protein-Fat-Fibre* nutrient pattern, women with the highest score (T3) had a significantly higher intake of coloured and non-starchy vegetables, nuts and seeds, unsaturated plant-based fats, saturated animal & coconut fats, meat, processed meats, eggs, shakes & protein powders, cheese, creamy dressings & sauces, savoury and sweet snack foods in comparison with those with the lowest score (T1) (P <0.05, **Figure 6.3**). No significant differences between high and low scores were found for the *High Vitamin B* nutrient pattern, although women with high scores had higher intakes of vitamin-B rich foods including eggs, fruits & juices and fish & seafood (**Figure 6.4**). Those with the highest score on the *High Carbohydrate* nutrient pattern had a significantly higher intakes of fast foods, sugar & artificially sweetened beverages, refined grains, savoury snack foods, cakes & puddings, other fruits & fruit juices, dairy milk, sugar-added to foods & sweet spreads, and breakfast cereals, while having a lower intake of coloured and non-starchy vegetables, coffee, tea, unsaturated plant-based fats, cheese, alcohol, eggs, and shakes & protein powders, in comparison with those with the lowest score (**Figure 6.5 & Appendix D.3)**.

Figure 6.2 - Food distribution across Micronutrient-Rich pattern tertiles (T1, T3)



*T3 vs T1 statistically significant ($P < 0.05$)

Figure 6.3 *- Food distribution across High Protein-Fat-Fibre pattern tertiles (T1, T3)*



*T3 vs T1 statistically significant ($P < 0.05$)

Figure 6.4 *- Food distribution across High Vitamin B pattern tertiles (T1, T3)*

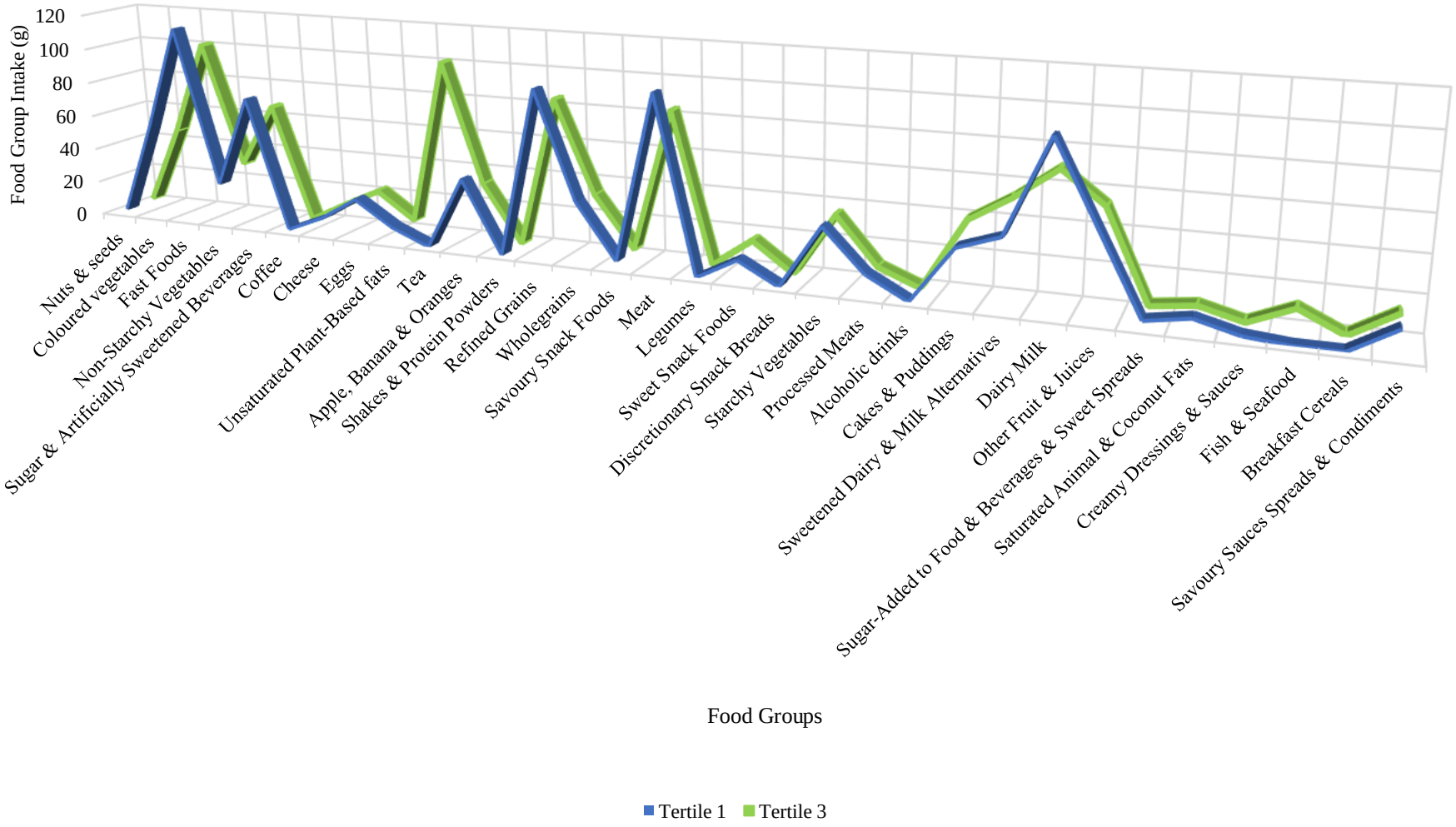
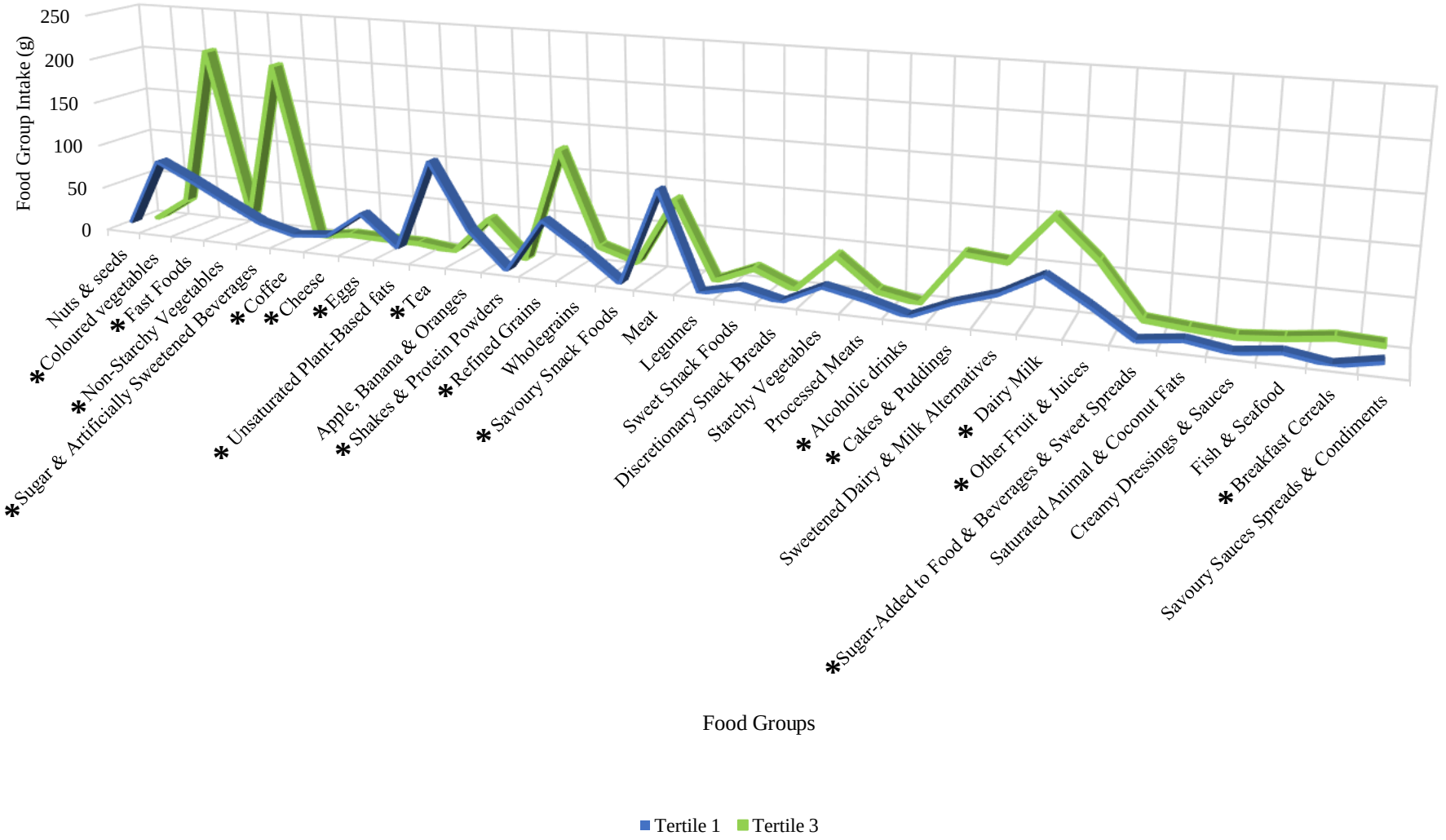


Figure 6.5 *- Food distribution across High Carbohydrate pattern tertiles (T1, T3)*



6.4.6 Associations between chronotype, body composition groups and nutrient patterns tertiles (T3 vs T1)

The odds ratio of scoring high (T3) on the *High Protein-Fat-Fibre* nutrient pattern was lower for ET women vs earlier chronotypes (MT-IT), and in contrast was higher on the *High Carbohydrate* pattern for ET vs. MT-IT women (**Table 6.7**). After adjustment, however, both these associations were no longer significant. For the BMI groups, the odds ratio of scoring high on only the *Micronutrient Rich* nutrient pattern decreased for women in the high BMI (OR = 0.34, 95% CI [0.18, 0.64]), but only after adjustment. None of the other nutrient patterns were significantly associated with BMI groups. For the BF% groups, the odds ratio for scoring high (T3) on the *Micronutrient Rich* pattern was lower for high BF%, for both unadjusted and adjusted models (OR = 0.41 [0.23, 0.73] vs OR = 0.39 [0.21, 0.70]). The *High Vitamin B* nutrient pattern was not associated with any groups (adjusted or unadjusted).

Table 6.7 *- The association between chronotype-, BMI and BF% groups & nutrient pattern tertiles 1 & 3*

a) Chronotype groups				**b) BMI groups**				**c) BF% groups**			
Univariate analysis		Multivariate analysis		Univariate analysis		Multivariate analysis		Univariate analysis		Multivariate analysis	
β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
Micronutrient Rich Pattern											
0.03	1.03 [0.57, 1.87]	0.31	1.37 [0.64, 2.92]	-1.06	0.35 [0.19, 0.64] *	**-1.09**	**0.34 [0.18, 0.64] ***	**-0.90**	**0.41 [0.23, 0.73] ***	**-0.95**	**0.39 [0.21, 0.70] ***
High Protein-Fat-Fibre Pattern											
-0.76	**0.47 [0.25, 0.86] ***	-1.42	0.24 [0.04, 1.42]$^{\delta}$	0.04	1.05 [0.59, 1.87]	0.31	1.37 [0.39, 4.75]$^{\delta}$	0.24	1.27 [0.78, 2.08]	-0.06	0.94 [0.49, 1.83]
High Vitamin B Pattern											
0.03	1.03 [0.56, 1.91]	0.39	1.47 [0.66, 3.28]	0.64	1.90 [1.05, 3.43]	0.74	2.09 [1.13, 3.86]	0.53	1.69 [0.96, 3.00]	0.60	1.82 [1.01, 3.31]
High-Carbohydrate Pattern											
1.07	**2.90 [1.54, 5.48] ***	0.52	1.69 [0.58, 4.93]	0.28	1.32 [0.74, 2.36]	0.24	1.27 [0.59, 2.73]$^{\delta}$	0.56	1.76 [0.99, 3.11]	0.38	1.46 [0.68, 3.10]$^{\delta}$

**P-value < 0.05*

Binary Logistic regression (enter method), ***Model 1 (univariate)****: Dependent variable: Nutrient pattern tertiles (T1 vs T3), Independent variable: a) chronotype groups of MT-IT and ET, b) normal BMI (<25 kg/m^2) and high BMI (≥25 kg/m^2) groups, c) normal (<35 %) and high (≥35 %) BF% groups,* ***Model 2 (multivariate)****: Dependent variable: Nutrient pattern tertiles (T1 vs T3), Independent variable: a) chronotype groups of MT-IT and ET, b) normal BMI (<25 kg/m^2) and high BMI (≥25 kg/m^2) groups, c) normal (<35 %) and high (≥35 %) BF%, groups. Adjustments for age, deprivation index, total daily energy intake & ethnicity. Reference category: MT-IT or normal BMI (<25 kg/m^2) or normal BF% (<35 %), Missing deprivation index (n = 3),*

$^{\delta}$Covariate ethnicity significant in the model.

6.5 Discussion

This study identified four each food patterns and nutrient patterns, with the nutrient patterns explaining more of the variance (56.3%) in the dietary intake than the food patterns (27.4%). Most women with an earlier chronotype (MT-IT) followed dietary (food and nutrient) patterns that consisted of healthy foods (such as coloured vegetables, wholegrains, apple, banana and oranges), higher micronutrients, protein, fat, and fibre content while lower in carbohydrates namely, the *Healthy* and *Animal Products* food patterns and the *High Protein-Fat-Fibre* nutrient pattern. In contrast, most ET women followed the *High Carbohydrate* nutrient pattern, which is characterised by high carbohydrate, starch and sugar nutrient intakes.

Both the *Healthy food* pattern and the *High Protein-Fat-Fibre* nutrient pattern were characterised by a high dietary fibre intake. The high fibre content of the nutrient pattern was derived from food sources such as coloured- and non-starchy vegetables, which was reflected in the food pattern that was characterised by factor loadings for coloured- and non-starchy vegetables, fruits, and wholegrains. It is well known that a high fruit and vegetable intake is associated with a higher fibre intake, which is protective against weight gain and obesity by enhancing satiety and lowering overall energy intake (Rolls et al. 2004, Svendsen et al. 2007, Rolls et al. 2010). A recent meta-analysis also showed that higher fibre intakes are associated with lower total cholesterol concentrations and systolic blood pressure and protective against cancer, type two diabetes and cardiovascular diseases (Reynolds et al. 2019). Indeed, the *Healthy* food pattern was associated with a lower BMI and BF%. This typical higher intake of fibre-rich foods such as fruit, vegetables, and wholegrains in early chronotypes have also been reported by others (Baron et al. 2011, Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020). A NZ based study identified a "Fruit and Vegetable" and "Healthy" pattern, that shares similarities with our *Healthy* food pattern in this study (Schrijvers et al. 2016). The major differences were that our food pattern was lower in wholegrains, legumes, non-starchy vegetables, coconut fats, and excluded breakfast cereals, starchy vegetables and alcohol.

The fat, cholesterol and protein nutrient content of the *High Protein-Fat-Fibre* nutrient pattern was derived from animal protein sources, saturated fats from animal & coconut sources (e.g., cream) and unsaturated plant-based fat sources (e.g., sunflower oil). The same profile was also captured by the *Animal Products* food pattern, that had high factor loadings for animal protein sources (meats, eggs, cheese, fish & seafood, and dairy milk) as well as the *Healthy* food pattern that had high factor loadings for meat and unsaturated plant-based fats (e.g., canola oil). This finding is contradictory to the majority of chronotype studies, that demonstrated that ET rather than MT were more likely to consume a diet high in fats such as butter, cream, margarine (Muscogiuri et al. 2020), cholesterol-rich foods (Mota et al. 2016), and animal protein (Sato-Mito et al. 2011, Silva et al. 2016, Muscogiuri et al. 2020). Another

nutrient pattern study identified a similar pattern, called "Animal Sourced", with similar high loadings on cholesterol, fat, SFA and animal protein. Their study found their "Animal Sourced" pattern to be associated with greater odds of metabolic syndrome, especially elevated triglyceride concentrations (Vajdi et al. 2020), while this study found lower BMI and BF% for women following the *Healthy* food pattern and no association between the *Animal Products* food-, or *High Protein-Fat-Fibre* nutrient patterns and body composition.

All three of the more MT-IT patterns (*High Protein-Fat-Fibre* nutrient-, *Animal Products* food- and *Healthy* food patterns) were lower in carbohydrates and the equivalent food sources. To compensate for the lower carbohydrate intake, a higher protein and fat intakes were seen in these patterns. Seidelmann et al. investigated the association between carbohydrate intake and mortality in adults over a period of 25 years (2018). The results showed that low carbohydrate intakes were associated with a higher risk of mortality compared with balanced intakes. Mortality risk increased when carbohydrates in the diet were substituted with animal protein and -fats, versus when carbohydrates were replaced with plant-based proteins and fats, thereby showing that that the food source of protein and fats modified the association between carbohydrate intakes and mortality risk (Seidelmann et al. 2018).

More MT-IT women also followed the *Discretionary Foods & Beverages* food pattern. This pattern was similarly lower in carbohydrate foods (e.g., starchy vegetables) to the other MT-IT patterns except for legumes and other fruit and juices, and discretionary snack breads (e.g., waffles, muffins, and scones) which are low-quality carbohydrate sources. The high fish & seafood and legume foods found in this food pattern were also reported among early chronotypes by others (van der Merwe et al. 2022). The food pattern was dominated by higher intakes of beverages (alcohol, milk alternatives, fruit juice and to a lower extent coffee and tea). The higher alcohol and coffee intake were not expected in MT-IT, as others have reported that ET consumed a higher amount of these beverages than MT (Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Vera et al. 2018, Najem et al. 2020). It has been suggested that ET consumed more caffeine to prolong wakefulness during night hours and that higher alcohol intake can be used as a method to release tension and aid in sleep onset in ET (Adan 1994, Wittmann, Paulus et al. 2010). More NZ European than Pacific women scored high on this food pattern. According to the NZ National Healthy Survey 2018/19, Pacific women were the least likely (29 %) to consume alcohol in comparison with other ethnic groups (Ministry of Health 2019), suggesting that ethnicity was an influencing factor for the high alcohol intakes seen in this pattern.

More ET than MT-IT followed the *High Carbohydrate* nutrient pattern, which was characterised by high carbohydrates, sugars, and starch intakes. Previous nutrient pattern analysis studies have identified a similar pattern high in carbohydrates named "starch rich" that was associated with increased pancreatic cancer risk (Bosetti et al. 2013). High carbohydrate intakes have also been associated with a

higher risk of mortality compared with balanced carbohydrate intakes in adults (Seidelmann et al. 2018). The food groups that contributed to the high carbohydrate content of this pattern included poor quality carbohydrate sources (such as sugar- & artificially sweetened beverages, refined grains, cakes & puddings). It is well known that good quality carbohydrates that are high in fibre and with low glycaemic index are associated with a reduced risk of obesity (Aller et al. 2011). While a diet rich in simple carbohydrates, such as sweets, biscuits and sugar-sweetened beverages are associated with increased obesity risk (Malik et al. 2010). Previous studies have reported similar high intakes of energy-dense foods such as confectionery and sweets (Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Muscogiuri et al. 2020), sugar-sweetened beverages (Baron et al. 2011, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020) in ET. Sugar- and artificially sweetened beverages for this more ET-dominated pattern had a median intake of 195 g for those with high scores, while another study by Yoshizaki et al. (2018) reported ET to consume 60.8 g per day. Interestingly this pattern had a negative loading for dietary cholesterol, which is contradictory to other studies that have found that ET consumed a high amount of cholesterol containing foods (Sato-Mito et al. 2011, Silva et al. 2016, Mota et al. 2016, Muscogiuri et al. 2020). This pattern is similarly high in added sugars and sweet snack foods as seen in the food pattern identified in this study named the *Sugary, Fatty Foods* pattern.

In this study two micronutrient containing patterns were extracted, the *Micronutrient-Rich* and *High Vitamin-B* nutrient patterns. This is similar to other studies that have found distinct micronutrient patterns that was inversely associated with cancer risk (Palli et al. 2001, Bosetti et al. 2013). The micronutrient dense foods from the *Healthy* food pattern (e.g., vegetables, fruits, nuts & seeds, and wholegrains) reflected the high loadings of magnesium, potassium, vitamin E, -A, -C, folate, iron, iodine, calcium, and some of the vitamin Bs for the *Micronutrient Rich* nutrient pattern. The high factor loading for animal foods from the *Animal sourced* food pattern reflected the high loadings for niacin, vitamin B12, -6, riboflavin, zinc, and sodium of the *High Vitamin B* nutrient pattern. Chronotype was not associated with either of these two micronutrient patterns. This lack of association may be due to the very low and often inadequate intakes of micronutrients found among this population group (Ministry of Health 2008/09). However, the food patterns that were followed by more of the MT-IT were higher in micronutrients, derived from the variety of micronutrient dense foods such as meat, fruits, vegetables, nuts & seeds, legumes, eggs, and cheese to name a few. This higher micronutrient intake in earlier chronotypes have also been reported by others (Sato-Mito et al 2011, Kanerva et al. 2012), mirroring the higher intakes of micronutrient-dense foods seen in this study.

6.6 Strengths & limitations

To our knowledge dietary pattern analysis has not been explored in chronotypes before, nor has both food and nutrient patterns been explored in the same study. This study uniquely contributes a comprehensive picture of chronotype's eating patterns beyond total dietary intakes. This study was limited by its cross-sectional design and the small sample size of MT. Although this is to be expected in a young study population, the MT and IT were combined for the purpose of statistical analysis. Statistically significant differences were seen in the distribution of ethnicity between the dietary patterns; however, the analysis could not be stratified by ethnicity due to a small sample size of MT in the Pacific group (n = 7) as well as the NZ European group (n = 29).

6.7 Conclusion

This study is the first to create dietary patterns, both food- and nutrient, among different chronotypes. In comparison to the food patterns, the nutrient patterns explained most of the variance seen in the dietary intake of this NZ population. The nutritional composition of the foods in the food patterns were reflected in the nutrient patterns. Similar to previous research, MT-IT were more likely to consume healthier foods (such as fruits, vegetables, and wholegrains) and nutrients such as MUFA, PUFA and fibre, but contrary than reported by others also consumed more coffee, alcohol, animal products and cholesterol. The ET consumed more simple carbohydrates, starch and sugar derived from food sources such as sweet snack foods, cakes & puddings. These results showed that both food and nutrient pattern analyses are valuable tools to assess links between diet, chronotype and body composition.

This chapter investigated the differences in dietary patterns between different chronotypes and therefore provided a whole diet picture beyond total food group and total nutrient intakes. The following chapter (seven) will focus on the eating behaviours and eating attitudes that precede and drive food intake.

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7 Chapter 7: Are unfavourable eating behaviours related to chronotype, body composition, hunger & satiety hormones in women?

Abstract

Background and aim: Individuals with an evening chronotype (ET) are prone to misalignment between their internal body clocks and external clock times of daily life, resulting in unfavourable eating behaviours and eating attitudes that may be associated with weight gain, adiposity and hunger and satiety hormones. The aim of this study was to investigate whether chronotype is associated with eating behaviours, eating attitudes, hunger and satiety hormones (ghrelin and leptin) and body composition among New Zealand (NZ) European and Pacific women.

Methods: Healthy women aged 18-45 years (n = 304; 142 Pacific and 162 New Zealand European, NZ European ethnicity) were recruited based on high and normal BMI kg/m^2. Height and weight were measured to calculate BMI, and dual-energy x-ray absorptiometry (DXA) was used to assess total body fat percentage (BF%). Chronotype was assessed using the Munich Chronotype Questionnaire (MCTQ), the Three Factor Eating Questionnaire (TFEQ) and the Eating Attitude Test -26 (EAT-26) to assess eating behaviour and eating attitudes respectively. Fasting venous blood samples were collected to assess leptin and ghrelin concentrations. Morning- (MT) and intermediate types (IT) were combined as MT-IT group to compare with ET. Linear regression models assessed associations between chronotype, eating behaviour and eating attitudes stratified by BMI and BF%, and adjusted for ethnicity, age and deprivation index.

Results: Approximately half of participants were classified as intermediate type (IT; n = 155, 54%) followed by ET (n = 97, 34%) and morning type (MT; n = 35, 12%), with most Pacific women being ET (n = 83, 86%) and most NZ Europeans being ITs (n = 115, 65%). The MT compared with ET had a lower median BMI 24 (22, 31) vs 31 (25, 37) kg/m^2) and BF% (32 ± 9 vs 36 ± 7 %), P<0.001 and P <0.02 respectively). Total daily energy, protein and fat intakes were similar across the three chronotypes (P >0.05), however, mean carbohydrate intake was higher in ET (223.1 ± 76.5 g) vs IT (191.7 ± 63.9 g, P <0.01). Median leptin concentrations were lower in MT (7210 (2716, 14683) pg/mL) than ET (15678 (7683, 23196) pg/mL, P <0.01). Ghrelin concentrations were higher in MT (74.8 (45.4, 120.8) pg/mL) vs ET (28.1 (17.3, 58.7) pg/mL), P <0.01. In the high BMI group, ET was associated with lower *restraint* (β = -1.86, P = 0.03), including *flexible* (β = -0.84, P = 0.01) and *rigid control* (β = -0.75, P = 0.04), but higher perceived *hunger* (β = 1.64, P = 0.01) and *internal locus for hunger* (β = 0.97, P = 0.01) only, and both higher *habitual disinhibition* (β = 0.66, P = 0.02) and *bulimia & food preoccupation* (β = 1.61, P <0.01), compared with MT-IT. In the high BF% group, ET was associated with higher *internal locus for hunger* (β = 0.38, P = 0.03), *bulimia & food preoccupation* (β = 1.75, P <0.01) and *oral control* (β = 0.29, P = 0.01) scores compared with MT-IT. ET with high BMI was associated with

increased odds for the eating behaviours combinations of *low restraint-high disinhibition (*OR = 3.93, 95% CI, [1.20, 12.8]), whilst ETs with a normal BMI were associated with decreased odds of h*igh hunger-high disinhibition* combination (OR = 0.16, 95% CI [0.03, 0.88]).

Conclusion: Pacific and NZ European women with an ET are prone to display unfavourable eating behaviours and eating attitudes leading to less control over their food intake and disordered dietary intake that favours an eating pattern that promotes weight gain and obesity. A higher body composition was associated with disturbances in hunger and satiety hormones (ghrelin and leptin), which may further exacerbate the unfavourable eating behaviours and eating attitudes found in ET.

Key words: Obesity; adiposity; eating habits; circadian; eveningness; morningness; eating attitudes; appetite regulating hormones; eating disorders

7.1 Introduction

Obesity prevalence has been rising rapidly with long-term successful weight loss interventions lacking (Schwartz et al. 2017). Individuals who are prone to weight gain are more vulnerable to problematic eating behaviours and disturbed eating attitudes (Dykes et al. 2004, de Lauzon-Guillain et al. 2006, Kruger et al. 2016). It is well known that eating behaviours affects energy intake by influencing the types and amounts of foods eaten, timing of food intake and where food intake occurs, ultimately affecting body weight (French et al. 2012). The Three Factor Eating Questionnaire (TFEQ) was developed in the 1980s to assess eating behaviours, while differentiating between normal weight and overweight or obese individuals. The questionnaire examines three eating behaviours domains, namely cognitive eating *restraint*- which is the conscious restriction of food intake to control body weight and shape; *disinhibition* of control- the loss of control of food intake that leads to overconsumption of food; and susceptibility to *hunger*- feelings and subjective perceptions of *hunger* that leads to food intake (Stunkard & Messick 1985). These eating behaviours are predictors of body composition (BMI and adiposity) (Dykes et al. 2004, de Lauzon-Guillain et al. 2006, Kruger et al. 2016), weight gain (Provencher et al. 2003, Kruger et al. 2016), and undesirable metabolic health outcomes (such as poor glycaemic control) (Martyn-Nemeth et al. 2014). This suggests that the association of diet with body weight may be mediated in part by dieting behaviour (Fleurbaix Laventie Ville Sante Study Group 2004). Dieters often use eating behaviours such as eating *restraint* as a strategy to control excess body weight (Tuschl et al. 1990). However, eating *restraint* and loss of eating control may have negative consequences such as the development of eating disorders (Allison & Heshka 1993), a condition frequently overlooked when assessing obesity risk (Rukavishnikov et al. 2021). The EAT (Eating Attitude Test)-26 is a validated tool that assesses eating attitudes related to eating disorders (Garner et al. 1982). Disturbed eating attitudes has been linked with the three main eating behaviours domains namely, *restraint* (Linardon & Mitchell 2017), *hunger* and *disinhibition* (Keeler et al. 2015, Vinai et al. 2015) as well as an increased obesity risk (Marchesini et al. 2004, Rukavishnikov et al. 2021).

A growing number of studies have shown a link between the circadian timing system and especially the influence of circadian preference with obesity and dietary outcomes. Individuals display their inter-individual differences by structuring their behaviour in terms of their preferred timing for sleep and wakefulness within the normal 24-hour day. Chronotype is the term used to describe these inter-individual differences of diurnal sleep-wake preferences, which is also based on endogenous biological rhythm control, (Ehret 1974, Horne 1976, Roenneberg et al. 2003, Roenneberg et al. 2007, Wirz-Justice 2007). Individual chronotypes lie in a range between morning types (MT) and evening types (ET) where MT habitually prefer an early bedtime and early morning rise time, and ET, on the other hand, prefer a later bedtime and a late morning rise time and intermediate types (IT) which are in between these two (Ehret 1974, Horne 1976, Adan et al 2012). Different chronotypes have different phases angles of

entrainment i.e., the time range between their sleep wake timing and external clock times (Roenneberg et al. 2007). In clock hours, this occurs earlier in MT (by several hours) in comparison with ET (Lack et al. 2009). Therefore, extreme ET may experience stronger misalignment between their internal body clocks and work and social demands (Wittmann et al. 2006), because most office and work hours begin early in the morning. This may result in adverse health outcomes (Covassin et al. 2016), including weight gain and obesity (Merikanto et al. 2013, Covassin et al. 2016). Apart from altered sleep-wake patterns (Wittmann et al. 2006), this misalignment may also be the result of or cause distinct dietary risk patterns (such as poor food choices) (Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Muscogiuri et al. 2020) and delayed meal timing (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019)) as well as the unfavourable eating behaviours (Schubert & Randler 2008, Lázár et al. 2012, Meule et al. 2012, Lai & Say 2013, Vera et al. 2018, Beaulieu et al. 2020) and eating attitudes (Natale et al. 2008, Schmidt & Randler 2010, Harb et al. 2012, Muñoz et al. 2017) that accompanies and influences dietary intake (van der Merwe et al. 2022).

It has been suggested that the link between chronotype and eating behaviours may be mediated by leptin and ghrelin since these hormones are known to show circadian rhythmicity (Takahashi et al. 1968, Cipolla-Neto et al. 2014). In normal conditions, leptin secretion is upregulated during the biological night (Cipolla-Neto et al. 2014), decreasing food intake, while ghrelin concentrations are at its highest during the day, promoting food intake. The satiety hormone, leptin acts on the hypothalamus by inhibiting, the neurotransmitter, neuropeptide Y (NPY) which results in a decrease in appetite and cessation of food intake (Klok et al. 2007). However, in obese individuals this is not the case despite having elevated leptin levels (Morioka et al. 2016). Conversely, ghrelin, the appetite-stimulating hormone, is released from the stomach in response to fasting or low energy intake, binding to receptors in the hypothalamus. As a result, NPY is released, which in turn triggers feelings of *hunger* leading to food intake. In obese individuals, ghrelin regulation is disrupted showing decreased concentrations (Klok et al. 2007). Any imbalances of these two hormones have drastic consequences on energy homeostasis and therefore body weight regulation (Morioka et al. 2016). Since leptin and ghrelin display a marked morning-evening difference, it stands to reason that there may be differences in the concentrations of these hormones between the different chronotypes (Takahashi et al. 1968, Cipolla-Neto et al. 2014).

To date, the association between chronotype, eating behaviours (Schubert & Randler 2008, Lázár et al. 2012, Meule et al. 2012, Lai & Say 2013, Vera et al. 2018, Beaulieu et al. 2020), eating attitudes and related behaviours (Natale et al. 2008, Schmidt & Randler 2010, Harb et al. 2012, Muñoz et al. 2017) have largely been unexplored, especially by using the TFEQ (Schubert & Randler 2008) and EAT-26 (Harb et al. 2012, Muñoz et al. 2017) investigating specific behaviours. The studies that have utilised

the TFEQ solely investigated the three main categories namely *disinhibition, restraint* and *hunger* only (Schubert & Randler 2008) or the total TFEQ score (Beaulieu et al. 2020), while those that used the EAT-26 (Harb et al. 2012, Muñoz et al. 2017), only explored the total score of this test. Both of these questionnaires/tests consist of several subcategories that can be used to create a comprehensive eating behaviour (Stunkard & Messick 1985, Westenhoefer et al. 1999, Lesdéma et al. 2012) and eating attitude profile among chronotypes (Garner et al. 1982). The study by Schubert et al have found that ET tended to display more unfavourable eating behaviours such as higher perceived *hunger* and *disinhibition* (2008). On the other hand, MT seemed to have better control over their eating behaviours and to have higher *restraint* and *flexible dietary control* strategies (Schubert & Randler 2008), while another study found no association between chronotype score and TFEQ score (Beaulieu et al. 2020). From the two studies that investigated eating attitudes in chronotypes, one study found a positive correlation between ET and a higher EAT-26 score (Harb et al. 2012), whilst the other found no differences in total score between MT and ET (Muñoz et al. 2017). Furthermore, to our knowledge, the relationship between chronotype, eating behaviours, eating attitudes, hunger and satiety hormones and body composition have been unexplored (van der Merwe et al. 2022). Therefore, the aim of this study was to investigate the association of chronotypes with eating behaviours, eating attitudes, leptin, ghrelin, and body composition among healthy NZ European and Pacific women with different body composition profiles.

7.2 Methods

7.2.1 Study design, participants & procedure

The cross-sectional PROMIsE study aimed to recruit 272 general healthy women between the ages of 18 and 45 years – 136 of NZ European and 136 of Pacific ethnicity. For each ethnicity, participants were categorised into two classes of BMI, those classified as 'normal or healthy' BMI 18.5 -24.9 kg/m^2 and those classified as 'obese' BMI ≥30 kg/m^2, with the aim to recruit 68 women in each group. This study population is of special interest for the reasons that obesity and overweight prevalence in NZ are increasing and is strongly related to ethnicity and socioeconomic inequalities (Ministry of Health 2019). Participants were excluded if they were pregnant or lactating; diagnosed with chronic illness (e.g., diabetes and cardiovascular disease); underwent bariatric surgery; had severe food allergies; used medication that could interfere with appetite or the immune system (e.g., appetite suppressants and corticosteroids); were current smokers or had severe dietary restrictions or avoidances (e.g., vegan). The final recruited sample were 304 women of which 162 were of NZ European and 142 of Pacific ethnicity. For the purpose of this study night shift workers (n = 17) were also excluded for the reason that these individuals exhibit altered sleep-wake and fasting-feeding cycles due to work obligations and not necessarily because of their chronotype. The final analysis included 287 participants of which 157 were NZ European and 130 were Pacific women (**Figure 7.1**). Participants visited the Massey

University Human Nutrition Research Unit in Auckland, NZ, between July 2016 and September 2017 for data collection. Written informed consent was obtained from all participants. Ethics approval was obtained from the Southern Health and Disability Ethics Committee (16/STH/32).

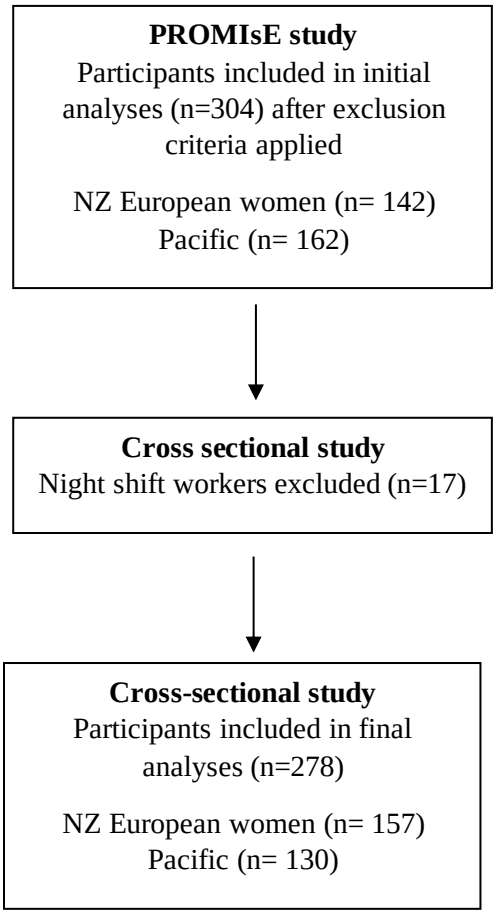

Figure 7.1 - Flow chart of participants included in analysis
(NZ) New Zealand

7.2.2 Anthropometrics measurements

All anthropometrics measurements were conducted according to the International Society for the Advancement of Kinanthropometry (ISAK) protocols by level one ISAK trained research staff (Stewart et al. 2011). Height and weight were used in the Quetelet index (weight/height^2) to determine BMI (stratified as low < 25 kg/m^2 or high ≥ 25 kg/m^2). Body composition measurements were performed using dual-energy x-ray absorptiometry (DXA; Hologic QDR Discovery A, Hologic Inc with APEX V. 3.2 software) to assess body fat percentage (BF%; stratified as normal <35% or high ≥35%) (Fitch and Bays 2022). Recognising that BMI does not accurately reflect adiposity on individual level, both BMI and BF% was measured to create a comprehensive body composition profile (De Lorenzo et al. 2016).

7.2.3 Demographic information & deprivation index

Demographic information, including health information, age and work patterns, was collected through individual interviews. The NZ Deprivation Index 2013 (NZDep2013) which is determined by combining census information such as home ownership, housing, qualifications, employment, income, access to transport, communications, and family structure, was used as a measure of socio-economic status. The deprivation scores are grouped into deciles, where one represents the geographical area with the least and 10 the most deprived scores (Crampton et al. 2020).

7.2.4 Chronotype & night shift workers

The MCTQ, was developed by Roenneberg and co-workers (Roenneberg et al. 2003). This questionnaire assesses habitual sleep-wake times of the last four weeks by differentiating between weekdays and work-free days. The parameters involved include midsleep (i.e., the midpoint between sleep onset and waketime) on weekdays (MSW), mid-sleep on free days (MSF). For this study, MSFsc is used which is mid-sleep on free days, that is corrected for sleep duration on weekdays (MSFsc). The questionnaire uses MSFsc as a chronotype predictor. A higher MSFsc reflects a later midsleep which is an indicator for late chronotypes (ET; midsleep > 5) and vice versa for early chronotypes (MT; midsleep time < 3) and a midsleep time between 3 - 5 indicates an IT (Roenneberg et al. 2003). A retrospective seven-day, sleep and work questionnaire was used to collect information on sleep and work schedules. A shift work questionnaire was used to identify the number of hours worked per day as well as the type of shift work (day or night). Participants were excluded from the analyses if they reported night shift work.

7.2.5 Eating behaviour & eating attitudes

The TFEQ was used to determine the eating behaviours profiles of participants, with a total of 51 items. It examines three eating behaviour domains, namely cognitive eating restraint (21 items); disinhibition (16 items) and hunger (14 items). All responses were scored as 0 or 1, with a higher score indicating higher levels of said eating behaviour (Stunkard & Messick 1985). Each of the three eating behaviour domains include sub-categories, namely rigid and flexible control (seven items each), habitual (five items), emotional (three items) and situational disinhibition (five items) and internal and external locus for hunger (six items each) (Westenhoefer et al. 1999). **Figure 7.2** provides a definition for each category.

Restraint and *disinhibition* were scored low (≤ 7) or high (>7) and *hunger* was scored as low (≤ 5) and high (> 5) (Lesdéma et al. 2012). From this, four eating behaviour combination categories were created for levels of *restraint* and *disinhibition* ranging from *high restraint – low disinhibition* (the ideal eating behaviour combination) to *low restraint - high disinhibition* (least ideal eating behaviour combination).

Four combination categories were also created for levels of *hunger* and *disinhibition* ranged from *low hunger – low disinhibition* (the ideal eating behaviour combination) to *high hunger - high disinhibition.*

Eating attitudes related to eating disorders were assessed by using the EAT-26, which includes a total score out of 26. The three sub-categories include: *dieting* (13 items); *bulimia & food preoccupation* (six items) and o*ral control* (seven items). Responses were rated on a six-point Likert scale and a score of ≥ 20 indicates disturbed eating attitudes with the three sub-categories creating a profile (Garner et al. 1982). **Figure 7.2** depicts the main categories as well as the various subcategories of the TFEQ and EAT-26.

Eating Behaviour Combinations

Ideal	*Least Ideal*	*Ideal*	*Least Ideal*
High Restraint	Low Restraint	**Low Hunger**	High Hunger
Low Disinhibition	High Disinhibition	**Low Disinhibition**	High Disinhibition

	Restraint-Disinhibition		Hunger-Disinhibition
TFEQ Categories	**Restraint**	**Disinhibition**	**Hunger**
	Conscious restriction of food intake to control body weight and shape	The loss of control of food intake that lead to overconsumption of food	Feelings and subjective perceptions of hunger that leads to food intake
	Flexible Restraint A non-obsessive and non-compulsive, neutral mindset to food intake control to regulate body weight	**Habitual** Particular circumstances and attitudes that habitually trigger disinhibition	**Internal Locus** Feelings of hunger that are self-translated and inner controlled
	Rigid Control A strict 'all or nothing' approach to control food intake to regulate body weight	**Situational** Environmental triggers (e.g., social events) that amplify lack of food control **Emotional** Overconsumption of food as a result of adverse mental state	**External Locus** Feelings of hunger derived from external triggers (e.g., buffets) rather that physiological cues

EAT-26 Categories

Dieting	Bulimia & Food Preoccupation	Oral Control
Preoccupation with being thinner and avoidance of fattening foods	Thoughts about food and bulimia	Self-control about food and social pressures to gain weight

Figure 7.2 - The TFEQ & EAT-26 categories (Garner et al. 1982, Stunkard & Messick 1985, Westenhoefer et al. 1999, Lesdéma et al. 2012).

7.2.6 Hunger & satiety hormones

Following an overnight fasting phase of at least 10 hours, fasting venous blood samples were collected by a trained phlebotomist between 07:30 and 09:00 to analyse ghrelin and ghrelin hormones. A tourniquet was applied to the arm of participants before venepuncture. A plasma sample (2mL Becton Dickinson vacutainer P800 EDTA, aprotinin, and dipeptidyl peptidase IV) was collected and immediately placed on ice until centrifugation. Within 1 hour of collection, the vacutainer tube was

centrifuged at 1500 g for 15 minutes at 4°C. Blood samples were analysed by the Liggins Institute, Auckland, New Zealand. Collection and processing of the blood samples have been discussed in detail elsewhere (Kindleysides et al. 2019).

7.2.7 Data handling & statistical data analysis

Analyses were conducted using IBM SPSS Statistics for Windows version 28 (Armonk, NY: IBM Corp) and a *P*-value of < 0.05 was accepted as statistically significant. To assess the association between chronotype, eating behaviours and eating attitudes, the study participants were grouped as ET (MSFsc >5), IT (MSFsc = 3 - 5) and MT (MSFsc <3). The MT and IT were combined for regression analysis due to the small MT sample. Also, chronotype groups (MT-IT and ET) were not further stratified by ethnicity as the number of participants in each chronotype group were insufficient for analysis. The study population was further stratified according to low and high body composition groups (BMI; normal <25 versus high ≥25 kg/m^2 and BF%; normal <35% versus high ≥35%) in each regression model. The distribution of the different variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests, boxplots, and histograms. Normally distributed data were presented as means and standard deviations, non-normal distributed data as medians and 25th and 75th percentiles and categorical data as frequency summary statistics. Log transformed data were back transformed to the original scale and reported as geometric mean [95% CI]. Welche's T-tests were used on normally distributed data and Mann-Whitney U tests on non-normal data for comparing two groups. When comparing more than two groups, ANOVA tests (with Tukey HSD as post-hoc tests) were used for normally distributed data and Kruskal - Wallis tests (with multiple comparison rank test, adjusted by Bonferroni correction) for non-normally distributed data.

The association between chronotype, eating behaviours and eating attitudes were assessed in two ways. Firstly, multiple regression models (enter method) were used within normal and high BMI and then within normal and high BF% groups using the main TFEQ categories and subcategories as well as the total EAT-26 score and subcategories as the dependent variable and chronotypes groups (MT-IT and ET) as the independent variable. Lastly, multinominal models were created to assess the association between chronotype and eating behaviour combinations within normal and high BMI and then within normal and high BF% groups. The eating behaviour combinations were created including the opposing normal and high categories for *restraint* (low ≤ 7 or high >7), *disinhibition* (low ≤ 7 or high >7) and for *hunger* (low ≤ 5 or high >5) (Lesdéma et al. 2012). All analysis were adjusted for age, ethnicity and deprivation index and the central limit theorem was applied. Furthermore, the correlations between MCTQ (individual midsleep times), TFEQ, EAT-26, leptin, and ghrelin were assessed using Pearson's correlation analysis.

7.3 Results

7.3.1 Study participants

After the exclusion criteria were applied 287 women were included in the final analysis: 157 (55%) NZ European, 130 (45%) Pacific women. The median age of the study population was 28 (22,35) years, with a median BMI of 29 (23, 34) kg/m^2 and BF% of 36 (28, 44) %. The majority of participants were classified as IT (n = 155, 54%) followed by ET (n = 97, 34%) and MT (n = 35, 12%). Among Pacific women, the lowest proportion were MT (5.4%), while the smallest proportion of NZ Europeans were ET. Due to the small sample for MT as well as the statistically significant differences between ET and MT or ET and IT, the IT and MT were combined for analysis. In comparison with the MT-IT, the ET were respectively younger [23 (21, 26) vs. 30 (24, 37) years, P <0.01], the ETs were predominantly from Pacific ethnicity (86 vs. 14% NZ European, P <0.01) and from higher deprived areas [8 (6, 10) vs. 5 (3, 7) NZDep score, P <0.01]. The ET vs MT-IT had a higher BMI [31 (25, 37) vs 26 (23, 33) kg/m^2, P <0.01] and higher BF% (36 ± 7 %) vs (34 ± 7 %), P = 0.02]. However, the mean BF% was higher in ET (36 ± 7 %) vs MT (32 ± 9 %, P = 0.02). A higher median leptin concentration was seen in the ET [15678 (7683, 23196) pg/mL] vs the MT-IT [10304 (4895, 19708) pg/mL]. On the other hand, a higher ghrelin concentration was seen in the MT-IT [48.9 (24.5, 83.6) pg/mL] vs the ET [28.1 (17.3, 58.7) pg/mL]. With regards to dietary intake, there was no difference in daily mean protein (82.4 g vs 86.3 g), fat (89.7 vs 92.9 g) and carbohydrate (223 vs 193 g) intakes between ET and MT-IT (P >0.05). Although, ET had a 213 kJ higher daily mean energy intake vs MT-IT (8745 vs 8532 kJ, respectively, P = 0.03) (**Table 7.1**).

Table 7.1 *- Participant characteristics*

Variable	MT (n=35)	IT n =155)	ET (n=97)	*P* - value [†]	MT-IT (n=190) [†]	*P* – value [ƶ]
Demographics						
Age (years)	35 (29, 38)	29 (24, 37)	23 (21, 26) [a, b]	**<0.01**	30 (24, 37)	**<0.01**
Ethnicity, n (%)						
NZ European (n=157)	28 (80)	115 (65)	14 (14)	**<0.01**	143 (75)	**<0.01**
Pacific (n=130)	7 (20)	40 (35)	83 (86)		47 (25)	
Deprivation index	5 (2, 6)	5 (3, 7)	8 (6, 10) [a, b]	**<0.01**	5 (3, 7)	**<0.01**
Body Composition						
BMI (kg/m^2)	24 (22, 31)	27 (23, 33)	31 (25, 37) [a, b]	**<0.01**	26 (23, 33)	**<0.01**
BMI groups, n (%)						
<25 kg/m^2	19 (54)	66 (43)	26 (27)	**0.01**	85 (45)	**<0.01**
≥25 kg/m^2	16 (46)	89 (57)	71 (73)		105 (55)	
BF%	32 ± 9	34 ± 7	36 ± 7 [a]	**0.02** [Ɣ]	34 ± 7	**0.02** [r]
BF% groups, n (%)						
< 35%	23 (66)	69 (45)	45 (46)		92 (48)	0.75
≥ 35%	12 (34)	86 (55)	52 (54)	0.07	98 (52)	
Hunger and satiety hormones						
Ghrelin (pg/mL)	74.8 (45.4, 120.8)	46.3 (22.1, 74.2)	28.1 (17.3, 58.7) [a, b]	**<0.01**	48.9 (24.5, 83.6)	**<0.01**
Leptin (pg/mL)	7210 (2716, 14683)	11291 (5726, 20459)	15678 (7683, 23196) [a]	**<0.01**	10304 (4895, 19708)	**0.01**
Dietary intake						
Energy intake (kJ)	8552.6 ± 1701.7	8527.4 ± 2056.3	8745 ± 2576.5	0.74 [Ɣ]	8532 ± 1991.6	**0.03** [r]
Protein intake (g)	85.6 ± 20.3	86.5 ± 21.1	82.4 ± 24.4	0.36 [Ɣ]	86.3 ± 20.9	0.17 [r]
Fat intake (g)	89.8 ± 26.7	93.6 ± 30.6	89.7 ± 28.5	0.55 [Ɣ]	92.9 ± 29.9	0.31 [r]
Carbohydrate intake (g)	196.9 ± 50.5	191.7 ± 63.9	223.1 ± 76.5 [b]	**<0.01** [Ɣ]	192.7 ± 61.6	0.05 [r]

(NZ) New Zealand; (BMI) Body Mass Index; (BF%) Body Fat Percentage; (MT) Morning type; (IT) Intermediate type; (ET)Evening type; (MT-IT) Morning- and intermediate types combined group, Values are reported as Median (25th ,75th percentiles), or means ±SD,
[†]Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data [a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, [ƶ]Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data; Chi-square test for categorical data; [Ɣ]One-way ANOVA for comparing three groups continuous, normally distributed data with Tukey HSD as post hoc test; [r]Independent T-test for comparing two continuous, normally distributed data.

Missing deprivation index (n = 3)

Table 7.2 presents the scores of the three main TFEQ categories and subcategories across chronotype groups. In comparison with the ET, the MT-IT group scored a higher *restraint* score and subsequently higher *rigid contro*l score (P = 0.03 and P <0.01, respectively). The ET had higher *hunger* and *internal locus for hunger* scores (P = 0.02 and P = 0.01, respectively), but lower *situational* (P = 0.02) and *emotional disinhibition* scores (P <0.01) in comparison with the MT-IT.

***Table 7.2** - Eating behaviours (TFEQ) across chronotypes*

Variable	MT (n = 35)	IT (n = 155)	ET (n = 97)	*P* - value †	MT-IT (n=190)	*P* - value[z]
Restraint	7 (4, 11)	8 (6, 12)	6 (4, 10) [a]	**0.04**	8 (5,11)	**0.03**
Flexible control	2 (1, 3)	3 (2, 4)	2 (1, 4)	0.13	3 (2, 4)	0.12
Rigid control	2 (1, 4)	3 (2, 4)	1 (0, 3) [a]	**<0.01**	3 (1, 4)	**<0.01**
Hunger	7 (3, 9)	5 (3, 8)	7 (4, 9)	0.06	5 (3, 9)	**0.02**
Internal locus	3 (1, 4)	2 (1, 4)	3 (1, 4) [b]	**0.01**	2 (1, 4)	**0.01**
External locus	2 (1, 4)	2 (1, 4)	2 (1, 4)	0.64	2 (1, 4)	0.41
Disinhibition	7 (5, 10)	8 (5, 11)	7 (5, 10)	0.12	8 (5, 11)	0.12
Situational	3 (2, 4)	3 (2, 4)	3 (1, 4) [a]	**0.04**	3 (2, 4)	**0.02**
Habitual	1 (0, 3)	1 (0, 3)	2 (1, 3)	0.09	1 (0, 3)	0.14
Emotional	1 (0, 2)	2 (0, 3)	1 (0, 2) [b]	**0.01**	2 (0, 3)	**<0.01**

(MT) Morning type; (IT) Intermediate type; (ET)Evening type; (MT-IT) Morning- and intermediate types combined group,
Restraint items ranged from 0 - 21, hunger items ranged from 0 - 14 and disinhibition items ranged from 0 - 16, Values are reported as Median (25^{th} ,75^{th} percentiles)
†Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data ,[a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167; [z]Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data;

Table 7.3 presents the total EAT-26 score as well as the subcategories across chronotype groups. Compared to the MT, ET scored a higher total EAT-26 score (24.6 [20.0, 30.4] vs 18.9 [14.9, 24.6], *P* = 0.04), and subsequently scored higher on the *bulimia* & *food preoccupation* (1 (0, 3) vs 0 (0, 1), *P* = 0.01) as well as the *oral control* subcategories (2 (0, 5) vs 1 (0, 2), *P* <0.01). Similarly, when collapsing the MT-IT, they scored lower on the *bulimia* & *food preoccupation* (0 (0, 2) vs 1 (0, 33), *P* = 0.01) as well as the *oral control* subcategories (1 (0, 2) vs 2 (0, 5), *P* <0.01) in comparison with the ET.

***Table 7.3** - Eating attitudes (EAT-26) across chronotypes*

Variable	MT (n = 35)	IT (n = 155)	ET (n = 97)	*P* - value [†]	MT-IT (n=190) [†]	*P* - value[ƶ]
Total EAT-26 Score	18.9 [14.6, 24.6]	26.3 [21.5, 32.2]	24.6 [20.0, 30.4][a]	**0.04**[Ɣ]	25.6 [21.2, 30.8]	0.05[r]
EAT-26 Score ≥ 20 n(%)	6 (17)	58 (37)	41 (42)	**0.03**	64	0.10
EAT-26 Score < 20 n(%)	29 (83)	97 (63)	56 (58)		126	
Dieting	3 (2, 6)	5 (2, 11)	5 (2, 9)	0.30	5 (2, 10)	0.99
Bulimia & Food Preoccupation	0 (0, 1)	0 (0, 2)	1 (0, 3) [a, b]	**0.01**	0 (0, 2)	**0.01**
Oral Control	1 (0, 2)	1 (0, 2)	2 (0, 5) [a, b]	**<0.01**	1 (0, 2)	**<0.01**

(EAT) Eating Attitudes Test 26,
Total EAT-26 score items ranged from 0 -26, EAT-26 ≥ 20 indicates disturbed eating attitudes, dieting items ranged from 0 – 13, bulimia & food preoccupation items ranged from 0 – 6, oral control ranged from 0 – 7, Values reported as Median (25th – 75th percentiles), or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale
P-value of < 0.05 was accepted as statistically significant,
[†]Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data [a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, [ƶ]Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data; Chi-square test for categorical data; [Ɣ]One-way ANOVA for comparing three groups continuous, normally distributed data with Tukey HSD as post hoc test; [r]Independent T-test for comparing two continuous, normally distributed data.

7.3.2 Association between chronotype, eating behaviour & eating attitudes within body composition groups

To investigate the association between chronotype and the main and subcategories for both eating behaviour and eating attitudes, the analysis was stratified by BMI and then BF% groups, and adjusted for age, ethnicity, and deprivation index (**Table 7.4**) (models unadjusted for deprivation index are presented in **Appendix E.1**).

In the high BMI group, ET predicted lower *restraint* (β = -1.86, *P* = 0.03), *flexible restraint* (β = -0.84, *P* = 0.01) and *rigid control* (β = -0. 75, *P* = 0.04). However, ET predicted higher perceived *hunger* (β = 1.64, *P* = 0.01), *internal locus for hunger* (β = 0.97, *P* = 0.01), as well as higher *habitual disinhibition* (β = 0.66, *P* = 0.02) among the eating behaviour scores, and higher *bulimia & food preoccupation* (β = 1.61, *P* <0.01) among eating attitude scores. In the normal BMI group, ET predicted overall lower eating behaviour scores for *hunger* (β = -2.10, *P* = 0.01), *external locus* (β = -1.05, *P* = 0.01), *internal locus for hunger* (β = -1.14, *P* = 0.02), *disinhibition* (β = -1.79, *P* = 0.04) and *situational disinhibition* (β = -1.12, *P* <0.01). Before adjustment for deprivation index, ET in the high BMI group also predicted higher *oral control* scores (β = 0.90, *P* = 0.01) (Appendix E.1).

Participants classified as ET in the high BF% group predicted higher *internal locus for hunger* (β = 0.82, = 0.03), and higher *bulimia & food preoccupation* (β = 1.75, *P* <0.01). Among participants with a normal BF%, ET predicted higher *oral control* (β = 1.64, *P* = 0.01), and lower *situational disinhibition* (β = -1.10, *P* <0.01).

7.3.3 Association between dietary intake & chronotype within body composition groups

Among participants with a normal BMI, ET predicted lower daily energy (β = -1209, *P* = 0.01), protein (β = -17.6, *P* <0.01), and fat (β = -14.6, *P* = 0.02) intake. However, ET in the high BMI group predicted higher carbohydrate intakes (β = 199, *P* <0.01). The ET in the normal BF% group predicted a lower daily protein intake (β = -12.9, *P* = 0.02) (**Table 7.4**).

Table 7.4 *- The association between chronotypes (ET vs MT-IT) & eating behaviours, eating attitudes & dietary intake stratified by BMI groups & BF% groups*

Eating behaviours & attitudes	**Normal BMI (<25 kg/m^2, n = 111)**				**High BMI (≥25 kg/m^2, n = 176)**				**Normal BF% (<35%)**				**High BF% (≥35%)**			
	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*
Restraint	1.2	-0.29	-2.67, 2.09	0.81	**0.84**	**-1.86**	**-3.52, -0.19**	**0.03**	-0.14	-1.36	-3.63, 0.91	0.24	-0.11	-0.96	-2.67, 0.76	0.27
	Adjusted R^2 = 0.04, *P* = 0.07				Adjusted R^2 = 0.02, *P* = 0.17				Adjusted R^2 = 0.01, *P* = 0.25				Adjusted R^2 = 0.03, *P* = 0.35			
Flexible Control	0.46	-0.35	-1.27, 0.57	0.45	0. 30	**-0.84**	**-1.43, -0.25**	**0.01**	0.42	-0.67	-1.50, 0.17	0.12	0.33	-0.50	-1.15, 1.15	0.13
	Adjusted R^2 = 0.02, *P* = 0.21				Adjusted R^2 = 0.04, *P* = 0.03				Adjusted R^2 = 0.02, *P* = 0.20				Adjusted R^2 = 0.02, *P* = 0.12			
Rigid Control	0.45	-0.24	-1.13, 0.65	0.59	0.36	**-0.75**	**-1.47, -0.04**	**0.04**	0.47	-0.56	-1.48, 0.37	0.24	0.35	-0.48	-1.17, 0.21	0.17
	Adjusted R^2 = 0.09, *P* = 0.01				Adjusted R^2 = 0.02, *P* = 0.17				Adjusted R^2 = 0.02, *P* = 0.17				Adjusted R^2 = 0.02, *P* = 0.17			
Hunger	**0.80**	**-2.10**	**-3.21, -0.22**	**0.01**	**0.65**	**1.64**	**0.35, 2.92**	**0.01**	0.78	-0.79	-2.34, 0.76	0.31	0.68	1.30	-0.05, 2.65	0.06
	Adjusted R^2 = 0.06, *P* = 0.04				Adjusted R^2 = 0.04, *P* = 0.04				Adjusted R^2 = 0.05, *P* = 0.03				Adjusted R^2 = 0.005, *P* = 0.32			
Internal locus	**0.46**	**-1.14**	**-2.05, -0.23**	**0.02**	**0.35**	**0.97**	**0.27, 1.67**	**0.01**	0.43	-0.40	-1.25, 0.46	0.36	**0.38**	**0.82**	**0.07, 1.57**	**0.03**
	Adjusted R^2 =0.04, *P* = 0.08				Adjusted R^2 = 0.06, *P* = 0.01				Adjusted R^2 = -0.002, *P* = 0.44				Adjusted R^2 = 0.05, *P* = 0.03			
External locus	**0.38**	**-1.05**	**-1.81, -0.30**	**0.01**	0.32	0.51	-0.13, 1.14	0.12	0.38	-0.62	-1.36, 0.13	0.11	0.12	0.40	-0.25, 1.06	0.23
	Adjusted R^2 = 0.05, *P* = 0.05				Adjusted R^2 = 0.04, *P* = 0.03				Adjusted R^2 = 0.01, *P* = 0.27				Adjusted R^2 = 0.03, *P* = 0.07			
Disinhibition	**0.87**	**-1.79**	**-3.51, 0.07**	**0.04**	0.64	1.00	-0.28, 2.27	0.12	0.86	-1.03	-2.72, 0.67	0.23	0.69	0.97	-0.40, 2.34	0.17
	Adjusted R^2 = 0.06, *P* = 0.03				Adjusted R^2 = 0.17, *P* < 0.01				Adjusted R^2 = -0.01, *P* = 0.57				Adjusted R^2 = 0.14, *P* <0.01$^{\delta}$			
Situational	**0.37**	**-1.12**	**-1.92, -0.46**	**<0.01**	0.26	0.20	-0.30, 0.71	0.43	**-0.35**	**-1.10**	**-1.81, -0.39**	**<0.01**	0.28	0.41	-0.13, 0.96	0.14
	Adjusted R^2 = 0.09, *P* = 0.01				Adjusted R^2 = 0.10, *P* < 0.01				Adjusted R^2 = 0.06, *P* = 0.02				Adjusted R^2 = 0.06, *P* = 0.01			
Habitual	0.36	-0.08	-0.78, 0.63	0.83	**0.29**	**0.66**	**0.09, 1.23**	**0.02**	0.34	0.24	-0.44, 0.92	0.48	0.17	0.55	-0.07, 1.17	0.82
	Adjusted R^2 = 0.02, *P* = 0.24				Adjusted R^2 = 0.07, *P* <0.01				Adjusted R^2 = 0.003, *P* = 0.37				Adjusted R^2 = 0.06, *P* = 0.15			
Emotional	0.30	-0.47	-1.07, 0.12	0.12	0.21	0.03	-0.39, 0.47	0.90	0.29	-0.16	-0.70, 0.45	0.67	-0.04	-0.11	-0.56, 0.33	0.62
	Adjusted R^2 = 0.05, *P* = 0.06				Adjusted R^2 = 0.19, *P* <0.01				Adjusted R^2 = -0.01, *P* = 0.70				Adjusted R^2 = 0.20, *P* <0.01$^{\delta}$			
Total EAT-26	3.50	-3.50	-10.4, 3.50	0.32	3.06	5.04	-1.01, 11.1	0.10	3.51	1.41	-5.54, 8.35	0.69	3.34	3.77	-2.82, 10.4	0.26

Eating behaviours & attitudes	Normal BMI (<25 kg/m², n = 111)				High BMI (≥25 kg/m², n = 176)				Normal BF% (<35%)				High BF% (≥35%)			
	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*
	Adjusted R^2 = 0.001, *P* = 0.40				Adjusted R^2 = 0.03, *P* =0.05				Adjusted R^2 = -0.01, *P* = 0.70				Adjusted R^2 = -0.01, *P* = 0.70			
Dieting	1.40	-2.03	-4.82, 0.75	0.15	1.19	0.68	-1.67, 3.04	0.57	1.43	-0.18	-3.01, 2.66	0.90	1.22	0.15	-2.26, 2.57	0.90
	Adjusted R^2 = -0.01, *P* = 0.60				Adjusted R^2 = -0.001, *P* = 0.44				Adjusted R^2 = -0.02, *P* = 0.80				Adjusted R^2 = -0.01, *P* = 0.51			
Bulimia & Food Preoccupation	0.53	-0.26	-1.32, 0.80	0.63	**0.54**	**1.61**	**0.56, 2.68**	**<0.01**	0.51	0.06	-0.94, 1.06	0.91	**0.29**	**1.75**	**0.60, 2.91**	<0.01
	Adjusted R^2 = -0.01, P = 0.56				Adjusted R^2 = 0.09, P < 0.01				Adjusted R^2 = 0.02, *P* = 0.19				Adjusted R^2 = 0.08, *P* <0.01[δ]			
Oral Control	0.63	1.09	-0.15, 2.33	0.08	0.44	0.75	-0.12, 1.762	0.09	**0.29**	**1.64**	**0.47, 2.81**	**0.01**	-0.01	-0.03	-0.96, 0.89	0.95
	Adjusted R^2 = 0.29, *P* <0.01				Adjusted R^2 = 0.12, *P* <0.01				Adjusted R^2 = 0.22, *P* <0.01				Adjusted R^2 = 0.10, *P* <0.01[δ]			
Dietary intake																
Energy intake (kJ)	**456**	**-1209**	**-2112, -305**	**0.01**	483	390	-563, 1344	0.42	535	-789	-1849, 270	0.14	473	293	-641, 1229	0.54
	Adjusted R^2 = 0.06, *P* =0.04				Adjusted R^2 = -0.01, *P* =0.74				Adjusted R^2 = 0.02, *P* =0.36				Adjusted R^2 = -0.01, *P* =0.66			
Protein intake (g)	**5.59**	**-17.6**	**-28.7, -6.56**	**<0.01**	4.50	2.89	-5.98, 11.8	0.52	**5.59**	**-12.9**	**-24.0, -1.87**	**0.02**	4.59	2.44	-6.63, 11.5	0.60
	Adjusted R^2 = 0.07, *P* =0.03				Adjusted R^2 = -0.003, *P* =0.49				Adjusted R^2 = 0.29, *P* =0.15				Adjusted R^2 = -0.29, *P* =0.58			
Fat intake (g)	**6.24**	**-14.6**	**-26.9, -2.19**	**0.02**	6.34	2.07	-10.4, 14.6	0.74	6.84	-11.6	-25.2, 1.89	0.09	6.43	2.12	-10.6, 14.8	0.74
	Adjusted R^2 = 0.10, *P* =0.01				Adjusted R^2 = -0.02, *P* =0.90				Adjusted R^2 = 0.02, *P* =0.18				Adjusted R^2 = -0.01, *P* =0.72			
Carbohydrate intake (g)	14.0	-17.1	-44.7, 10.6	0.23	**14.2**	**199**	**-15.93**	**<0.01**	16.1	-7.38	-39.3, 24.6	0.65	13.8	9.30	-17.9, 36.5	0.50
	Adjusted R^2 = 0.29, *P* =0.02				Adjusted R^2 = 0.29, *P* <0.01				Adjusted R^2 = 0.10, *P* =0.002				Adjusted R^2 = 0.06, *P* =0.01			

(NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (EAT) Eating Attitudes Test – 26
Multiple linear regression models (Enter method) repeated within normal (<25 kg/m²) and high BMI (≥25 kg/m²) groups and then within normal (<35 %) and high (≥35 %) BF% groups, Dependant variable: Eating Behaviour/Attitude, Independent variable: Chronotype groups (MT-IT and ET), adjusted for age, deprivation index & ethnicity, Missing TFEQ data (n = 2), Missing deprivation index (n = 3), P-value of < 0.05 was accepted as statistically significant
[δ] Ethnicity significant in model

7.3.4 Association between eating behaviour combination categories & chronotype within body composition groups

The association between chronotype and the four combination categories for levels of *restraint* and *disinhibition* ranged from *high restraint – low disinhibition* (the ideal eating behaviour combination) to *low restraint - high disinhibition* (least ideal eating behaviour combination) stratified by BMI and BF% groups is presented in **Figure 7.3-6** and **Appendix E.2**. The odds ratios increased for ET in the high BMI group of having *low restraint - high disinhibition* (OR = 3.93, 95% CI, [1.20, 12.8], **Figure 7.3**). When stratifying for BF% groups, the odds ratios decreased for ET in the high BF% group of having *low restraint – high disinhibition* (OR = 0.28 [0.80, 0.96], **Figure 7.5**).

The four combination categories for levels of hunger and disinhibition ranged from *low hunger – low disinhibition* (the ideal eating behaviour combination) to *high hunger - high disinhibition*. The odds ratio of having *high hunger - high disinhibition* (OR = 0.16, 95% CI [0.03, 0.88]) decreased for normal BMI participants with an ET vs. MT-IT (reference group, **Figure 7.4**). In those models which were not adjusted for deprivation index score, the odds ratio of having *high hunger – high disinhibition* increased for ET in the high BMI group (OR= 2.78 [1.12, 6.90], **Appendix E.3**).

Figure 7.3

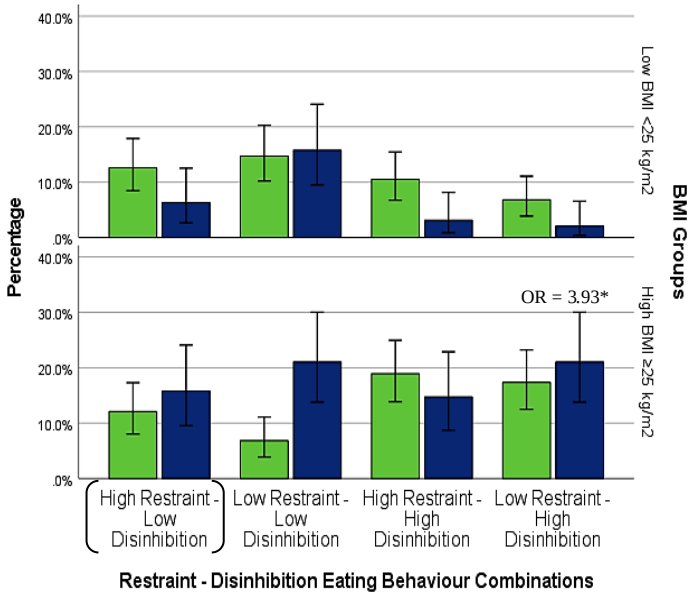

Figure 7.4

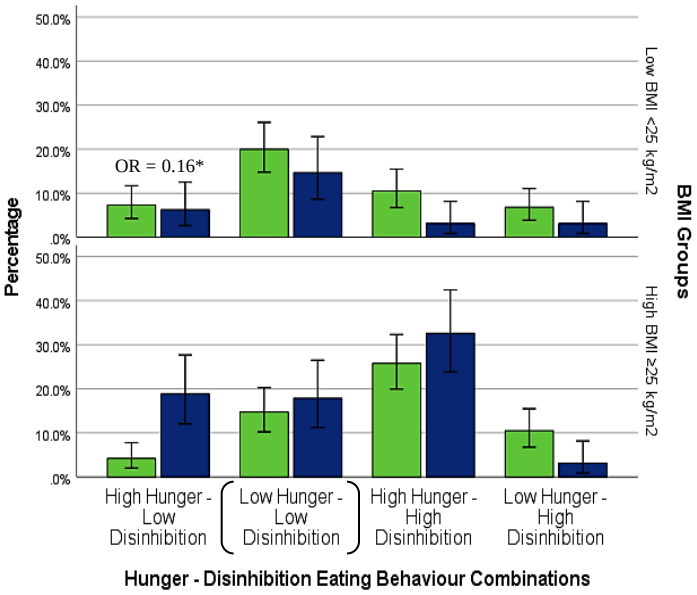

Figure 7.5

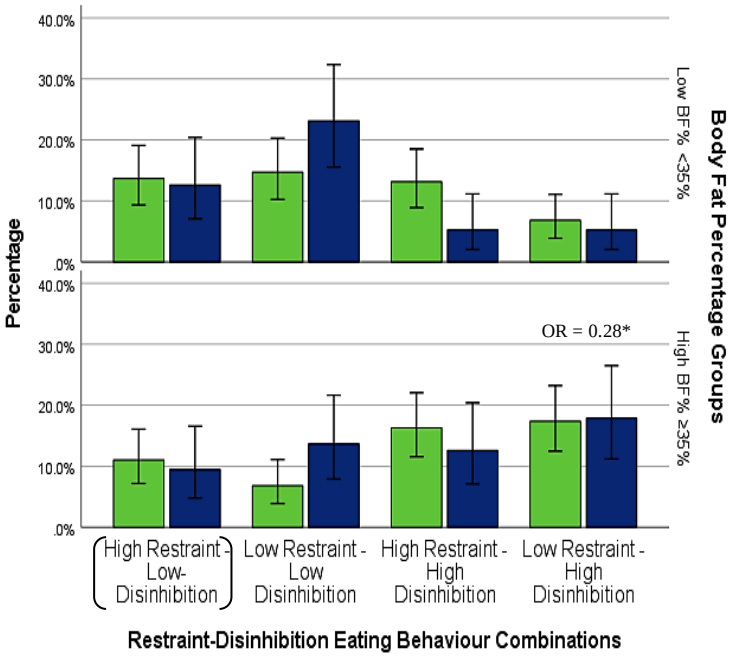

Figure 7.6

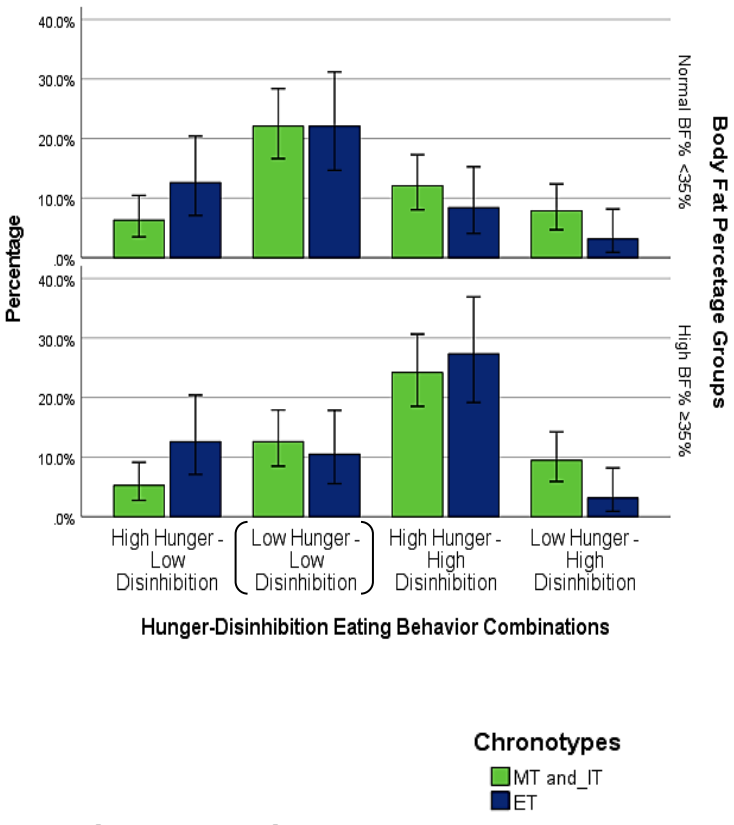

Figure 7.3 - 7.6 *- Eating behaviour combination categories according to BMI and BF% groups*

**P value <0.05 determined by multinominal regression*
[ ] denotes the ideal eating behaviour combination
Missing deprivation index (n = 3), Missing TFEQ (n = 2)

7.3.5 Correlation between chronotype, hunger and satiety hormones, TFEQ & EAT-26

In correlation analysis (**Table 7.5**), MCTQ (i.e., higher MCTQ indicates later midsleep and goes towards eveningness and vice versa for morningness) was positively correlated with higher BMI, BF%, the higher concentrations of the satiety hormone, leptin, *total EAT-26* score, as well as the subcategories of *bulimia & food preoccupation*, and *oral control* scores, while being negatively correlated with ghrelin, *rigid control* and *emotional disinhibition* ($P < 0.05$). Ghrelin was negatively correlated with *hunger*, both the *hunger* subcategories of *internal* and *external locus for hunger*, *disinhibition, habitual disinhibition, total EAT-26* and *bulimia & food preoccupation* scores, while being positively correlated with *restraint* score (and *rigid control* ($P < 0.05$). Leptin was positively correlated with *hunger*, both subcategories of *internal* and *external locus for hunger*, *disinhibition*, two of its subcategories *habitual* and *emotional disinhibition*, *total EAT-26* score and two of the subcategories, *dieting and bulimia & food preoccupation*, while being negatively correlated with *flexible control* score ($P < 0.05$).

Table 7.5 *- Correlations with MCTQ, hunger & satiety hormones, TFEQ & EAT-26*

Eating behaviours & attitudes	MCTQ	Ghrelin (pg/mL)	Leptin (pg/mL)
MCTQ	-	-0.29***	0.16**
BMI	0.20***	-0.36***	0.70***
BF%	0.14*	-0.32***	0.75***
Restraint	-0.11	0.12*	-0.11
Flexible Control	-0.06	0.09	-0.16**
Rigid Control	-0.19**	0.15*	-0.08
Hunger	0.11	-0.20***	0.17**
Internal locus for Hunger	0.11	-0.16**	0.12*
External locus for Hunger	0.03	-0.17**	0.17**
Disinhibition	-0.05	-0.16**	0.23***
Situational Disinhibition	-1.00	-0.10	0.11
Habitual Disinhibition	0.09	-0.17**	0.24***
Emotional Disinhibition	-0.13*	-0.12	0.15**
Total EAT-26	0.15*	-0.12*	0.17**
Dieting	0.06	-0.09	0.16**
Bulimia & Food Preoccupation	0.14**	-0.13*	0.19***
Oral Control	0.34***	-0.06	-0.07

(MCTQ) Munich Chronotype Questionnaire (BMI) Body Mass Index, (BF%) Body Fat Percentage, (EAT) Eating Attitudes Test – 26,
Restraint items ranged from 0 -21, hunger items ranged from 0 – 14 and disinhibition items ranged from 0 -16, Total EAT-26 score items ranged from 0 -26, EAT-26 ≥ 20 indicates disturbed EA, dieting items ranged from 0 – 13, bulimia & food preoccupation items ranged from 0 – 6, oral control ranged from 0 – 7,
**P < 0.05, **P < 0.01, ***P ≤ 0.001*

7.4 Discussion

This study showed a significant relationship between circadian preference, hunger and satiety hormones, eating behaviour, eating attitudes, and body composition, thereby painting an obesity promoting picture for ET. We found that ET vs MT and IT were associated with a higher BMI and BF%, which may be explained by their unhealthy eating behaviour (higher *hunger* and *disinhibition* with lower *restraint* scores), and eating attitudes (higher *bulimia & food preoccupation*, and *oral control* scores), as well as lower ghrelin and higher leptin concentrations seen in this study. The combined MT-IT on the other hand, displayed more favourable eating behaviours (higher *restraint* and lower *hunger*) and less disturbed eating attitudes (lower *bulimia & food preoccupation* and lower *oral control* scores), all of which is reflected by their lower BMI and BF%.

Eveningness was a significant predictor of all three of the TFEQ's main categories namely *hunger* (high) and *restraint* (low) in the high BMI group, and *disinhibition* (low) in the normal BMI group. In other words, ET are more susceptible to feelings of *hunger* and are more prone to relax dietary restrictions, all of which leads to overconsumption of foods and a high BMI. However, ET that have more control over food intake have a healthy BMI. Moreover, ET in the high BMI group were 2.67 times more likely to score high in the *hunger* category (≥5) (**Appendix E.4**). It would also seem that higher perceived feelings of *hunger* in the ET were internally interpreted, as evident by the positive association with the sub-category of *internal locus for hunger* among the high BMI ET participants. Conversely, ET in the normal BMI groups showed lower *external locus for hunger* thus they were less affected by environmental cues such as buffets or driving past a fast-food drive through. These findings are in line with both chronotype and non-chronotype studies, showing a significant relationship between ET and high *hunger* (Schubert & Randler 2008) and high body composition (French et al. 2014) respectively. The higher *hunger* displayed by ET in both body composition categories could be explained by the two to three hour phase delay in their day, resulting in mealtimes shifting across the day seen in ET vs MT (e.g., ET may not feel hungry early in the morning and skip breakfast, but feel more hungry later). Based on their intrinsic preferences, ETs have been shown to have later clock times for breakfast (Sato-Mito et al. 2011, Baron et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019), or may even skip breakfast (Sato-Mito et al. 2011, Silva et al. 2016, Teixeira et al. 2018). Individuals who omit breakfast may be hungrier later in the day (Stanton Jr 1989), which was shown in part by Meule and colleagues (2012), who reported that ET skipped breakfast more often than MT, although there was no difference in perceived *hunger* between chronotypes (Meule et al. 2012), which is in contrast to this study that found higher hunger scores in ET.

Restraint, which is the conscious restriction of food intake, is used as a strategy by dieters to control body weight (Stunkard & Messick 1985). Evidence from other studies that have not investigated

chronotypes, showed that higher *flexible control* is linked with a lower body composition and greater weight loss success (McGuire et al. 2001, Provencher et al. 2003), while higher *rigid control* is linked to a higher body weight due to the associated lack of control (Mills et al. 2018). This study found that ET in the high BMI group was 0.37 times less likely of scoring in the high *restraint* group (≥7) and ET in the high BMI group was associated with lower *restraint* as well as lower *flexible* and *rigid control*, in comparison with MT-IT (Appendix E.4), suggesting that ET have less successful weight loss attempts. Conversely, studies have found similar results with MT having higher *restraint* (Schubert & Randler 2008), although in their study the subcategories of *restraint* were not explored.

Lower total *disinhibition* was predicted by ET in the normal BMI group, which is in accordance with findings from Schubert et al. (2008) that found lower *disinhibition* scores among the ET. Furthermore, lower *situational disinhibition* was predicted by ET in both the normal BMI and BF% groups, demonstrating that ET with a normal body composition were less likely to overeat in response to environmental cues such as a social occasion. Among the high BMI individuals, *habitual disinhibition* (that is the susceptibility to overeat in reaction to everyday circumstances) was predicted by ET. Other studies have found similar results with *habitual disinhibition* being correlated with weight gain (Hays & Roberts 2008), likely contributing to overconsumption of food on a habitual basis. Other studies that have not investigated chronotypes, have shown that high *disinhibition* is a significant predictor of high BMI (French et al. 2014, Kruger et al. 2016, Blumfield et al. 2018) and a NZ based study among women have shown this for both BF% and BMI (Kruger et al. 2016). It has been suggested by the creators of the TFEQ, Stunkard and Messick, that individuals with high *disinhibition* might benefit from emotional behavioural management strategies, for example management of loneliness and anxiety (1985). However, in this study, although IT, reported a higher median score of 2 (0, 3) vs ET (1 (0, 2)), no associations were found between the subcategory of *emotional disinhibition* and the different chronotypes, which is in contrast with previous research (Vera et al. 2018). On the contrary, Hays et. al. (2008) has demonstrated that weight change over time was largely explained by *habitual disinhibition* (Partial r = 0.31) followed by *emotional disinhibition* (Partial r = 0.22) and may therefore be a more important correlating factor of weight gain, than *emotional disinhibition* (Hays & Roberts 2008).

From the 68 participants that had the ideal eating behaviour combination *high restraint- low disinhibition*, a lower percentage of 31% were ET in comparison with 69% that were MT-IT (data not shown). In the *restraint-disinhibition* combination categories, ET in the high BMI group were 3.93 times more likely of having *low restraint - high disinhibition* – the less ideal behaviour combination. A lower percentage of 32% of ET had the ideal eating behaviour combination *low hunger - low disinhibition* in comparison with 68% of MT-IT from the 97 participants that reported this eating behaviour combination (data not shown). Furthermore, in the normal BMI group ET were 0.16 times

less likely of having the eating behaviour combination of *high hunger -low disinhibition* when compared with MT and IT. These findings suggest that there is a relationship between the three main TFEQ traits and that combining these traits creates a more thorough profile of eating behaviour differences between chronotype as well as differences between body composition groups. As of yet, there is no chronotype study to compare our findings to, although studies focussing on body composition found that a high BMI was associated with the least ideal eating behaviour combinations of *high disinhibition - low restraint* (Dykes et al. 2004) and *high disinhibition - high hunger* (Provencher et al. 2003), similar to findings of this study.

All three of the TFEQ's main categories as well as some of the subcategories have been shown to be related to disturbed eating behaviour and eating disorders in non-chronotype studies (Keeler et al. 2015, Vinai et al. 2015, Linardon & Mitchell 2017). In this study, chronotype did not predict the EAT-26 score, however a higher number of ET (n = 41) vs MT (n = 6) reported a score of ≥20, which may denote more disturbed eating attitudes seen among ET. Furthermore, a positive correlation was seen between the MCTQ- and total EAT-26 scores, meaning that a score towards eveningness was associated with a higher total EAT-26 score. A previous study by Harb et al. 2012 that explored EAT-26, found no correlation with chronotype (2012). In our study, ET in both the high BMI and high BF% groups predicted higher scores in the EAT-26 subcategory of *bulimia & food preoccupation*. The ET had a higher median score of 1(0,3) for *bulimia & food preoccupation* in comparison with MT-IT 0 (0,2). This is in line with chronotype studies, that demonstrated that ET were associated with higher risk of bulimic behaviour (Kasof 2001) or correlated with a higher Binge Eating Scale score (Harb et al. 2012). It has been suggested that this higher risk of bulimia & behaviours such as binging and purging, may be due to light exposure during the biological night (Kasof 2001) as well as later sleep and wake times (Kasof 2001). Moreover, during the night individuals are more at their leisure, which makes it easier to overeat or relax their weight loss goals (Kasof 2002). During the evening TV watching and alcohol consumption are more prevalent (Sinnett et al. 1983), which creates an environment for mindless eating, binge eating (Seddon & Berry 1996) and *habitual disinhibition*. These factors were not investigated in this study; however, ET are known to have later sleep and wake times as they habitually extend their wakefulness to night hours, which may account for the associations seen.

Interestingly, in this study, a link between the hunger and satiety hormones, body composition, the TFEQ as well as the EAT-26 was found. It is well known that ghrelin concentrations are decreased in obese individuals, while leptin concentrations are elevated (Klok et al. 2007, Morioka et al. 2016), which was also shown in this study; the ET had higher body composition, higher leptin, and lower ghrelin concentrations in comparison with the MT-IT. Negative correlations between ghrelin and *disinhibition* (including *habitual*), *hunger* (both *internal* and *external*) scores were seen, while a positive correlation with *restraint* (including *rigid control*) scores, was seen, which aligns with other studies

(Blundell et al. 2008, Schur et al. 2008, Langlois et al. 2011). It is suggested that individuals with high *restraint* counteract their elevated appetite by increasing their conscious restriction of food intake in a bid to maintain body weight (Schur et al. 2008). For example, individuals with higher ghrelin (appetite) rigidly restrict their food intake and may employ strategies to avoid situations where habitual disinhibition (e.g., eating snacks while TV watching) may occur. This may in part account for the more obesity protecting eating behaviours including higher *restraint, rigid control* and lower *situational* and *emotional disinhibition* scores reported by the MT-IT in comparison with the ET. Studies in obese individuals as well as night shift workers have shown that, the normal suppression of ghrelin after a meal is lessened, and consequently, these individuals still experience hunger, leading to overconsumption of food (Le Roux et al. 2005, Klok et al. 2007), which may partly explain the higher body composition and *hunger* scores seen in ET. Negative correlations between ghrelin, *total EAT-26* and *Bulimia & Food Preoccupation* were seen, suggesting that individuals with lower appetite (ghrelin) are less likely to display disturbed eating attitudes.

On the other hand, leptin was positively correlated with MCTQ score (towards eveningness), BMI and BF%. In obese individuals, resistance to the satiety hormone, leptin, is common. The reduced satiety in obese individuals may result in overconsumption of food and subsequent eating behaviours. In this study leptin was negatively correlated with *flexible control*, while being positively correlated with *hunger* and both *hunger* subcategories, *disinhibition*, *habitual* and *emotional disinhibition*, which further describe the association seen among ET in the high body composition groups and higher *hunger*, *disinhibition*, and lower *restraint* scores. In a forced desynchronisation study (which allowed to disentangle clock hours from sleep-wake times and homeostatic sleep drive), leptin was shown to not follow a 24-hour pattern but rather follow the sleep-wake and feeding-fasting cycle. They found a decrease in leptin concentrations (satiety hormone) (Scheer et al. 2009), which could possibly lead to reduced satiety in these individuals and consequently higher food intake. This suggests that scheduled meal timing is an important synchroniser of leptin concentrations (Scheer et al. 2009). ET are known to delay their meal timing, distributing their dietary intake towards the biological night (van der Merwe et al. 2022), which in conjunction with the higher adiposity may explain the higher leptin concentrations seen in the ET. These findings suggest that together with a higher body composition, the hunger and satiety hormones are linked with eating behaviour that promote excessive / poor quality food intake among ET.

7.5 Strengths & limitations

To our knowledge this is the first study to not only investigate the main TFEQ categories and the total EAT-26 score but also the subcategories of each, creating a more comprehensive behaviour profile of each chronotype. This study also showed the relationship between chronotype, eating behaviour and eating attitudes and body composition, which has not been explored previously. This study was limited

in a couple of ways. Firstly, the number of MT participants were very few and to account for this, the MT were combined with the IT. Secondly the study was of cross-sectional design, which limits the investigation of eating behaviours and eating attitudes over time. Furthermore, investigating the concentrations of the hunger and satiety hormones at different time points e.g., before and after food intake or in the day vs the evening could provide a clear picture of how these hormones affect behaviour.

7.6 Conclusion

Individuals with an ET are prone to display eating behaviour that led to overconsumption of food (including higher *hunger* and *habitual disinhibition* and lower *restraint*) and more disturbed eating attitudes (including *bulimia & food preoccupation*) all of which favours an eating pattern that promotes weight gain and obesity over time. Furthermore, this study showed a link between the hunger and satiety hormones regulation and the eating behaviour and eating attitudes, which suggests that hormonal regulation may mediate behaviours among different chronotypes and body composition behaviours.

Individuals that are prone to weight gain are more vulnerable to problematic eating behaviours and disturbed eating attitudes. This chapter investigated the associations between eating behaviour and eating attitudes among different chronotypes. Furthermore, the association between eating behaviour, eating attitudes, body composition as well as leptin and ghrelin was investigated.

7.7 References

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DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of candidate:	Carlien van der Merwe
Name/title of Primary Supervisor:	Professor Rozanne Kruger
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8 The Chapter 8: Final discussion

8.1 Study synopsis

The rates of overweight and obesity has been rising at alarming rates globally (World Health Organization 2021) and as well as in the overall NZ population, however with glaring differences between the ethnic groups of NZ European and Pacific women (Ministry of Health 2019, Ministry of Health 2020). The aetiology of obesity is complex with a myriad of interplaying factors (biological, environmental and socio-economic) each with a contributing role in obesity development (Blüher 2019). Years of research have been dedicated to understanding how obesity comes to be and why some individuals seem to be more affected or predisposed than others (Schwartz et al. 2017). The diet is considered one of the major contributing factors to obesity, but simply advising overweight and obese individuals to "eat less" and "exercise" more does not always provide long term successful weight reduction (Taubes et al. 2013). Obesity is a disorder of the energy homeostasis system and requires insights beyond the quantity of consumed and expended energy (Heymsfield & Wadden 2017, Blüher 2019). Dietary related factors such as the composition of meals in terms of energy density (Rolls et al. 2006, Ledikwe et al. 2007, Galgani & Ravussin 2008, Rouhani et al. 2016), macronutrients (Galgani & Ravussin 2008, Flatt 2011) and food combinations (Miller et al. 1990, Satia-Abouta et al. 2002, Newby et al. 2003, Ahluwalia et al. 2009, Paradis et al. 2009, Austin et al. 2011, Suliga et al. 2015); timing of intake (Morikawa et al. 2008, Garaulet et al. 2013, Jakubowicz et al. 2013, Mota et al. 2013, Balieiro et al. 2014, Bo et al. 2014, Raynor et al. 2018, Garaulet 2020, Samhat et al. 2020) as well as certain eating behaviours (Provencher et al. 2003), all play a role in obesity development.

This PhD thesis formed part of the PRedictors linking Obesity and the MIcrobiomE (PROMIsE) study. The PROMIsE study sought to explore some of the interplaying factors involved in obesity development. The aim was to compare different gut microbiome profiles in the ethnic group with the highest risk of obesity (Pacific) vs the groups with a lower risk (NZ European) in NZ. Associations with dietary intake, physical activity, taste perception, eating behaviour, and sleep were further explored. From the wider Auckland region, 304 healthy NZ European (n = 157) and Pacific women (n = 130) aged between 18 and 45 years were included in the PROMIsE study (Kindleysides et al. 2019). Pacific women compared to NZ European women are known to have different body composition profiles (Rush et al. 2007) and may therefore have different metabolic disease risk profiles (Rhee 2022). Therefore, the participants were specifically recruited as groups having different body composition profiles within each ethnicity (normal and obese profiles) (Kindleysides et al. 2019). Data collection took place from May 2016 until May 2017 and included body composition measures (BMI, BF%, WC, HC, lean and fat mass distribution), metabolic biomarkers (glucose homeostasis, lipid profiles and endocrine regulators), dietary intake, eating behaviours and sleep data.

Dietary data (energy, macro- and micronutrient intakes) as well as timing of intake were collected by using non-consecutive five-day food records (5DFR). The food records included two weekend- and three weekdays. Preliminary dietary data analysis showed no statistically significant difference in the total daily energy, protein, and fat intakes ($P > 0.05$) between lean and overweight/obese women of both ethnicity groups (Renall 2020). Other researchers have also showed that there are not many differences in the "how much food" is eaten between obese and non-obese individuals, but there are differences in the "when", "what" and the "how" food is eaten (Dykes et al. 2004, de Lauzon-Guillain et al. 2006, Arble et al. 2009, French et al. 2012, Garaulet et al. 2013, Garaulet 2014, Kruger et al. 2016, Ruiz-Lozano et al. 2016). Looking at the PROMIsE study's individual food records it was clear that the population consumed food at varying times during the day and especially the night, with a vast range of food combinations. This led to this enquiry where we decided to look at the likely core of "what makes us tick", namely the circadian biology (Roenneberg et al. 2003, Sato-Mito et al 2011, Bass 2012, Kanerva et al. 2012, Maukonen et al. 2016, Jiang & Turek 2017, Manoogian and Panda 2017, Maukonen et al. 2017, Muñoz et al. 2017, Blüher 2019, Maukonen et al. 2019). Humans are physiologically adapted to consume foods during the light cycle of the earth's daily rotation, as reflected in the metabolic processes that makes us more suitable to gather and store energy during the light cycle, and to fast and rest while using stored energy during the dark cycle (Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Covassin et al. 2016). So, if we are 'programmed' to follow this day-eating and night-fasting cycle, why do some individuals extend their wakeful period into the night and still consume food, while others prefer an early sleep and rise routine? The answer could lie in our societal 24/7 structure, where the availability of the electric light has made it possible to stay awake for longer periods during the night with easy access to food. But the question remains that, apart from forced nightwork schedules (Roenneberg et al. 2012), why do individuals structure their sleep- wake- and mealtimes either to be more evening-orientated or more morning-orientated? This question prompted the exploration of different chronotypes, which is one reason for this behavioural manifestation of an individual's internal clock (Roenneberg et al. 2003). In this group of women, we were able to determine chronotype using the chronotype questionnaire (MCTQ), which was assessed in the PROMIsE study (Roenneberg et al. 2003, Roenneberg et al. 2007).

Therefore, the aim of this PhD thesis was to explore the diet in-depth (eating patterns and eating behaviours) as it relates to different chronotypes and metabolic health markers of NZ European and Pacific women with different body fat profiles and varied metabolic disease risk factors.

Figure 8.1 illustrates the three identified research areas as well as the related variables that was explored in this PhD thesis.

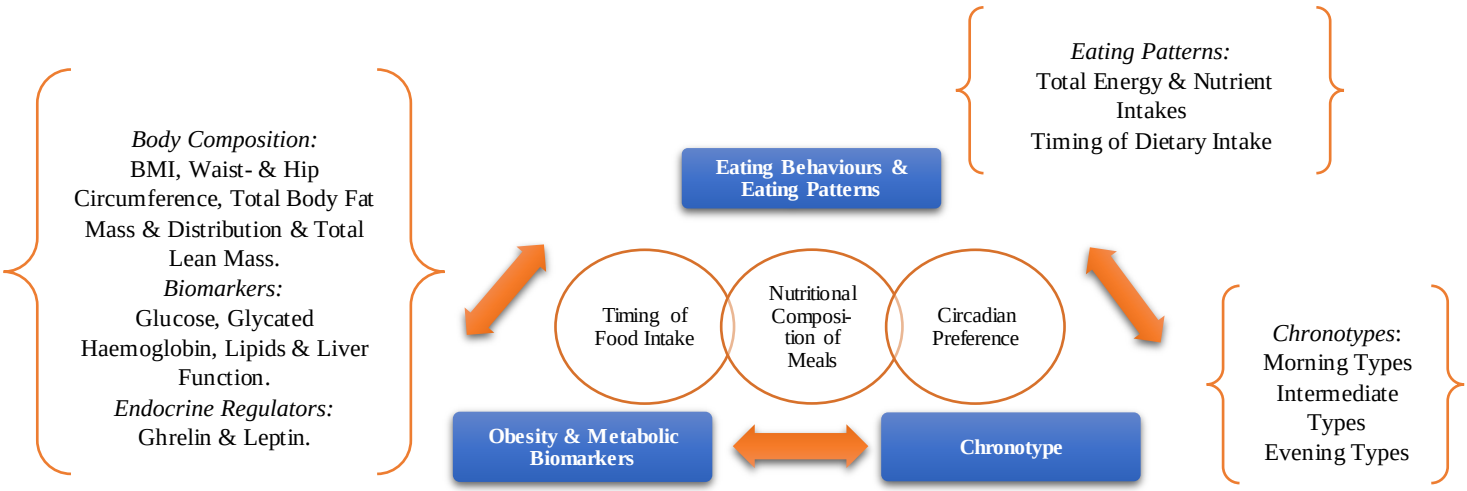


Figure 8.1 *Schematic overview of the key research variables*

8.2 Discussion of results by thesis objectives

Thesis objective 1*: to conduct a systematic review of the literature (i) in order to assess published evidence whether chronotype is associated with body composition, and (ii) if this has an effect on dietary intake, eating behaviours, and metabolic biomarkers in healthy adults (chapter 4).*

The systematic literature search was conducted (according to PRISMA principles) on PubMed, CINAHL, MEDLINE, SCOPUS, and Cochrane library, and identified 4404 articles. Of these, 24 full text articles were included in the systematic review. The systematic review (van der Merwe et al. 2022) was unique from other reviews because we did not include studies that recruited participants with acute, pre-existing and chronic diseases (Almoosawi et al. 2019, Lotti et al. 2021) or night shift workers (Mazri et al. 2020), which may have influenced chronotype. We compiled a comprehensive framework that grouped body composition and metabolic biomarker outcomes, eating behaviours, together with dietary components for each chronotype group in a healthy population of lean and obese women. These findings from the review gave further insight into the higher rates of overweight/obesity and unhealthier metabolic biomarkers among ET.

Based on the findings of the review the objectives were answered as follows:

(i) Chronotype was associated with body composition. More specifically, a later chronotype (ET) was associated with either a higher BMI (Baron et al. 2011, Vera et al. 2018, Maukonen et al. 2019, Xiao et al. 2019, Zeron-Rugerio et al. 2019, Muscogiuri et al. 2020) or a greater increase

in BMI over a period of time (Culnan et al. 2013, Lucassen et al. 2013, Maukonen et al. 2019), as well as a greater increase in weight over time when compared to MT (Maukonen et al. 2019).

(ii) Chronotype was associated with dietary intake. The different chronotypes consumed similar quantities of energy (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Vera et al. 2018, Maukonen et al. 2019, Xiao et al. 2019, Beaulieu et al. 2020, Muñoz et al. 2020, Zerón-Rugerio et al. 2020) and macronutrients (Baron et al. 2011, Sato-Mito et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2016, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Teixeira et al. 2018, Vera et al. 2018, Muñoz et al. 2020), despite having different body composition profiles. Interestingly, distinct lower morning and higher evening energy and macronutrient intakes were found among ET when compared with MT (Baron et al. 2011, Baron et al. 2013, Lucassen et al. 2013, Maukonen et al. 2017, Maukonen et al. 2019, Xiao et al. 2019, Zerón-Rugerio et al. 2020). The format of the dietary intake between chronotypes differed, with earlier types consuming healthier, micronutrient-rich foods such as fruits (Baron et al. 2011, Yoshizaki et al. 2018), vegetables (Baron et al. 2011, Sato-Mito et al. 2011, Yoshizaki et al. 2018), pulses (Sato-Mito et al. 2011), eggs (Sato-Mito et al. 2011), cereals (Maukonen et al. 2016, Vera et al. 2018), fish (Maukonen et al. 2017), and dairy (Sato-Mito et al. 2011). In comparison, ET consumed more energy-dense foods including sugar-sweetened beverages (Baron et al. 2011, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020), confectionery (Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Muscogiuri et al. 2020), fast foods (Baron et al. 2011) and cholesterol containing foods (Mota et al. 2016).

(iii) Specific eating behaviours were linked to chronotype. Those classified as ET, displayed more undesirable eating behaviours, such as later clock times for main meals (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019) and breakfast skipping (Sato-Mito et al. 2011, Silva et al. 2016, Teixeira et al. 2018).

(iv) With regards to eating behaviour scores, the ET reported higher food cravings, less control over food intake (*disinhibition*) and emotional eating. Conversely, MT displayed healthy eating behaviours such as less stress-related eating and more control over foods eaten (*restraint*) (Vera et al. 2018).

(v) Lastly, an unhealthy metabolic biomarker profile was found among ET, with higher lipid profiles and poorer glucose homeostasis when compared with MT (Vera et al. 2018).

The systematic review identified the following research gaps which guided the analysis of the PROMIsE study dataset among women recruited with different body composition profiles and chronotypes.

- The majority of studies included in the systematic review used BMI as measure of body composition whilst fewer investigated other body composition measures. In those studies, the authors found no statistically significant differences between chronotypes. Following the systematic review (van der Merwe et al. 2022) we thus addressed the gaps in the literature by using a total of 18 different body composition markers to investigate differences in lean body and fat mass distribution between chronotypes.
- From the systematic review (van der Merwe et al. 2022) it was evident that the main research focus was on absolute dietary intake differences between chronotypes in terms of total energy and macronutrient intakes, but lacking investigation of micronutrient intakes. Only selected food group intakes were explored rather than using a comprehensive food group approach. Few studies investigated the distributions of energy, macronutrient or food intake throughout the 24-hour day.
    - We addressed these gaps using the data set of the PROMIsE study and identified the chronotype groups, explored the diet in depth, and by investigating differences in the total energy, macro- and total micronutrient intakes between chronotype groups.
    - Furthermore, we explored the distribution of energy intake by first assessing the average energy intake per hour, and then creating four main dietary intake windows (showing the mean energy and macronutrient intakes for each window)
    - In addition to total dietary intakes, we determined both food- and nutrient patterns by creating dietary patterns using Principal Component Analysis (PCA). These dietary patterns gave insight into the typical foods and nutrients consumed together by different chronotypes and body composition groups.
- Only one of the included systematic review studies explored the effect of energy and macronutrient distribution on body composition (only using BMI), between different chronotypes (Xiao et al. 2019). Therefore, to address this gap, in this thesis work we investigated whether different chronotypes predicted the quantity of energy- and macronutrient intakes within low and high AG ratio and BF% groups based on the PROMIsE data set.
- Differences in eating behaviours and eating attitudes among chronotypes have not been explored using validated questionnaires such as the TFEQ (Lázár et al. 2012, Beaulieu et al. 2020), and none of the included studies used the EAT-26. Furthermore, none of the studies of our systematic review investigated the possible relationship between eating behaviours, eating attitudes, and body composition. The studies that have utilised the TFEQ have either investigated only the main categories (Lázár et al. 2012) or the total TFEQ score (Beaulieu et al. 2020). Both the TFEQ and the EAT-26 consist of several subcategories that can be used to create a comprehensive profile of eating behaviours (Stunkard & Messick 1985, Westenhoefer et al. 1999, Lesdéma et al. 2012) and -attitudes among chronotypes (Garner et al. 1982). We addressed these gaps in the literature and investigated the main categories of the TFEQ and the EAT-26 as well as the subcategories in

relation to high and normal BMI and BF% groups, creating a comprehensive eating behaviour and - attitudes profile for each chronotype in our PROMIsE data set.

- Only four of the included studies investigated differences in metabolic biomarker profiles between the different chronotypes. We identified this gap and included a total of 11 metabolic biomarkers, including lipid profiles, glucose homeostasis markers, an inflammatory marker and endocrine regulators for the analysis of the PROMIsE data set.

Thesis objective 2*: to investigate if chronotype is associated with body composition in healthy young women (NZ European and Pacific women) (chapter 5).*

Similar to the results from our systematic review (van der Merwe et al. 2022), we found that ET in the PROMIsE study were associated with a higher BMI, while a MT were associated with a lower BMI. It is well established that BMI as sole body composition measure is limited as it does not reliably reflect body composition, metabolic disease, and fat mass disease risk on an individual level (De Lorenzo et al. 2016). In addition to having a higher BMI, the ET women in the PROMIsE study were also associated with higher WC and HC, android fat- and visceral fat percentage in comparison with those with an earlier chronotype (MT and IT were collapsed together into a mixed MT-IT group for most analyses). The AG ratio and FM:FFM ratio was investigated for the first time among chronotypes. In this group of women, a higher AG ratio was found in ET in comparison with the women with an earlier chronotype (MT-IT). The ratio of AG is rarely examined in the literature (Kang et al. 2011, Fu et al. 2014, Okosun et al. 2015), although it has been associated with an elevated risk of metabolic syndrome in healthy adults (Kang et al. 2011, Fu et al. 2014). It also emerged to be a more sensitive predictor of NCDs such as cardiometabolic dysregulation and risk factors than other measures such as BMI, android fat- and visceral fat mass (Okosun et al. 2015). The ratio of FM:FFM is a useful indicator of the joint effect of FM and FFM (Prado et al. 2012, Xiao et al. 2019). Adults with a higher ratio are more likely to be diagnosed with NCDs such as metabolic syndrome, hypertension prediabetes, diabetes mellitus. Interestingly, the results from the PROMIsE study showed that later types (ET) had a higher lean body and muscle mass when compared to earlier types (MT and IT). However, correlation analysis revealed that a later chronotype (towards ET) was positively correlated with a higher FM: FFM. The higher lean body and muscle mass may have been influenced by the ethnic differences in the study population. Pacific peoples (which were more ET) are known to have a higher FFM (Rush et al. 2007) as well as higher lean mass to fat mass in comparison with NZ Europeans, whilst still having a greater absolute fat mass (Health 1997). These results are important findings and contribute to current reports such that a higher body composition may contribute to metabolic changes and an elevated risk of NCD (Rhee 2022) in ET.

Thesis objective three*: To describe the diet of these women of in terms of eating patterns (nutritional composition and timing of meals) and to investigate whether chronotype and body composition are associated with the eating patterns (chapter 5).*

For this part of the doctoral thesis, particular focus was placed on the distribution of energy and macronutrient intake during the 24-hour day in relation to BF% and AG ratio of the PROMIsE data. We found similar total daily energy and macronutrient intakes for MT and ET, which concurs with the findings from the systematic review (van der Merwe et al. 2022). However, the ET group showed a higher energy intake than the combined MT-IT group.

With regards to micronutrient intakes, the entire study population consumed inadequate quantities of micronutrients (potassium, calcium, iron, iodine, and vitamin E). The ETs had the lowest intakes (folate, vitamin A, -C, -E, magnesium potassium, calcium, iron, and iodine) among all the chronotypes, which is similar to findings from others (Sato-Mito et al 2011, Kanerva et al. 2012). Micronutrients form an integral part of a healthy diet and low intakes such as was seen among the ET, are associated with increased risk of cancer (Palli et al. 2001, Bosetti et al. 2013).

Previous studies have shown that ET delay their meal timing with later clock times for main meals (Baron et al. 2011, Sato-Mito et al. 2011, Lucassen et al. 2013, Silva et al. 2016, Teixeira et al. 2018, Vera et al. 2018, Xiao et al. 2019). Few studies have investigated the distribution of energy (Baron et al. 2011, Maukonen et al. 2017, Maukonen et al. 2019, Zerón-Rugerio et al. 2020) and macronutrient intake throughout the day (Baron et al. 2013, Maukonen et al. 2017, Xiao et al. 2019), showing that chronotypes consumed their meals according to their preferred sleep-wake timing. We found similar results, showing that ET distributed their daily energy and macronutrient intakes towards night-time, consuming 23% of their total energy intake after 20:00 and only 9% in the morning before 10:00. While the MT-IT distributed their intakes towards the early part of the day, consuming 15% of their total energy intake before 10:00 and only 11% at night after 20:00).

The timing of dietary intake is important as there is clear evidence that macronutrient metabolism and energy expenditure vary across the 24-hour light-dark cycle (Roberts 1964, Takahashi et al. 1968, Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Saad et al. 2012, Cipolla-Neto et al. 2014, Covassin et al. 2016, Rácz et al. 2018), having different effects on energy utilisation and ultimately body composition (Garaulet et al. 2013, Jakubowicz et al. 2013, Jakubowicz et al. 2017). However, to the best of our knowledge, only two studies have examined the effect of timing of intake on body composition and found conflicting results (Muñoz et al. 2017, Xiao et al. 2019). Results from our study showed that the timing of dietary intake was associated with body composition. Being a later chronotype (ET) in the high AG ratio and BF% groups, predicted lower energy, protein and carbohydrate intakes in the morning, and higher energy, carbohydrate, and fat intakes at night. Thereby showing that nutritionally ET do not make the most of the early part of the day, which is

metabolically the ideal circadian phase of the day for food intake and optimal nutrient utilisation (Roberts 1964, Takahashi et al. 1968, Shapiro et al. 1991, Boden et al. 1996, Van Cauter et al. 1997, Damiola et al. 2000, Saad et al. 2012, Cipolla-Neto et al. 2014, Covassin et al. 2016 , Rácz et al. 2018).

***Thesis objective 4**: to investigate whether chronotype is associated with metabolic biomarkers (chapter 5).*

This PhD thesis is one of a handful of studies that explored metabolic biomarker differences among a healthy chronotype population (Lázár et al. 2012, Lucassen et al. 2013, Vera et al. 2018, Muñoz et al. 2020). Earlier chronotypes (MT-IT) had an overall healthier metabolic profile and lower concentrations of triglycerides, insulin, leptin, HbA1c and higher HDL-cholesterol and ghrelin concentrations in comparison with ET. Higher total cholesterol and LDL-cholesterol concentrations were found in the MT-IT, which has not been reported by other studies. The unexpected higher total- and LDL-cholesterol concentrations seen among MT-IT in this data set could be related to their dietary patterns (see also chapter 6) that revealed lower intakes of carbohydrates and higher intakes of saturated fat well known to influence total- and LDL- cholesterol concentrations (Noakes et al. 2006, Bueno et al. 2013).

We further demonstrated that the timing of energy intake was correlated with certain biomarkers. A higher evening energy intake was positively correlated with higher HbA1c, insulin and triglyceride concentrations. In contrast, a higher morning energy intake was negatively correlated with lower HbA1c, insulin and leptin concentrations, and positively correlated with a higher HDL-cholesterol concentration. This study found that MT-IT consumed most of their total daily energy during the morning and in comparison, ET consumed the most during the evening. These findings suggest that MT-IT, who consume more energy during the morning may have an overall healthier metabolic profile, whilst the ET, who consume more energy during the evening have a less healthy metabolic profile. A clear link between timing of energy intake and metabolic biomarkers cannot be established by this study due to the cross-sectional design.

***Thesis objective 5**: To identify dietary patterns (food and nutrient patterns) and investigate if the patterns are associated with chronotype and body composition (chapter 6).*

To our knowledge this is the first study to not only investigate dietary patterns among chronotypes, but also the first to extract both food- and nutrient dietary patterns in the same study creating a whole diet picture

Firstly, we extracted food patterns, by categorising the foods from 5DFR into 33 food groups. The following food patterns were extracted and named according to the high correlating foods in each

pattern: *Healthy, Discretionary Food & Beverages-, Animal Products-* and the *Sugary, Fatty Foods* food patterns. The majority of the MT-IT women followed the *Healthy-, Discretionary Food & Beverages-, and Animal Products* food patterns, while ET were 0.08, 0.39 and 0.49 times less likely (respectively) to follow these food patterns.

The *Healthy* food pattern consisted of fibre- and micronutrient dense foods such as coloured- and non-starchy vegetables, fruits, and wholegrains. Women classified as having a high BMI and BF% were 0.36 and 0.33 times less likely (respectively) to follow the *Healthy* food pattern. These results suggest that the MT-IT who follow this food pattern, were more likely to have a healthy body composition. These findings are important as a high fibre diet is associated with lower body composition (Rolls et al. 2004, Svendsen et al. 2007, Rolls et al. 2010), total cholesterol concentrations and systolic blood pressure and protective against cancer, type two diabetes and cardiovascular diseases (Reynolds et al. 2019). Our *Healthy* food pattern was similar to a NZ based study that identified a "Fruit and Vegetable" and "Healthy" food pattern (Schrijvers et al. 2016), with the exception that our food pattern excluded breakfast cereals, starchy vegetables and alcohol.

The *Discretionary Food & Beverages* food pattern was high in beverages such as alcohol, milk alternatives, fruit juice and to a lower extent coffee and tea. This more MT-IT pattern was interesting as others have shown that ET consumed higher amounts of alcohol and caffeine (Sato-Mito et al. 2011, Lázár et al. 2012, Culnan et al. 2013, Maukonen et al. 2016, Maukonen et al. 2017, Vera et al. 2018, Najem et al. 2020). Furthermore, this food pattern did contain some micronutrient-dense foods such as fish & seafood, sweetened dairy products, fruits and legumes which was supported by the higher intakes of vitamin E, -C, folate, iodine, magnesium, calcium, and potassium.

The *Animal Products* food pattern was characterised by high animal protein sources such as meats, eggs, cheese, fish & seafood, and dairy milk and no carbohydrate food sources. This finding is contradictory to the majority of chronotype studies, that found that ET are more likely to consume a diet high in cholesterol-rich foods (Mota et al. 2016), and animal protein (Sato-Mito et al. 2011, Silva et al. 2016, Muscogiuri et al. 2020) rather than earlier chronotypes, as shown by our results. These results are important as a diet that is low in carbohydrates but higher in animal proteins and fats are associated with higher mortality risk (Seidelmann et al. 2018).

The *Sugary, Fatty Foods* food pattern was high in added sugar and sweet spreads, sweet snack foods such as sweets, and saturated fats from animal and coconut sources. A diet high in added sugar and fats is known to be associated with a high BMI (Newby et al. 2003, Paradis et al. 2009, Suliga et al. 2015, Bray et al. 2018), which was also shown by our results. The women in the high BMI group were 2.09 times more likely to follow this food pattern. The pattern was however not associated with a specific chronotype group.

Secondly, we extracted also nutrient patterns, by using the total nutrient intakes derived from the 5DFR. Four patterns were extracted: the *Micronutrient-Rich-*, *High Protein-Fat-Fibre-*, *Vitamin B Rich* and *High Carbohydrate* nutrient patterns. Most of the MT-IT women followed the *High Protein-Fat-Fibre* nutrient pattern, while most of the ET followed the *High Carbohydrate* nutrient pattern.

Two of the nutrient patterns were micronutrient specific, namely the *Micronutrient-Rich and High Vitamin-B* nutrient patterns, which were not associated with chronotype. Previous researchers have extracted distinct micronutrient patterns that was inversely associated with cancer risk (Palli et al. 2001, Bosetti et al. 2013). When inspecting the food sources of these micronutrient patterns it was more representative of the MT-IT *Healthy* - and *Animal Products* food patterns.

The *High Protein-Fat-Fibre* nutrient pattern was high in fat, cholesterol, and protein and lower in carbohydrates. Inspecting the food sources, the nutrient content of this pattern was derived from animal and plant protein sources, saturated fats from animal & coconut sources and unsaturated plant-based fat sources. More MT-IT followed this pattern, than ET women. Apart from the quantity of nutrients in the diet, the quality of these nutrients, is a crucial determining factor of the effect of the diet on body weight regulation. For example, in the case of population groups who consume a diet favouring unsaturated fatty acids intake above SFA intake, such as the Mediterranean diet, are at a lower risk of developing obesity (Bes-Rastrollo et al. 2006, Gulati and Misra 2017, Beulen et al. 2018). This nutrient pattern was high in both healthy unsaturated and unhealthy saturated fats, with saturated fats being the lowest of the two. This nutrient pattern was high in both animal- and plant-based protein food sources, which is important as diets that are lower in carbohydrates but higher in plant-based protein is associated with a reduced risk of mortality (Seidelmann et al. 2018).

The last nutrient pattern was the *High Carbohydrate* nutrient pattern, due to its high carbohydrate, starch, and sugar content. When inspecting the food sources of this nutrient pattern, it was high in simple carbohydrates such as sugar- and artificially sweetened beverages, refined grains, savoury snack foods, discretionary snack breads, cakes and puddings, fruit juices, sugar-added to foods and beverages and sweet spreads, which is typical of ET (Baron et al. 2011, Sato-Mito et al. 2011, Mota et al. 2016, Maukonen et al. 2017, Yoshizaki et al. 2018, Li et al. 2018, Muscogiuri et al. 2020). These findings are important as diets rich in simple carbohydrates, are associated with increased obesity risk (Malik et al. 2010) and high carbohydrate intake with high mortality risk (Seidelmann et al. 2018). Moreso, a nutrient pattern analysis study found their "Starch Rich" nutrient patterns to be associated with elevated cancer risk (Bosetti et al. 2013). These findings suggest that ET that follow the *High Carbohydrate* nutrient pattern, may be at higher risk for obesity, cancer, and mortality.

Thesis objective 6*: to investigate the associations between chronotype, eating behaviours (restraint, hunger, and disinhibition), eating attitudes (dieting, oral control, and bulimia & food preoccupation), and body composition (chapter 7).*

We utilised the TFEQ and the EAT-26 from the PROMIsE study to collect data on eating behaviours and – attitudes (respectively). For the first time, eating behaviours, and -attitudes among chronotypes were explored in depth, by investigating not only the main TFEQ and EAT-26 categories but the subcategories as well, within different body composition groups. Exploring eating behaviours in relation to body composition is vital, as several researchers (that have not investigated chronotypes) showed the TFEQ behaviours to be predictors of BMI (Dykes et al. 2004, Kruger et al. 2016), BF% (Kruger et al. 2016) and body adiposity (de Lauzon-Guillain et al. 2006), thereby suggesting that eating behaviour is a modifiable risk factor for weight gain and obesity.

Our results showed that a later chronotype (ET) predicted all three of the main TFEQ categories, namely lower *restraint* (conscious restriction of food intake), and higher *hunger* scores, in the high BMI group and lower *disinhibition* score (loss of control over food intake) in the normal BMI group. By way of explanation, ET in this population were more likely to relax self-set dietary restrictions and act on feelings of *hunger*, which may ultimately lead to overconsumption of food, less successful weight loss attempts and a high BMI. However, the ET that had lower *disinhibition*, had more control over their food intake and consequently had a healthy BMI. A previous study looking at different chronotypes, also found higher *hunger* but lower *disinhibition* scores in ET, while MT had high *restraint* scores (Schubert & Randler 2008).

We further investigated the seven subcategories of the TFEQ in our data set of obese and lean women and found that a later chronotype (ET) predicted a lower score of the obesity-protecting behaviour of *flexible restraint,* and higher obesity-promoting behaviours of *habitual disinhibition,* and *internal locus for hunger* scores in the high BMI group. Equivalent results were seen among the ET in the high BF% group, linking ET-specific behaviours to a higher body composition. The subcategories of the TFEQ have been shown to be important predictors of body composition. For example, higher *flexible control* equals a more balanced approach to dieting (Westenhoefer 1991) and is associated with a lower body composition and greater weight loss success (McGuire et al. 2001, Provencher et al. 2003, Hays & Roberts 2008). Our result showed higher *restraint* and *flexible control* to be behaviour displayed by the earlier chronotypes (MT-IT), rather than the ET. Higher susceptibility to *habitual disinhibition* was seen in the ET, meaning that they habitually overeat in reaction to everyday circumstances leading to weight gain (Hays & Roberts 2008).

The TFEQ's eating behaviours interact with other behaviours such as *disinhibition, which* in turn has an interaction effect with either a low/high level of *restraint* and *hunger* (Hays 2002). Combinations of these eating behaviours (Lesdéma et al. 2012) are also associated with body composition (Provencher et al. 2003, Dykes et al. 2004, Kruger et al. 2016). These eating behaviours combinations created by Lesdéma et al (Lesdéma et al. 2012) are rarely explored and have not been investigated among chronotype groups. Our results showed that MT-IT women displayed a more ideal combination of *high*

restraint- low disinhibition when compared to ETs. On the other hand, the ET in the high BMI group, were 3.93 times more likely to display the least ideal combinations of *low restraint - high disinhibition* in comparison with MT-IT. Furthermore, in the hunger and disinhibition categories, the majority of MT-IT women had the ideal combination of *low hunger - low disinhibition*. Interestingly the ET in the normal BMI group were 0.16 times less likely to display opposing *high hunger - low disinhibition* combination when compared with MT-IT, showing that not all ET displayed unhealthy behaviours.

Individuals with high *restraint* and *disinhibition* are shown to have a higher risk of developing eating disorders (Allison & Heshka 1993). The EAT-26 does not diagnose eating disorders but rather identifies eating attitudes that are related to eating disorders. The total EAT-26 score was not predicted by chronotype in this study, nor in another study (Harb et al. 2012), although more of the ET women had a score of ≥20, indicating more disturbed eating attitudes. With regards to the subcategories of the EAT-26, a later chronotype (ET) predicted higher *bulimia & food preoccupation* scores in both the high BMI and BF% groups. This is similar to others, that found a higher risk of bulimic behaviour (Kasof 2001) and higher Binge Eating Scale score (Harb et al. 2012) in ET. Theories explaining this higher risk of bulimia and behaviours included later sleep and wake times (Kasof 2001), that was seen in this study, as well as light exposure during the biological night (Kasof 2001).

The satiety and hunger hormones of leptin and ghrelin are integral in energy homeostasis, with imbalances of these hormones affecting body weight regulation (Morioka et al. 2016). Only few studies have shown associations between these hormones and eating behaviours (Blundell et al. 2008, Schur et al. 2008, Langlois et al. 2011, Benbaibeche et al. 2021) and no study were done with respect to chronotype differences. We showed that leptin and ghrelin were associated with certain eating behaviours and - attitudes. The more ET-behaviours of *hunger*, *disinhibition* and *bulimia & food preoccupation* was positively correlated with a higher leptin blood concentration. While the more MT-IT behaviour of *restraint* was positively correlated with ghrelin concentrations, suggesting that individuals, like those with an earlier chronotype (MT-IT) and elevated appetite (higher ghrelin) consciously increased their *restriction* of food intake as a measure to potentially prevent weight gain (Schur et al. 2008).

8.3 Conclusion

This PhD work showed that women with a later chronotype displayed obesity-promoting dietary factors, pertaining to their (1) timing of food intake ("when", chapter 5), (2) types and combinations of food choices ("what", chapter 6), and the (3) eating behaviours and - attitudes ("how", chapter 7) that precede and drive food intake (**Figure 8.2**).

We found that ET women were predisposed to consume food at the incorrect time during the light-dark cycle, which may have a desynchronising effect on the rhythm of circadian clocks', altering the body's normal daily metabolic processes. All this could in the long-term result in obesity and poor metabolic health outcomes (Salgado-Delgado et al. 2010, Nelson & Chbeir 2018, Challet 2019). The late eating among ET is most probably related to their later sleep-wake timing (Roenneberg et al. 2007), where they rise later in the morning and go to bed later at night in comparison with earlier chronotypes. The ET are plausibly not hungry early in the morning, resulting in increased vulnerability to feelings of hunger later in the day and especially during the evening, with subsequent "catch-up" of the missed meals. Further exacerbating this nightly catch-up of missed meals are certain eating behaviours that might create a setting for mindless eating which leads to overeating. The evening could be a more relaxed part of the day for most people, coupled with TV watching, social activities and alcohol (Sinnett et al. 1983) intake, making it easier to relax self-set dietary restrictions and weight goals (Seddon & Berry 1996) and lose control of food intake. This leads to higher quantities of food intake as well as poorer food choices for example as snack foods such as potato crisps, popcorn, sweets, and sugar sweetened beverages. This research study showed that the women who consumed the highest proportion of their energy intake during the night correlated positively with higher glucose homeostasis markers and higher triglyceride concentrations. In contrast, women who consumed the most during the morning correlated negatively with lower glucose homeostasis markers and leptin concentration, and positively correlated with higher HDL-cholesterol concentration. Thereby creating a vicious cycle starting with late rise times, skipped or delayed early meals, higher feelings of hunger, loss of control over intakes and ending with higher food intake during the normal fasting phase of the light/dark cycle. These habits result in altering metabolic processes and enhancing fat storage (Salgado-Delgado et al. 2010 , Nelson & Chbeir 2018, Challet 2019).

This PhD work found that Pacific women in our sample were more likely to be ET and that the NZ European women were more likely to be MT. Unfortunately, statistical analysis for chronotype groups could not be stratified according to ethnicity for this analysis due to the low sample size of MT in the Pacific women as well as low MT and ET in NZ European women. However, it seems that Pacific women who are more likely ET are therefore more likely to display ET-dietary aspects that are linked with a higher body composition and poorer metabolic health profile.

Thus, this work confirms that chronotype might be an important risk factor for obesity that is worth assessing at an individual level. Results from this thesis indicate that along with the standard body composition measurements, 24-hour diet recalls and diet histories, that chronotype assessed by health care practitioners could be a helpful tool. Strategies aimed at restructuring sleep-, wake times and subsequent nutrient distribution and food choices to align with the body's normal circadian timing, favouring daytime for food intake may be helpful for weight reduction.

In conclusion, morning food intake is an important start to the day and completely skipping the morning meal is not advisable. Breaking the nightly fast at some point in the morning, may prevent eating behaviours that precedes higher dietary intakes during the night. *The early bird does indeed catch the worm.*

Eating Behaviours:

Among high BMI group, ET predicted ($P < 0.05$):

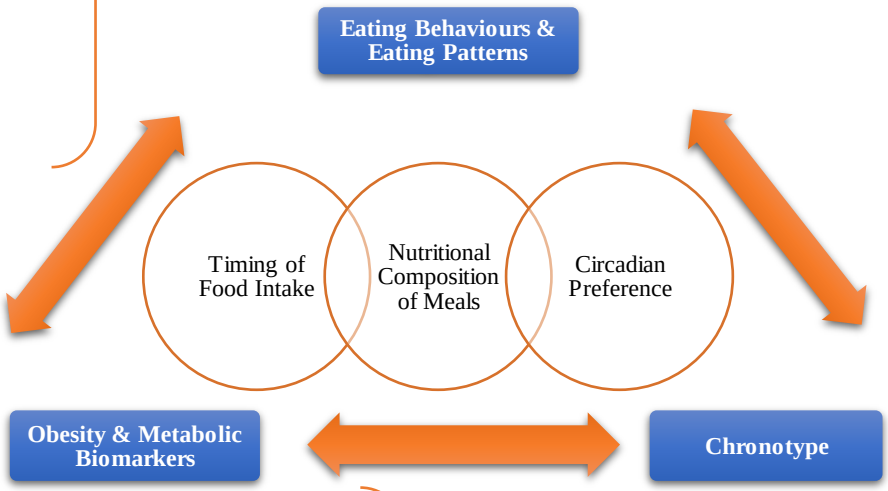
↓ *restraint (β = -1.86),*
↓ *flexible control (β = -0.84),*
↓ *rigid control (β = -0.75),*
↑ internal hunger (β = 0.97),
↑ hunger (β = 1.64),
↑ habitual disinhibition (β = 0.66),
↑ bulimia & food preoccupation (β = 1.61)

Among high BF% group, ET predicted ($P < 0.05$):

↑ internal hunger (β = 0.82),
↑ bulimia & food preoccupation (β = 1.75),
↓ *situational disinhibition (β = -1.10)*

Eating Patterns (timing of intake):

Morning Intake (MT-IT vs ET had higher intakes, $P < 0.01$)
MT-IT: Energy: 1306 kJ, Protein:12.6 g, Carbohydrates: 32.8 g, Fat: 13.5 g
ET: Energy: 788 kJ, Protein: 7.58 g, Carbohydrates: 21.9 g, Fat: 7.35 g
Evening Intake (ET vs MT-IT had higher intakes, $P < 0.01$)
MT-IT: Energy: 941 kJ, Protein: 8.10 g, Carbohydrates: 21.6 g, Fat: 10.2 g
ET: Energy: 1979 kJ, Protein: 17.8 g Carbohydrates: 48.7g, Fat: 20.8 g



Eating Patterns (dietary pattern analysis):

Odds ratio for ET vs MT-IT to score high:

↓ *Healthy food pattern*
↓ *Discretionary Food & Beverages food pattern*
↓ *Animal Products food pattern*
↓ *High Protein-Fat-Fibre nutrient pattern*
↑ *High Carbohydrate* nutrient pattern

Body Composition:

MT-IT vs ET, $P < 0.05$

MT-IT: *BMI: 26.1 kg/m^2, BF%: 34%, AG ratio: 0.87*
ET: BMI: 31.4 kg/m^2, BF%: 36%, AG ratio: 0.98
Among high BF% and high AG ratio groups, ET predicted intakes in the morning ($P < 0.05$):
↓ energy intake (β = -513 and β = -327),
↓ protein intake (β = -4.48 and β = -3.14),
↓ carbohydrate intake (β = -10.7 and β = -9.38)
Among high BF% and high AG ratio groups, ET predicted intakes in the evening ($P < 0.05$):
↑ energy intake (β = 603 and β = 684),
↑ carbohydrate intake (β = 12.9 and β = 16.3),
↑ fat intake (β = 7.25 and β = 7.50)

Chronotypes:

MT-IT: 66%
ET: 34%
Average wake times:
MT-IT: 07:18 vs **ET: 09:48**, $P < 0.01$)
Average sleep onset:
MT-IT: 22:54 vs **ET:** 00:48, **$P < 0.01$)**

Figure 8.2 *Main findings from this PhD thesis of the PROMIsE study.*

8.4 Strengths

This PhD research was the first to explore the diet in-depth as it relates to chronotypes as a mediator in the development of obesity and adverse metabolic health. We contributed to the limited studies that explored this field, creating an overall picture of the eating patterns, eating behaviours, body composition and metabolic biomarker differences between chronotype groups. Specific strengths included:

- Body composition was explored beyond BMI, including body fat percentage and -distribution. Thereby addressing the limitations of BMI as adiposity measurement and creating a comprehensive body composition profile for each chronotype group.
- This study delved into chronotype-specific dietary intakes beyond absolute energy and nutrient intakes. We compiled a meticulously detailed analysis of eating patterns including nutritional composition and timing of meals. The chronotype-specific distribution of dietary intake in relation to body composition was placed at the centre of this body of work.
- This PhD research was the first to create not only food patterns but also nutrient patterns by use of principle component analysis and investigated the association with chronotypes and body composition. This approach provided a thorough analysis of the typical food profiles as well as the typical nutrient profiles consumed together at meals.
- Metabolic biomarker differences in chronotypes have previously been explored in unhealthy populations. The PROMIsE study participants only included healthy women; thus, the absence of all unhealthy participants allowed us to create a clear picture of the consequences of chronotype-specific eating patterns on metabolic health markers.
- The relationship between chronotype-specific- eating behaviours, - attitudes and body composition was investigated in-depth, which is a first among chronotype research. We not only investigated the main TFEQ categories, and the total EAT-26 scores but also the subcategories of each of the measuring tools, creating a more comprehensive behaviour profile for different chronotypes.

8.5 Limitations

- The original study design did not contain measures of the ‘gold standard’ for assessment of circadian phase, the DLMO, (e.g., from saliva or blood samples) (Kantermann et al. 2015, Lewy & Sack 1989). Therefore, for this thesis, chronotype was assessed from midsleep times (corrected for sleep durations on free days) from the MCTQ. These midsleep times served as proxy for circadian phase.

- Sleep duration and quality certainly have a crucial impact on body composition and metabolic health outcomes (Gutiérrez-Repiso et al. 2014). Eveningness is associated with decreased sleep quality (Carciofo et al. 2014) and increased sleep debt (Bakotic et al. 2017). However, regression models in this PhD research were not adjusted for sleep duration and quality for the reason that the main focus of the analysis was not to investigate sleep-related eating behaviours. However, self-reported sleep duration was indirectly considered by using the MSFsc (midsleep corrected for sleep duration on free days) derived from the MCTQ.
- The cross-sectional nature of the study allowed only for the examination of dietary intake, metabolic biomarkers, and body composition as a snapshot in time and therefore changes over time was not possible.
- The PROMIsE study aimed to recruit equal numbers of participants in each body composition and ethnic group and at the time did not select different chronotypes. As a result, we had a small sample size for MT. This is not unexpected in this young sample of women as morningness is associated with older adults (Roenneberg et al. 2004, Tonetti et al. 2008, Randler & Engelke 2019). We thus collapsed the MT and IT into one group of chronotypes for statistical analyses, but by always showing descriptive statistics for all three chronotype groups (MT, IT and ET).
- Statistical analysis could not be further stratified by ethnicity, due to the inadequate sample size of MT in the Pacific group as well as the NZ European group, and therefore ethnicity was treated as a covariate. Regression models showed where ethnicity was statistically impacting results.
- The study sample was not representative of the entire NZ population as the aim was to include the ethnic group with the highest (Pacific) and the lowest risk (NZ European) of obesity. Therefore, other ethnicities were not included in this study.
- The recruitment strategies used, for example the email lists from the Massey University previous nutrition studies, may have attracted more health-conscious women, which may be considered as a potential source of bias.
- Women from only urban areas of Auckland was included in the study, which may have impacted the types of foods reported in the 5DFR. Individuals from urban areas have easy access to restaurants and fast foods than for example individuals from rural areas.
- Socioeconomic status may also have had an indirect impact on outcome variables. Even though in our cohort Pacific women were later chronotypes and had lower socio-economic status, it is not clear, to which extend chronotype is affected by socio-economic status. The question is rather whether ethnic inequities are the more important driver, as it was shown in a recent cross-sectional study which compared sleep durations of Māori vs. non-Māori. The authors found that Māori individuals had shorter sleep durations which was best explained by socio-economic status (Paine & Gander 2016). These differences remained, even when adjusting for other

health factors and chronotype. In another previous study with a NZ cohort including adult Maori and Non-Maori, it was concluded that '…morningness/eveningness preference is largely independent of ethnicity, gender, and socioeconomic position, indicating that it is a stable characteristic that may be better explained by endogenous factors (Paine et al. 2006). Therefore, the interaction and impact of socioeconomic status on other factors, such as dietary intake might also have contributed to the differences, we found between the Pacifica women and European women in this study. However, this study only had one measure of socio-economic status namely the NZ deprivation index and therefore could not be fully assessed.

8.6 Recommendations for future research

- The main recommendation for future studies would be 1) assessing chronotype and 2) having an equal number in each group for each ethnicity, by screening potential participants with the shortened version of the MCTQ for example.
- It was evident form the systematic review that included studies used a mixed methodology to classify chronotype, including using median chronotype score or tertiles of chronotype scores. The result being that "true extreme chronotypes" were not unambiguously identified by these studies. Therefore, it is recommended that future studies define chronotype according to the determined cut off points of the specific chronotype questionnaire.
- From the literature, it is evident that metabolic processes and endocrine markers show circadian rhythmicity. Thus, investigating the concentrations of metabolic biomarkers at different time points e.g., before and after food intake or in the day vs the evening, may provide more precise information on how chronotype affect biomarkers. For example, a postprandial RCT where a standardised meal is provided to participants. After which leptin and ghrelin concentrations are measured at different time points to see if there are differences in the postprandial response of these hormones between chronotypes.
- It is recommended that changes in body composition markers in chronotype groups should be studied using a longitudinal study design to investigate for example weight gain or weight loss over time between chronotypes.
- This study described the effects of chronotype-specific meal timing in relation to bodyweight at one time point using external clock hours. However, different chronotypes have different phases of entrainment with regards to external clock times, thus optimal meal timing may differ among individuals in accordance with their chronotype. To explore this further, information about the circadian phase relative to mealtimes are required. This can be achieved by determining circadian phase with measurement of DLMO, e.g., in saliva samples of each study participant. In addition, to determine internal desynchronisation, additional measures would be necessary, as described by Qian and colleagues (2022). This study by Qian and colleagues

(2022) was conducted under very controlled laboratory conditions in an intensive and long-lasting protocol. A simpler approach would be to provide a standardised meal (i.e., adjusted for kg body weight and height) during each of chronotypes active and feeding phase, relative to a standard measure of internal circadian phase once under real life conditions, and once under preferred conditions, which suit sleep-wake timing of each chronotype, and the outcomes can be compared.

- Time restricted eating has been shown to have a role in body weight regulation. We recommend that the differences in fasting period length (between the last meal of the day and first meal of the preceding day) between chronotype groups be further explored.

8.7 Recommendations for preventing obesity and poor metabolic health

- Be physically active during the day and consume the largest proportion of your daily dietary intake during the early morning and afternoon. Daytime is the circadian phase that is metabolically suited to food intake.
- Consume breakfast before 10:00. Do not skip breakfast. This will prevent feelings of hunger later in the day and consequent overconsumption of food and unhealthy food choices later in the day, especially at night.
- Prioritise night-time for sleep and rest. Get enough sleep (7-8 hours) during the night as insufficient sleep is associated with weight gain, insulin resistance and type 2 diabetes.
- Avoid excessive light exposure during the night, for example light from television and cell phone screens. Nocturnal light exposure delays melatonin onset as well as decrease total melatonin secretion, which delays sleep onset and may cause disturbances in glucose metabolism.
- Consume the smallest proportion of your daily dietary intake during the early evening and ensure an overnight fast. Consuming food late at night forces the body to digest and metabolise food during the normal fasting phase of the light/dark cycle. Consequently, the stored energy forms are not mobilised during the night to maintain homeostasis and enhanced fat storage is seen.
- Avoid situations that lead to night-time snacking after having an early dinner for example, snacking while watching television or drinking an energy dense drink in bed.
- Have regular mealtimes as it will help the body anticipate food intake at the correct time.

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Declaration

I, Carlien van der Merwe, hereby declare that this PhD thesis is my own work. I further declare that:

1. I contributed to the conceptualisation, data curation, data processing, statistical analysis, creation of figures and data interpretation of all the manuscript chapters. I also prepared the first and final drafts of all the manuscript chapters,
2. I wrote the text of all the remaining chapters contained in this thesis,
3. the text and bibliography reflect the sources I have accessed, and
4. sections with no source referrals are my own ideas, and/or conclusions.
5. where I have made use of any graphic works (such as figures) from someone else, I have obtained the necessary written approval of the relevant publisher(s)/creator(s) of such works and/
6. I have reproduced the published systematic review titled: Chronotype Differences in Body Composition, Dietary Intake and Eating Behavior Outcomes - A Scoping Systematic Review in this PhD thesis under the terms of the Creative Commons Attribution-Non-commercial License and have referenced the original place of publication.

Signature: 

Date: 29-05-2023

Appendix A.1 Ethics committee approval letter

Health and Disability Ethics Committees

Health and Disability Ethics Committees
Ministry of Health
Freyberg Building
20 Aitken Street
PO Box 5013
Wellington
6011

0800 4 ETHICS
hdecs@moh.govt.nz

30 March 2016

Professor Bernhard Breier
Private Bag 102904
North Shore Mail Centre
Auckland 0745

Dear Professor Breier

Re:	**Ethics ref:**	**16/STH/32**
	Study title:	The gut microbiome: a new pathway to obesity prevention and metabolic health

I am pleased to advise that this application has been *approved* by the Southern Health and Disability Ethics Committee. This decision was made through the HDEC-Expedited Review pathway.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Southern Health and Disability Ethics Committee is required.

Standard conditions:

1. Before the study commences at *any* locality in New Zealand, all relevant regulatory approvals must be obtained.

2. Before the study commences at *a given* locality in New Zealand, it must be authorised by that locality in Online Forms. Locality authorisation confirms that the locality is suitable for the safe and effective conduct of the study, and that local research governance issues have been addressed.

Non-standard conditions:

- Please remove the words 'exciting' and 'greatly' from the opening paragraph.
- Replace the word 'encourage' with 'invite'.
- Data needs to be kept for 10years (says 5years on the consent form).
- Please add the study name to the footer and version number.
- The sections in the information sheet about breastfeeding are confusing – please re-word.
- Currently it is not clear whether participants could not be breastfeeding for longer than 6 months (i.e be breastfeeding but planning to stop within the next 6

months); whether they could not have breastfed for more than 6 months in total any sometime in the past; or whether they could not have breastfeed within the last 6 months.

Non-standard conditions must be completed before commencing your study. Non-standard conditions do not need to be submitted to or reviewed by HDEC before commencing your study.

If you would like an acknowledgement of completion of your non-standard conditions letter you may submit a post approval form amendment. Please clearly identify in the amendment that the changes relate to non-standard conditions and ensure that supporting documents (if requested) are tracked/highlighted with changes.

For information on non-standard conditions please see section 128 and 129 of the Standard Operating Procedures at http://ethics.health.govt.nz/home.

After HDEC review

Please refer to the *Standard Operating Procedures for Health and Disability Ethics Committees* (available on **www**.ethics.health.govt.nz) for HDEC requirements relating to amendments and other post-approval processes.

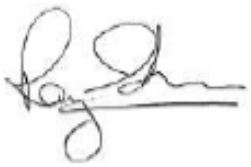
Your next progress report is due by 29 March 2017.

Participant access to ACC

The Southern Health and Disability Ethics Committee is satisfied that your study is not a clinical trial that is to be conducted principally for the benefit of the manufacturer or distributor of the medicine or item being trialled. Participants injured as a result of treatment received as part of your study may therefore be eligible for publicly-funded compensation through the Accident Compensation Corporation (ACC).

Please don't hesitate to contact the HDEC secretariat for further information. We wish you all the best for your study.

Yours sincerely,



Ms Raewyn Idoine
Chairperson
Southern Health and Disability Ethics Committee

Encl: appendix A: documents submitted
appendix B: statement of compliance and list of members

Appendix A
Documents submitted

Document	*Version*	*Date*
CV for CI: Bernhard Brier CV	1	14 March 2016
Protocol: Protocol Promise study	2	21 March 2016
PIS/CF: Information Sheet PROMISE study	1	21 March 2016
PIS/CF: Consent form PROMISE study	1	21 March 2016
Survey/questionnaire: Screening questionnaire	1	21 March 2016
Survey/questionnaire: Demographic questionnaire	1	21 March 2016
Survey/questionnaire: Dietary TFEQ Three factor eating questionnaire	1	08 March 2016
Survey/questionnaire: Dietary DDQ dietary diversity questionnaire	1	08 March 2016
Survey/questionnaire: Dietary FFQ food frequency questionnaire	1	08 March 2016
Survey/questionnaire: Dietary EAT-26 questionnaire	1	02 March 2016
Survey/questionnaire: Dietary food record & instructions	1	08 March 2016
Survey/questionnaire: PA RPAQ recent physical activity questionnaire	1	08 March 2016
Survey/questionnaire: PA Actigraph instructions	1	18 March 2016
Survey/questionnaire: PA physical activity diary	1	21 March 2016
Survey/questionnaire: Sleep Pittsburgh questionnaire	1	21 March 2016
Survey/questionnaire: Sleep general Sleep disturbance scale	1	21 March 2016
Survey/questionnaire: Sleep Munich chronotype questionnaire	1	21 March 2016
Survey/questionnaire: Sleep Berlin questionnaire	1	21 March 2016
Survey/questionnaire: Sensory sample preparation & testing	1	21 March 2016
Survey/questionnaire: Sensory TASTE questionnaire	1	07 March 2016
Survey/questionnaire: Sensory RANK questionnaire	1	07 March 2016
Survey/questionnaire: Faecal collection instructions	1	21 March 2016
Evidence of scientific review: HRC grant notification	1	21 March 2016
Application		

Appendix B
Statement of compliance and list of members

Statement of compliance

The Southern Health and Disability Ethics Committee:

- is constituted in accordance with its Terms of Reference
- operates in accordance with the *Standard Operating Procedures for Health and Disability Ethics Committees*, and with the principles of international good clinical practice (GCP)
- is approved by the Health Research Council of New Zealand's Ethics Committee for the purposes of section 25(1)(c) of the Health Research Council Act 1990
- is registered (number 00008713) with the US Department of Health and Human Services' Office for Human Research Protection (OHRP).

List of members

Name	*Category*	*Appointed*	*Term Expires*
Ms Raewyn Idoine	Lay (consumer/community perspectives)	27/10/2015	27/10/2018
Dr Devonie Eglinton	Non-lay (intervention studies)	01/07/2013	01/07/2016
Mrs Angelika Frank-Alexander	Lay (consumer/community perspectives)	27/10/2015	27/10/2018
Dr Sarah Gunningham	Non-lay (intervention studies)	27/10/2015	27/10/2018
Assc Prof Mira Harrison-Woolrych	Non-lay (intervention studies)	27/10/2015	27/10/2018
Dr Fiona McCrimmon	Lay (the law)	27/10/2015	27/10/2018
Dr Nicola Swain	Non-lay (observational studies)	27/10/2015	27/10/2018
Dr Mathew Zacharias	Non-lay (health/disability service provision)	27/10/2015	27/10/2018

Unless members resign, vacate or are removed from their office, every member of HDEC shall continue in office until their successor comes into office (HDEC Terms of Reference)

http://www.ethics.health.govt.nz

Appendix A.2 Information sheet

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE STUDY

(PRedictors linking Obesity and gut MIcrobiomE)

INFORMATION SHEET

We are looking for women aged between 18 and 45 years of age to take part in the PROMISE study. We want to understand how the bacteria in our gut are affected by diet, physical activity, sleep and taste perception, and how this is linked to health and a healthy body weight. Please read this information sheet carefully before deciding whether or not to participate.

What is this research about?

Here in New Zealand we are experiencing an epidemic of obesity and metabolic diseases, such as diabetes. A key contributing factor is our diets and the types of food we eat. Food has a large impact on the number and types of bacteria in our bowel (gut microbiome). There are at least 1000 different types of bacteria. It is thought that these bacteria play a very important role in our health, and may be contributing to obesity in some people. In this study we will investigate how diet, taste perception, sleep and physical activity affect the gut microbiome. This new knowledge will help us understand obesity and how best to prevent it.

Who are we looking for?

We are looking for women to participate in this study. To take part in this study you need to:

- Be between 18 and 45 years of age
- Be of NZ European ethnicity OR of Pacific ethnicity
- Not be pregnant or breastfeeding
- Not have any chronic diseases such as heart disease, diabetes or cancer
- Not have any food-allergies
- Not be following a severely restricted diet

What is involved in the study?

If you decide to take part in the study, you will be asked to complete a screening questionnaire (online or over the phone) that will assess your eligibility. If you meet these eligibility criteria you will be invited to take part in the study, which includes 2 visits to the Massey University Human Nutrition Research Unit in Albany and collection of data at home.

Visit 1 (approximately 2.5 hours)

You will have to visit the Human Nutrition Research Unit early in the morning (between 7.30 and 8:30am), without having eaten breakfast. The visit will take approximately 2.5 hours. On the day we will first explain to you what is involved in the study and answer all your questions. We will then ask you to sign a consent form.

The following procedures will take place during this visit:

- Your height, weight and body composition will be measured.
- A blood sample will be taken from a vein in your arm by a trained person (a phlebotomist). For this blood sample you need to have fasted overnight. **You should not eat or drink anything (other than water) from 10pm the previous evening until after the blood sample has been taken.** We will use the blood samples to measure cholesterol and glucose levels, markers of inflammation and small molecules related to fat usage and storage.
- You will be asked to taste a few sweet, fat and bitter solutions by sipping a mouthful (10mL or 2 teaspoons), swirling it in your mouth and swallowing it, and then rating your preference on a scale.
- A buffet-style continental breakfast will be provided prior to further measures to ensure that you break your fasted state.
- You will also be asked to complete some questionnaires regarding your diet and sleep patterns.
- We will ask you to provide a small urine sample.

At home between Visits 1 & Visit 2

You will be asked to:

- Wear an Accelerometer (a small device similar to a step-counter that you wear around your waist to measure physical activity), and an Actiwatch (a watch worn on your wrist which records your sleep patterns) for 7 days. The accelerometer and watch will be provided by the researchers and you will receive detailed instructions on how to use them.
- Record your physical activity for 7 days while you are wearing the small devices above.
- Record your food intake for 5 days while you are wearing the small devices above.
- Provide two small faecal (stool) samples and bring these to Visit 2. It is important that these two small stool samples are frozen (in the containers provided by us) immediately after collection in your freezer. These samples allow us to determine the bacteria (microbes) in your gut.

Visit 2 (approximately 2 hours)

You will be asked to visit the Human Nutrition Research Unit during the day. For this visit we ask that you eat as usual before coming as no food will be provided during Visit 2. The visit will take about 2 hours.

At this visit:

- You will bring back all your devices and the stool samples and your physical activity and food records;
- An in-depth interview will be conducted regarding your data collection tasks including devices and food recording by the research staff.
- Your weight and height will be measured.
- Your blood pressure will be measured.
- You will undergo a full body composition assessment including waist, hip and abdominal height measurements as well as a whole body scan using a DXA machine. These measurements will take place in a private, enclosed room.
- You will also be asked to complete some questionnaires regarding your diet and activity patterns.

DXA machine

What are the benefits and risks of taking part in this study?

There will be no charges made for any of the tests that you undertake. You will receive information regarding your own individual measurements (blood results including blood sugar, cholesterol, blood pressure, % body fat and BMI) and an explanation of the data. In recognition of your participation, you will receive a $100 supermarket or petrol voucher at the end of visit 2. You will also receive a brief report summarising the main findings of the project via mail or e-mail after analysis of the data has been completed. This is also a chance for you to receive some information about your current health status. In addition, this information will help us to provide better recommendations for preventing obesity.

Some people may have a fear of having a blood sample taken or experience discomfort when the blood samples are taken. Occasionally a slight bruise will result. We will take every measure to ensure that you are comfortable and respected. You may also be accompanied by a support person if required.

With the DXA body composition scan you will be exposed to a very low dose of radiation, unlikely to cause any harm. The total dose of radiation will be less than the amount of radiation you are exposed to during a trans-Tasman flight.

Project procedures

Sample Handling and Storage

Samples will be stored in a secure laboratory freezer at the Human Nutrition Research Unit until completion of the study – for a maximum of 10 years. Samples will be analysed by fully-accredited laboratories, either in NZ or overseas, depending on the type of analysis being done. The data will be used only for the purposes of this project and no individual will be identified. Only the investigators and administrators of the study will have access to personal information and this will be kept secure and strictly confidential. Participants will be identified only by a study identification number to ensure anonymity and confidentiality of these samples. Following analysis samples will be destroyed following usual procedures, however, these blood samples can be returned to you upon request.

Data Handling

Results of this project may be published or presented at conferences or seminars. No individual will be able to be identified. At the end of this study the list of participants and their study identification number will be disposed of. Any raw data on which the results of the project depend will be retained in secure storage for 10 years, after which it will be destroyed.

Genetic analysis

Each person has a DNA make-up (their genes) which is different from that of everybody else – except in the case of identical twins. As part of this study we will be performing some DNA-based measures on blood samples that you provide us with. This will allow us to investigate possible links between the gut microbiome and genetic makeup, in particular, links that may relate to diet, body composition, sleep patterns, taste perception and physical activities. This may further our understanding of specific risk factors for obesity. The genetic or DNA-based measures that we will be performing will be research-based and will not have any medical significance for you. Therefore you will not receive the results of these genetic measures as any information gathered will be purely theoretical. As with all other aspects of the study, ID numbers will be used so that your samples are not identifiable and all information will be kept confidential and will only be used for the specific purposes of this study.

Consenting to allow DNA-based measures to be performed on your samples is entirely optional. If you do not wish to participate in this aspect of the research please indicate this on your consent form. You may withdraw your consent to the use of your sample in this research at any time and your samples will be destroyed.

Who is funding the research?

This research is funded by the Health Research Council (HRC) of New Zealand (Grant #15/273).

Participant's rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at any time;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project contacts

If you have any further questions or concerns about the project, either now or in the future, please contact the research team on 09 212 7013 or 021 082 74425 or e-mail promise@massey.ac.nz or website www.massey.ac.nz/promise.

The lead researchers for this study are Professor Bernhard Breier and Associate Professor Rozanne Kruger. If you have any concerns please contact Bernhard [b.breier@massey.ac.nz, (09) 213 6652] or Rozanne [r.kruger@massey.ac.nz, (09) 213 6661]

Committee approval statement

This project has been reviewed and approved by the Health and Disability Ethics Committee, Southern Region, Ethics Reference 16/STH/32. If you have any concerns about the conduct of this research, please contact the Southern Health and Disability Ethics Committee on 0800 438442 or hdecs@moh.govt.nz

Compensation for injury

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Accident Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. If your ACC claim is not accepted you should immediately contact the researchers.

Appendix A.3 Consent form

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE Study

Consent Form for Participants

I have read the Information Sheet* and have had the details of the study explained to me. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet*. This consent form will be held for a period of ten (10) years.

☐ Yes ☐ No

DNA analysis will be performed on parts of the blood samples obtained. Do you consent to parts of your samples being used for DNA analysis?*

☐ Yes ☐ No

The findings of this study may be the basis of further investigations by our team. Would you be happy for us to contact you in future about research related to this study?

☐ Yes ☐ No

*Please see the study Information Sheet for more details

Signature: _____ **Date:** _____

Full Name (printed) _____

Massey Institute of Food Science and Technology – School of Food and Nutrition
Private Bag 102904, North Shore, Auckland 0745, New Zealand T +64 9 213 6653 http://sfn.massey.ac.nz

Appendix A.4 Complete list of PROMIsE study data collected

Measurement	Methods	Outcomes
Body composition profile	Anthropometric measurements (height, weight, hip and weight circumference, Sagittal height)	Linear body measurements – height, waist, hip, saggital height - BMI profiling (height and weight) -
Body composition distribution	Bioelectrical Impedance (BIA)	Weight. Body composition - (fat and lean mass) – total and distribution
	Dual XRay Absorptiometry (DXA)	Body composition - (fat and lean mass). - total - regional
Metabolic heath - biomarkers	Analysis will be conducted by fully accredited laboratory with IANZ to the ISO 15189.	Biomarkers related to metabolic health (lipid profile, glucose control, inflammation, hormonal control)
Metabolic health – blood pressure	-	Blood pressure related to metabolic health
Metabolic health – gene expression	-	MiRNA related to energy expenditure
Diet quality	Food Frequency Questionnaire (FFQ)	Energy intake, macronutrient intake and daily food intake habits.
Dietary behaviour	Three Factor Eating Questionnaire (TFEQ) EAT-26 Questionnaire (EAT)	Restraint, disinhibition and hunger measurement Attitudes to food intake
Dietary variety	Dietary Diversity Questionnaire (DDQ)	Dietary diversity. Food variety.
Physical Activity patterns	Physical activity expenditure	Objective real-life physical activity Physical activity expenditure - sedentary activities - intensity of activity.
	Physical activity diary	Self-reported accelerometer nonwear time Self-reported intentional exercise – time, duration, type, intensity.
Physical Activity behaviour	Recent Physical Activity Questionnaire (RPAQ)	Self-reported physical activities - sedentary activities - time, intensity
Sleep behaviour	-	Objective sleep activity –duration

Measurement	Methods	Outcomes
		- time and -pattern
	Pittsburgh sleep quality index General sleep disturbance scale Munich chronotype questionnaire Berlin questionnaire	Sleep quality Sleep pattern Circadian phase Obstructive sleep apnoea identification
	Sleep diary	Self-reported Actiwatch sleep commencement and wake
Taste perception, intensity, and hedonic preference	-	Sweet and fat taste sensitivity and preference.
Gut microbiome	DNA extraction & sequencing	Ratio of bacterial phyla Fitmicutes to Bacteroidetes Measures of gene counts Prevalence and abundance of methanogens & biological pathways Relative abundance of bacterial species and microbial genes

Appendix A.5 Five-day food record

Participant ID ……. Participant Name …….

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE Study

5 Day Food Record

Thank you very much for taking part in the PROMISE Study. We are extremely grateful for your time, effort and commitment!

If you have any questions, please contact PROMISE staff on:
414 0800 (extn 49013) email: promise@massey.ac.nz

All information in this diary will be treated with the strictest confidence. No one outside the PROMISE study will have access to this data.

Please bring this food diary with you to visit 2 at the Nutrition Laboratory

1

Participant ID ..... Participant Name .....

What to do?

- Record all that you eat and drink on the following dates.

_____
_____
_____
_____
_____

- If possible, record food at the time of eating or just after – try to avoid doing it from memory at the end of the day.
- Include all meals, snacks, and drinks, even tap water.
- Include anything you have added to foods such as sauces, gravies, spreads, dressings, etc.
- Write down any information that might indicate **size or weight** of the food to identify the portion size eaten.
- Use a new line for each food and drink. You can use more than one line for a food or drink. See the examples given.
- Use as many pages of the booklet as you need.

Describing Food and Drink

- Provide as much detail as possible about the type of food eaten. For example, **brand names and varieties / types** of food.

General description	Food record description
Breakfast example – cereal, milk, sugar	1 cup Sanitarium Natural Muesli 1 cup Pam's whole milk 1 tsp Chelsea white sugar
Coffee	1 tsp Gregg's instant coffee 1 x 200ml cup of water 2 Tbsp Meadow fresh light green milk
Pasta	1 cup San Remo whole grain pasta spirals (boiled)
Pie	Big Ben Classic Mince and Cheese Pie (170g)

- Give details of all the **cooking methods** used. For example, fried, grilled, baked, poached, boiled…

General description	Food record description
2 eggs	2 size 7 eggs fried in 2tsp canola oil 2 size 6 eggs (soft boiled)
Fish	100g salmon (no skin) poached in 1 cup of water for 10 minutes

- When using foods that are cooked (eg. pasta, rice, meat, vegetables, etc), please record the **cooked portion** of food.

General description	Food record description
Rice	1 cup cooked Jasmine rice (cooked on stove top)
Meat	90g lean T-bone steak (fat and bone removed)
Vegetables	½ cup cooked mixed vegetables (Wattie's peas, corn, carrots)

- Please specify the **actual amount of food eaten** (eg. for leftovers, foods where there is waste)

General description	Food record description
Apple	1 x 120g Granny Smith apple (peeled, core not eaten – core equated to ¼ of the apple)
Fried chicken drumstick	100g chicken drumstick (100g includes skin and bone); fried in 3 Tbsp Fern leaf semi-soft butter

- **Record recipes** of home prepared dishes where possible and the proportion of the dish you ate. There are blank pages for you to add recipes or additional information.

Participant ID ……. Participant Name …….

Recording the amounts of food you eat

It is important to also record the quantity of each food and drink consumed. This can be done in several ways.

- By using household measures – for example, cups, teaspoons and tablespoons. eg. 1 cup frozen peas, 1 heaped teaspoon of sugar.
- By weight marked on the packages – eg. a 425g tin of baked beans, a 32g cereal bar, 600ml Coke
- For bread – describe the size of the slices of bread (eg. sandwich, medium, toast) – also include brand and variety.
- Using comparisons – eg. Meat equal to the size of a pack of cards, a scoop of ice cream equal to the size of a hen's egg.
- Use the food record instructions provided to help describe portion sizes.

General description	Food record description
Cheese	1 heaped tablespoon of grated cheese 1 slice cheese (8.5 x 2.5 x 2mm) 1 cube cheese, match box size Size 10B grated cheese,

- If you go out for meals, describe the food eaten in as much detail as possible.
- ***Please eat as normally as possible - don't adjust what you would normally eat just because you are keeping a diet record and be honest! Your food record will be identified with a number rather than your name.***

Participant ID ……. Participant Name …….

Example day

Time food was eaten	Complete description of food (food and beverage name, brand, variety, preparation method)	Amount consumed (units, measures, weight)
7:55am	Sanitarium weetbix	2 weetbix
" "	Anchor Blue Top milk	150ml
" "	Chelsea white sugar	2 heaped teaspoons
" "	Orange juice (Citrus Tree with added calcium – nutrition label attached)	1 glass (275 ml)
10.00am	Raw Apple (gala)	Ate all of apple except the core, whole apple was 125g (core was ¼ of whole apple)
12.00pm	Home made pizza (recipe attached)	1 slice (similar size to 1 slice of sandwich bread, 2 Tbsp tomato paste, 4 olives, 2 rashers bacon (fat removed), 1 Tbsp chopped spring onion, 3 Tbsp mozzarella cheese)
1.00pm	Water	500ml plain tap water
3.00pm	Biscuits, chocolate covered Girl Guide biscuits	6 x (standard size)
6.00pm	Lasagne	½ cup cooked mince, 1 cup cooked Budget lasagne shaped pasta , ½ cup Wattie's creamy mushroom and herb pasta sauce, ½ cup mixed vegetables (Pam's carrots, peas and corn), 4 Tbsp grated Edam cheese
6.30pm	Banana cake with chocolate icing (homemade, recipe attached)	1/8 of a cake (22cm diameter, 8 cm high), 2 Tbsp chocolate icing
" "	Tip Top Cookies and Cream ice cream	1 cup (250g)
7.30pm	Coffee	1 tsp Gregg's instant coffee 1 x 300ml cup of water 2 Tbsp Meadow fresh blue top milk 2 tsp sugar

Participant ID ……. Participant Name …….

Date_____ DAY 1 – 5 (continued)

Time food was eaten	Complete description of food (food and beverage name, brand, variety, preparation method)	Amount consumed

Participant ID ..... Participant Name .....

Recipes

Appendix A.6 PROMIsE study food groups

Code	Food group	Food items included
1	Full fat dairy milk	Dark blue, purple, silver top milk, lactose free regular fat milk
2	Low fat dairy milk	Lite and trim milk (green or light blue), lactose free reduced fat milk
3	Milk alternatives, including sweetened	Soy, almond, rice, oat, coconut varieties, milk alternative based drinks from cafés
4	Sweetened dairy milk products	Flavoured milk, fermented or evaporated milk, breakfast drinks, yakult fermented milk drink, hot chocolates, milk-based smoothies, milk-based drinks from cafés, coffee sachets, coffees made with syrup and cream e.g., caramel macchiato
5	Dairy Yoghurt	All types of cow's milk yoghurt. Soy yoghurt under soy products, coconut yoghurt under coconut fats
6	High fat cheese	Cream cheese, goat cheese, haloumi, parmesan, cheddar, processed cheese, blue vein, mascarpone
7	Low fat cheese	Brie, bocconcini, edam, feta, mozzarella, camembert, cottage, ricotta, paneer,
8	Apple, banana, orange	Apple, banana orange
9	Other fruit	All other fruit (fresh, canned, dried)
10	Tomatoes	Fresh, canned, cooked tomatoes
11	Dark yellow vegetables	Carrots, pumpkin, butternut squash
12	Green vegetables	Lettuce, spinach, cabbage, broccoli, watercress, green beans, brussels sprouts, courgette
13	Other non-starchy vegetables	Capsicum, onion, mushrooms, frozen mixed vegetables, beetroot, squash
14	Potatoes / potato dishes (excluding chips)	Potato (boiled, mashed, baked, salad, scalloped, roasted)
15	Starchy vegetables	Kumara, yam, parsnip, turnip, swedes (boiled, mashed, baked) Taro (flesh, roots, stalks), green banana, sweet corn kernels breadfruit, cassava, green banana
16	White breads	Plain white bread, wraps, focaccia, bagel, pita, bread, rewena bread, doughboys, breadcrumbs, including gluten free options, naan (plain)
17	Discretionary snack breads	Crumpets, scone, savoury muffin, plain croissant, pancakes, waffles, iced bun, savoury pin wheels, garlic bread, fruit bread, roti, naan (garlic),
18	Crackers	All crackers made from grains, cream crackers, Cruskits, rice crackers,
19	Whole grain breads	High fibre, wholemeal, wholegrain, including gluten free options
20	Refined grains	White rice, white pasta, noodles (instant, egg, rice), canned spaghetti, cous cous, including gluten free pasta
21	Refined grain mixed dishes	Macaroni and cheese, Carbonaro, white rice salad, two minute noodles
22	Wholegrains	Quinoa, buckwheat, bulgur wheat, brown rice, wholemeal pasta, wholegrain gluten free pasta e.g., brown rice
23	Wholegrain mixed dishes	Tabbouleh, brown rice salad
24	Oats	Porridge, rolled oats, oat bran
25	Sweetened cereal	Sultana bran, light and fruity cereal, chocolate based cereals, Nutrigrain, honey puffs, milo cereals, fruit loops, oat sachets
26	Unsweetened cereals	Weetbix, bran cereals, rice bubbles, cornflakes,
27	Muesli	All muesli and granola

Code	Food group	Food items included
28	Red meats	Beef, lamb, venison, mince, patties,
29	Red meat mixed dishes	Stir fry, curry, stew
30	White meats	Chicken, pork, turkey,
31	White meat mixed dishes	Stir fry, curry, stew
32	Processed meats	Corned beef (canned), corned silverside, smoked chicken, smoked hock and salami, ham, sausages, frankfurters, bacon, chorizo, luncheon meat
33	Fish and seafood	Canned and fresh
34	Fish and seafood mixed dishes	Ota ika, curry, stew
35	Eggs	Whole eggs (boiled, poached, fried, plain omelette)
36	Egg mixed dishes	Quiche, frittata, omelette with filling, egg and banana pancake
37	Legumes, legume mixed dishes	Baked beans, black beans, dahl, canned or dried legumes, hummus, and legume based vegetarian meals and products
38	Soy products	Edamame beans tofu, soy yoghurts
39	Peanut butter and peanuts	Peanut butter and peanuts
40	Nuts and seeds	Brazil nuts, walnuts, almond, cashew, pistachio, chia, linseed, pumpkin, sesame
41	Fats	Cream, sour cream, reduced cream, butter, lard, dripping, ghee,
42	Coconut fats and products	Coconut oil, cream, milk, desiccated, yoghurt, fresh
43	Oil, oil-based dressings	Avocado (whole fruit), canola, sunflower, olive, vegetable oil, cooking spray, salad dressings (French/Italian)
44	Margarine	All margarines
45	Creamy dressings and sauces	Creamy readymade meal-based sauces, dips, mayonnaise, aioli, tartare sauce, white sauce, cheese sauce
46	Savoury sauces and condiments	Curry pastes, herb and spices, stock, vinegar, gravy and garlic sauce, instant and packet soups, oil based condiments such as sundried tomatoes / olives in oil, pesto, tomato, barbeque, mint, soy, gravy, mustard, chutney, miso, pasta sauce, tomato paste, sweet chilli sauce
47	Sweet spreads	Jam, honey, marmalade, syrup (maple, golden), Nutella, chocolate peanut butter, chocolate butter
48	Savoury spreads	Vegemite, marmite
49	Cake and biscuits	Slices, cakes, loaves, muffins, biscuits, doughnuts, sweet pies, pastries, tarts
50	Puddings and other desserts	Including all milk alternative ice creams, ice cream, custard, milk-based puddings e.g., rice, instant, semolina, pavlova, sticky date, fruit pies and crumbles, jelly, ice blocks
51	Sweet snack foods	Fruit and nut mixes, bliss balls, chocolate, lollies, muesli bars,
52	Savoury snack foods	Popcorn, potato crisps, corn chips, twisties, bujha mix,
53	Crumbed and deep fried	Hot chips/fries, hash browns, and packaged home baked chips, wontons, paraoa bread, schnitzel, nuggets, crumbed fish
54	Fast-food (burgers)	Pies, dumpling, burgers, pizzas, curries, noodle-based dishes, Nando's chicken, egg fu yong
55	Fast food (salads & sushi)	Salads, sandwiches, wraps, sushi, vegetable-based stir fry
56	Fruit and vegetable juice	Fruit and/or vegetable juice, fruit and/or vegetable smoothies

Code	Food group	Food items included
57	Fruit drinks, soft drinks, and other beverages	All cordials, flavoured water, sports drinks, soft drinks, fruit drinks, iced tea, energy drinks
58	Diet drinks	All exclusively artificially sweetened beverages
59	Tea	Black, green, herbal, chai, kombucha
60	Coffee	instant, brewed, espresso, pre-mixed sachet, filter, cold brew
61	Beer	Beer (standard and low alcohol)
62	Wine	Wine (stanard and low alcohol)
63	Water	Water (unflavoured, soda, tap)
64	Spirits and other alcoholic beverage	Cider, spirits, RTDs, sherry, port, liqueurs
65	Sugar added to food and drink	All sugar added to food and drink
66	Shakes & Protein Powders	Protein powders, cacao powder, baking powder, baking soda, weight loss shakes, psyllium husk powder, cornflour, vanilla essence, natural sweeteners, yeast, flours

Appendix A.7 Munich Chronotype Questionnaire

1. On days when you are scheduled to work, study, care for others or have other regular commitments:

a. I have to get up at:

(Please specify am/pm)

[HH:MM AM/PM]

b. To wake up I need:

[MINUTES]

c. I regularly wake up:

O Before the alarm

O With the alarm

O Don't use an alarm

d. I am fully awake from:

(Please specify am/pm)

[HH:MM AM/PM]

e. I have an energy dip at:

(Please specify am/pm)

[HH:MM AM/PM]

f. On nights before scheduled (e.g. work) days, I go to bed at:

(Please specify am/pm)

[HH:MM AM/PM]

g. To fall asleep when I go to bed takes me:

[MINUTES]

h. If I get the chance, I like to take a nap:

Yes O No O

i. If **yes,** I like to nap at:

(Please specify am/pm)

[HH:MM AM/PM]

for:

[MINUTES]

2. Imagine having free days (days when you are **not** scheduled to work, study, care for others or have no other regular commitments). On free days:

a. Ideally, I would sleep in until:

(Please specify am/pm)

[HH:MM AM/PM]

b. I normally wake up at:

(Please specify am/pm)

[HH:MM AM/PM]

c. If I wake up at around the normal (scheduled/work day) alarm time, I try to get back to sleep:

Yes O No O

d. If I get back to sleep, I sleep for another:

[MINUTES]

e. I am fully awake from:

(Please specify am/pm)

[HH:MM AM/PM]

f. I have an energy dip at around:

(Please specify am/pm)

[HH:MM AM/PM]

g. On nights before free days, I go to bed at:

(Please specify am/pm)

[HH:MM AM/PM]

h. To fall asleep when I go to bed takes me:

[MINUTES]

i. If I get the chance, I like to take a nap:

Yes O No O

j: If **yes**, I like to nap at:

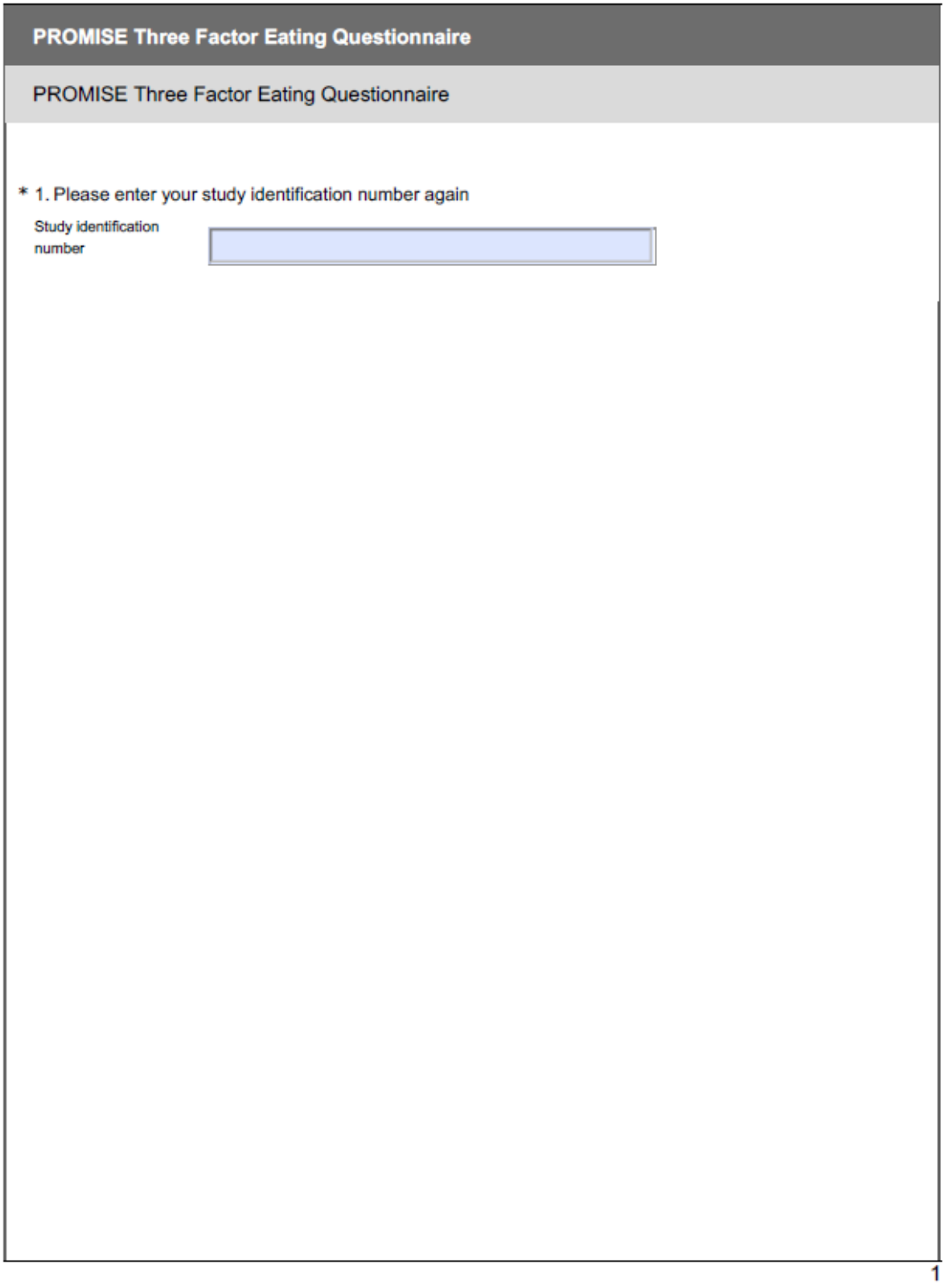
(Please specify am/pm)

[HH:MM AM/PM]

for:

[MINUTES]

Appendix A.8 Three Factor Eating Behaviour Questionnaire



PROMISE Three Factor Eating Questionnaire

PROMISE Three Factor Eating Questionnaire

* 1. Please enter your study identification number again

Study identification number

1

PROMISE Three Factor Eating Questionnaire

PROMISE Three Factor Eating Questionnaire

Please answer each question by choosing the the appropriate answer (True or False)

2. When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal

- ◯ True
- ◯ False

3. I usually eat too much at social occasions, like parties and picnics

- ◯ True
- ◯ False

4. I am usually so hungry that I eat more than three times a day

- ◯ True
- ◯ False

5. When I have eaten my quota of calories, I am usually good about not eating any more

- ◯ True
- ◯ False

6. Dieting is so hard for me because I just get too hungry

- ◯ True
- ◯ False

7. I deliberately take small helpings as a means of controlling my weight

- ◯ True
- ◯ False

8. Sometimes things just taste so good that I keep on eating even when I am no longer hungry

- ◯ True
- ◯ False

9. Since I am often hungry, I sometimes wish that while eating, an expert would tell me that I have had enough or that I can have something more to eat

- ◯ True
- ◯ False

10. When I feel anxious, I find myself eating

- ◯ True
- ◯ False

11. Life is too short to worry about dieting

- ◯ True
- ◯ False

12. Since my weight goes up and down, I have gone on reducing diets more than once

- ◯ True
- ◯ False

13. I often feel so hungry that I just have to eat someting

- ◯ True
- ◯ False

14. When I am with someone who is overeating, I usually overeat too

- ◯ True
- ◯ False

15. I have a pretty good idea of the number of calories in common food

- ◯ True
- ◯ False

16. Sometimes when I start eating, I just can't seem to stop

- ◯ True
- ◯ False

17. It is not difficult for me to leave something on my plate

- True
- False

18. At certain times of the day, I get hungry because I have gotten used to eating someting then

- True
- False

19. While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it

- True
- False

20. Being with someone who is eating often makes me hungry enough to eat also

- True
- False

21. When I feel blue, I often overeat

- True
- False

22. I enjoy eating too much to spoil it by counting calories or watching my weight

- True
- False

23. When I see a real delicacy, I often get so hungry that I have to eat right away

- True
- False

24. I often stop eating when I am not really full as a conscious means of limiting the amount that I eat

- True
- False

25. I get so hungry that my stomach often seems like a bottomless pit

- ◯ True
- ◯ False

26. My weight has hardly changed at all in the last ten years

- ◯ True
- ◯ False

27. I am always hungry so it is hard for me to stop eating before I finish the food on my plate

- ◯ True
- ◯ False

28. When I feel lonely, I console myself by eating

- ◯ True
- ◯ False

29. I consciously hold back at meals in order not to gain weight

- ◯ True
- ◯ False

30. I sometimes get very hungry late in the evening or at night

- ◯ True
- ◯ False

31. I eat anything I want, any time I want

- ◯ True
- ◯ False

32. Without even thinking about it, I take a long time to eat

- ◯ True
- ◯ False

33. I count calories as a conscious means of controlling my weight

- ◯ True
- ◯ False

34. I do not eat some foods because they make me fat

- ◯ True
- ◯ False

35. I am always hungry enough to eat at any time

- ◯ True
- ◯ False

36. I pay a great deal of attention to changes in my figure

- ◯ True
- ◯ False

37. While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods

- ◯ True
- ◯ False

PROMISE Three Factor Eating Questionnaire

Please answer the following questions by choosing the response that is appropriate to you.

38. How often are you dieting in a conscious effort to control your weight?

- ◯ Rarely
- ◯ Sometimes
- ◯ Usually
- ◯ Always

39. Would a weight fluctuation of 2.5 kg (5 lbs) affect the way you live your life?

- ◯ Not at all
- ◯ Slightly
- ◯ Moderately
- ◯ Very much

40. How often do you feel hungry?

- ◯ Only at mealtimes
- ◯ Sometimes between meals
- ◯ Often between meals
- ◯ Almost always

41. Do your feelings of guilt about overeating help you to control your food intake?

- ◯ Never
- ◯ Rarely
- ◯ Often
- ◯ Always

42. How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours?

- ◯ Easy
- ◯ Slightly difficult
- ◯ Moderately difficult
- ◯ Very difficult

43. How conscious are you of what you are eating?

- ◯ Not at all
- ◯ Slightly
- ◯ Moderately
- ◯ Exremely

44. How frequently do you avoid 'stocking up' on tempting foods?

- ◯ Almost never
- ◯ Seldom
- ◯ Usually
- ◯ Almost always

45. How likely are you to shop for low calorie foods?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very likely

46. Do you eat sensibly in front of others and splurge alone?

- ◯ Never
- ◯ Rarely
- ◯ Often
- ◯ Always

47. How likely are you to consciously eat slowly in order to cut down on how much you eat?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very Likely

48. How frequently do you skip dessert because you are no longer hungry?

- ◯ Almost never
- ◯ Seldom
- ◯ At least once a week
- ◯ Almost every day

49. How likely are you to consciously eat less than you want?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very likely

50. Do you go on eating binges though you are not hungry?

- ◯ Never
- ◯ Rarely
- ◯ Sometimes
- ◯ At least once a week

51. On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never 'giving in'), what number would you give yourself?. Choose the answer which best describes you.

- ◯ 0. Eat whatever you want, whenever you want it
- ◯ 1. Usually eat whatever you want, whenever you want it
- ◯ 2. Often eat whatever you want, whenever you want it
- ◯ 3. Often limit food intake, but often 'give in'
- ◯ 4. Usually limit food intake, rarely 'give in'
- ◯ 5. Constantly limiting food intake, never 'giving in'

52. To what extent does this statement describe your eating behaviour ?
'I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow.'

- ◯ Not like me
- ◯ A little like me
- ◯ Pretty good description of me
- ◯ Describes me perfectly

10

Appendix A.9 Eating Attitudes Test-26

Eating Attitudes Test (EAT-26)©

Instructions: This is a screening measure to help you determine whether you might have an eating disorder that needs professional attention. This screening measure is not designed to make a diagnosis of an eating disorder or take the place of a professional consultation. Please fill out the below form as accurately, honestly and completely as possible. There are no right or wrong answers. All of your responses are confidential.

Part A: Complete the following questions:

1) Birth Date	Month:		Day:	Year:	2) Gender:	Male	Female
3) Height	Feet :		Inches:			□	□
4) Current Weight (lbs.):			5) Highest Weight (excluding pregnancy):				
6) Lowest Adult Weight:			7: Ideal Weight:				

Part B: Check a response for each of the following statements:		Always	Usually	Often	Some times	Rarely	Never
1.	Am terrified about being overweight.	□	□	□	□	□	□
2.	Avoid eating when I am hungry.	□	□	□	□	□	□
3.	Find myself preoccupied with food.	□	□	□	□	□	□
4.	Have gone on eating binges where I feel that I may not be able to stop.	□	□	□	□	□	□
5.	Cut my food into small pieces.	□	□	□	□	□	□
6.	Aware of the calorie content of foods that I eat.	□	□	□	□	□	□
7.	Particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)	□	□	□	□	□	□
8.	Feel that others would prefer if I ate more.	□	□	□	□	□	□
9.	Vomit after I have eaten.	□	□	□	□	□	□
10.	Feel extremely guilty after eating.	□	□	□	□	□	□
11.	Am preoccupied with a desire to be thinner.	□	□	□	□	□	□
12.	Think about burning up calories when I exercise.	□	□	□	□	□	□
13.	Other people think that I am too thin.	□	□	□	□	□	□
14.	Am preoccupied with the thought of having fat on my body.	□	□	□	□	□	□
15.	Take longer than others to eat my meals.	□	□	□	□	□	□
16.	Avoid foods with sugar in them.	□	□	□	□	□	□
17.	Eat diet foods.	□	□	□	□	□	□
18.	Feel that food controls my life.	□	□	□	□	□	□
19.	Display self-control around food.	□	□	□	□	□	□
20.	Feel that others pressure me to eat.	□	□	□	□	□	□
21.	Give too much time and thought to food.	□	□	□	□	□	□
22.	Feel uncomfortable after eating sweets.	□	□	□	□	□	□
23.	Engage in dieting behavior.	□	□	□	□	□	□
24.	Like my stomach to be empty.	□	□	□	□	□	□
25.	Have the impulse to vomit after meals.	□	□	□	□	□	□
26.	Enjoy trying new rich foods.	□	□	□	□	□	□

Part C: Behavioral Questions: In the past 6 months have you:		Never	Once a month or less	2-3 times a month	Once a week	2-6 times a week	Once a day or more
A	Gone on eating binges where you feel that you may not be able to stop? *	□	□	□	□	□	□
B	Ever made yourself sick (vomited) to control your weight or shape?	□	□	□	□	□	□
C	Ever used laxatives, diet pills or diuretics (water pills) to control your weight or shape?	□	□	□	□	□	□
D	Exercised more than 60 minutes a day to lose or to control your weight?	□	□	□	□	□	□
E	Lost 20 pounds or more in the past 6 months	Yes □		No □			

* Defined as eating much more than most people would under the same circumstances and feeling that eating is out of control

Appendix A.10 Characteristics of study population according to ethnic groups

The analysis was conducted for the whole study population (n = 304) before exclusion of the night shift workers (n = 17), stratified by ethnic groups.

Variable	**NZ Europeans (n = 162)**	**Pacific women (n = 142)**	***P*-value**
MCTQ score (h:min)	3.7 [3.5, 3.8]	5.4 [5.1, 5.6]	**0.00**[r]
MT (<3) (%)	29 (9.5)	7 (2.3)	**0.00**
IT (3 -5) (%)	118 (38.8)	45 (14.8)	
ET (>5) (%)	15 (4.9)	90 (29.6)	
Normal BMI (%)	48.8	25.4	**0.00**
High BMI (%)	51.2	74.6	
Normal BF%	54.3	39.4	0.11
High BF%	45.7	60.6	

(MT) Morning types; (IT) Intermedite types, (ET) Evening types; (BMI) Body mass index; (BF%) Body fat percentage,
Values are reported as Median (25^{th} ,75^{th} percentiles), means ±SD, or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale,
Chi-square test for categorical data, [r]Independent T-test for comparing two continuous, normally distributed data.

Appendix B.1 PRISMA checklist

Section/topic	#	Checklist item	Reported on page # of document
TITLE: Associations between Chronotype and Obesity Outcomes and Effects of Dietary Intake and Eating Behaviour in Healthy Adults – A Systematic Review			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	83
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	83
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	85 - 87
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	88- 89 & Appendix B.3
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	Published at PROSPERO 87
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	88 - 89 & Appendix B.3
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	87 - 88
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Appendix B.2
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	88 - 89, 91
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	89 - 90

Section/topic	#	Checklist item	Reported on page # of document
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	88 - 89 & Appendix B.3
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	89 & Appendix B.4-6
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	88 - 90
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	88 - 90
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	89 & Appendix B.4-6
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	90
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	91 - 92
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	91 – 92 & Table 4.1
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	89 & Appendix B.4-6
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	91 – 147 & Table 4.1 – 4.4
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	NA
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	89 & Appendix B.4-6
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	91 – 147 & Table 4.1 – 4.4
DISCUSSION			

Section/topic	#	Checklist item	Reported on page # of document
Summary of evidence	24	Summarise the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	148 -152
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	153
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	153
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	Title pg.

Appendix B.2 Systematic review search term & syntax

Search terms & syntax
(adult OR adults) AND (chronotype OR chrono OR "circadian rhythm*" OR "circadian clock*" OR "clock system" OR circadian OR "timing system" OR circadian OR "morning-type" OR "evening-type" OR morningness OR eveningness OR "chrono-nutrition" OR "intermediate-type" OR "neither-type" OR "social jetlag") AND (anthropometry OR "body composition" OR body fat distribution OR adiposity OR "body mass index" OR "normal body weight" OR "ideal body weight" OR "ideal body mass" OR obese OR obesity OR "morbid obesity" OR overweight) AND ("dietary intake" OR "nutrient intake" OR "feeding behaviour" OR "feeding behavior" OR "eating behavior" OR "eating behaviour" OR "dietary behaviour" OR "dietary behavior" OR "feeding pattern*" OR "habit* food" OR "eating habit*" OR "dietary habit*" OR "food intake" OR "meal timing" OR "food intake timing" OR "food preference" OR macronutrient* OR nutrient OR "calorie intake" OR "energy intake" OR micronutrient* OR biomarkers)

Appendix B.3 PICOS table

Review question	**Is body composition, dietary intake, eating behaviour and biomarker outcomes in healthy adults dependent on chronotype?**
Population	Inclusion criteria: Healthy adults 18 years and older. Exclusion criteria: Individuals <18 years; pregnant and lactating women, night shift workers, diagnosed acute, pre-existing and chronic conditions (e.g., eating disorders, trauma, surgical or hospitalized patients, depression, mental illness, sleep disorders, diabetes and hypertension).
Intervention	Classification according to chronotype using validated chronotype questionnaires such as the Munich Chronotypes Questionnaire and the Morning-Eveningness Questionnaire as well as studies using mid-point of sleep to define chronotype.
Comparator	Body composition profile (BMI, body fat profiles, waist- and hip circumference, etc.).
Outcomes	Must include one of the following: Dietary intake (total and distribution of macro- and micronutrient intakes as well as food group intakes), and/or Eating behaviours/habits (meal timing, meal frequency, binge eating, perceived hunger, eating restraint, eating control, emotional eating, eating context e.g., meal intake while watching television, emotional eating and other relevant behavious) and/or Biomarkers (such as blood glucose, glycated hemoglobulin levels, lipid profiles, liver function, endocrine regulators, and other relevant biomarkers and hormone levels).
Study design	All relevant study design except for conference proceedings, editorial letters, review articles and pharmacological studies.

Appendix B.4 Joanna Briggs institute checklists for analytical cross-sectional studies

Reference	Criteria for sample inclusion clearly defined?	Study subjects and setting described in detail?	Exposure measured in a valid and reliable way?	Objective, standard criteria used for measurement of the condition?	Confounding factors identified?	Strategies to deal with confounding factors stated?	Outcomes measured in a valid and reliable way?	Appropriate statistical analysis used?	Score /8
Sato-Mito et al. 2011	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Vera et al.2018	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Najem et al. 2020	Yes	Yes	Yes	Yes	Unclear	Unclear	Yes	Yes	6
Lázár et al. 2012	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Yoshizaki et al. 2018	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Silva et al. 2016	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Lai & Say. 2013	Unclear	Yes	Yes	Yes	Yes	Yes	Yes	Yes	7
Lucassen et al. 2013	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Mota et al. 2016	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Zerón-Rugerio et al. 2019	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Maukonen et al. 2017	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Maukonen et al. 2016	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Teixeira et al. 2018	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Li et al. 2018	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
De Amicis et al. 2020	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Baron et al. 2011	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Baron et al. 2013	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Beaulieu et al. 2020	Yes	Unclear	Yes	Yes	Unclear	Unclear	Yes	Yes	5
Muscogiuri et al. 2020	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8
Zerón-Rugerio et al. 2020	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	8

Appendix B.5 Joanna Briggs institute checklists for cohort studies

Reference	Groups similar/ from same population?	Exposures measured similarly to assign people to both exposed & unexposed groups?	Exposure measured in a valid and reliable way?	Confounding factors identified?	Strategies to deal with confounding factors stated?	Participants free of the outcome at start of study (or at the moment of exposure)?	Outcomes measured in a valid and reliable way?	Follow up time reported & sufficient for outcomes to occur?	Follow up complete, and if not, were the reasons to loss to follow p described and explored?	Strategies to address incomplete follow up utilised?	Appropriate statistical analysis used?	Score /11
Xiao et al. 2019	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	11
Maukonen et al. 2019	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	11
Culnan et al. 2013	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Unclear	Yes	10

Appendix B.6 Joanna Briggs institute checklists for randomised controlled trials

Reference	True randomization used for assignment of participants to treatment groups?	Allocation to treatment groups concealed	Treatment groups similar at baseline	Participants blind to treatment assignment	Those delivering treatment blind to treatment assignment	Outcome assessors blind to treatment assignment	Treatment groups treated identically other than the intervention	Follow up complete and if not, were differences between groups in terms of their follow up adequately described and analysed	Participants analysed in the groups to which they were randomised	Outcomes measured in the same way for treatment groups	Were outcomes measured in a reliable way	Appropriate statistical analysis used	Appropriate trial design, and any deviations from the standard RCT design (individual randomisation, parallel groups) accounted for in conduct and analysis of trial	Score /13
Muñoz, 2020	Yes	Yes	Yes	Yes	Yes	Unsure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	12

Appendix B.7 Chronotype assessment methods

Reference	Questionnaire used and method of chronotype classification
Xiao et al. 2019	MCTQ MT was defined when midsleep was earlier than median midsleep i.e., before 03:04 AM. ET was defined for median midsleep later than 03:04 AM.
Sato-Mito et al. 2011	MEQ Participants reported bedtimes and rise times in a lifestyle questionnaire as the time when they usually went to bed on weekdays and when they arose in the morning. The midpoint of sleep was calculated as the halfway point between bedtime and rise time. Participants were assigned to quintiles according to their midpoint of sleep, from the earliest Q1: (2:32 ± 0:23 h: min), (Quintile 2: (3:10 ± 0:08 h: min, (Quintile 3: 3:37 ± 0:07 h: min), (Quintile 4: 4:11 ± 0:13 h:min) to the latest quintile (Q5: 5:31 ± 0:55 h: min).
Vera et al. 2018	MEQ The score was divided into "more evening" and "more morning" type based on the median MEQ score of the total population (i.e., a score <53: more evening; a score ≥53: more morning).
Najem et al. 2020	MEQ The following MEQ scores were used to assign participants to different chronotype groups: Definite ET: ≤ 30 Moderate ET: 31 - 41 IT: 42 - 58 Moderate MT: 59 - 69 Definite MT: 70 - 86
Lázár et al. 2012	MEQ The total score of the MEQ and the single question from the Munich Chronotype Questionnaire referring to self-assessed chronotype were used.
Yoshizaki et al. 2018	MEQ Participants were divided into tertials according to the MEQ score: Moderate ET: 1st third: 34 – 53 Intermediate: 2nd third: 54 – 59 MT: 3rd third: 60 – 76
Silva et al. 2016	MEQ Chronotype was defined using midsleep time on free days and a correction for sleep duration on work and free days by using the difference between average sleep duration on weekends and on weekdays.
Lai & Say, 2013	MEQ Participants were divided into chronotypes according to the MEQ score: IT: 42 – 58 Definitely ET: 16 - 30 Moderately ET: 31 - 41
Muñoz, 2020	MEQ Participants with a low MEQ score (16-51 points), were assigned to ET, and those with high scores (52-86 points) were assigned to MT.

Reference	Questionnaire used and method of chronotype classification
Lucassen et al. 2013	MEQ Participants were divided into chronotypes according to the MEQ score where MT includes moderate and definite MT and vice versa for ET: Score range: 16–86 MT (50–86) ET (16–49)
Mota et al. 2016	MEQ Participants were classified as ET (16–41), IT (42–58) or MT (59–86).
Zerón-Rugerio et al. 2019	MEQ Score range: 10–86. Participants were classified as IT (42–58), MT (>58) or ET (<42).
Maukonen et al. 2019	MEQ (short version) The sum MEQ of the final short version of the MEQ score ranged from 6 to 27. Participants were classified into ET (6–12), IT (13–18) and MT (19–27).
Maukonen et al. 2017	MEQ (short version) Participants were classified into ET (6–12), IT (13–18) and MT (19–27).
Maukonen et al. 2016	MEQ (short version) The final MEQ score varied from 5 (extreme ET) to 27 (extreme MT). Tertials were used for analysis (MT, IT and ET).
Teixeira et al. 2018	MEQ Participants were assigned to ET (coefficient: 16–41), IT (coefficient: 42–58) or MT (coefficient: 59–86).
Li et al. 2018	MEQ The MEQ scores range from 59 to 86, the intermediate scores range from 42 to 58, and the ET scores range from 16 to 41.
De Amicis et al., 2020	MEQ (short version) The final MEQ score ranged from < 12 (extreme ET) to > 17 (extreme MT). Intermediate scores were associated with IT (12–17 points).
Culnan et al. 2013 (72)	MEQ (short version) Participants were classified into ET (6–12), IT (13–18) and MT (19–27).
Baron et al., 2011 and Baron et al. 2013	MEQ The MEQ scores range from 16 to 86, with higher scores indicating greater preference for morning. Participants were dichotomized as IT if their midpoint of sleep was between 1:00 am to 5:29 am, and as ET if midpoint of sleep was 5:30 am or later, which was past the 50th percentile of sleep times in the population (4:00 am).
Beaulieu et al. 2020	MEQ Chronotype classification was determined by MEQ score median split, stratified for sex (calculated separately for each sex). MEQ scores range between 16–86.
Muscogiuri et al. 2020	(MEQ) (short version) The MEQ scores ranged from 16 to 86. Participants were classified as being a MT (59–86), IT (42–58), or ET (16–41) based on their MEQ score.
Zerón-Rugerio et al. 2020	(MEQ) Participants completed a 6-day sleep diary on consecutive days (including 3 weekdays and 2 weekend days) in which they recorded bedtime and wakeup timing. From this data, the midpoints of sleep were calculated. Sleep timing was classified using the median splits of the time in which each subject went to bed and woke up during the week. Bedtime was classified as follows: "Early-bedtime" (<23:48 h) and "Late-bedtime" (≥23:48 h). Then, for each bedtime group, the median splits of wakeup timing were used.

Reference	Questionnaire used and method of chronotype classification
	Early-bedtime Participants were divided into “Early-rise” (wakeup time <7:12 h) and “Late-rise” (wakeup time ≥ 7:12 h). “Late-bedtime” Participants were divided into “Early-rise” (wakeup time <7:52 h) and “Late-rise” (wakeup time ≥ 7:52 h). Consequently, four sleep timing categories were defined: early-bedtime/early-rise (EE), early-bedtime/late-rise (EL), late-bedtime/early-rise (LE), and late-bedtime/late-rise (LL). We assigned LL to ET and EE to MT and EL and LE to IT in the chapter.

Appendix C.1 Timing of eating occasions & overnight fasting period according to chronotype

Variable‡	MT-IT (n = 190)	Evening Type (n = 97)	*P*-value ᵣ
First eating occasion (hh:mm)	08:41 [08:28, 08:55]	10:54 [10:29, 11:20]	<**0.001**
Duration between sleep onset and first eating occasion (hrs)	14.3 (13.5, 15.0)	14.0 (12.8, 15.3)	0.29 ᶻ
Last eating occasion clock time (hh:mm)	19:23 ± 01:03	20:01 ± 01:31	<**0.001**
Duration between last eating occasion and wake time (hrs)	11.6 ± 1.29	10.0 ± 1.68	<**0.001**

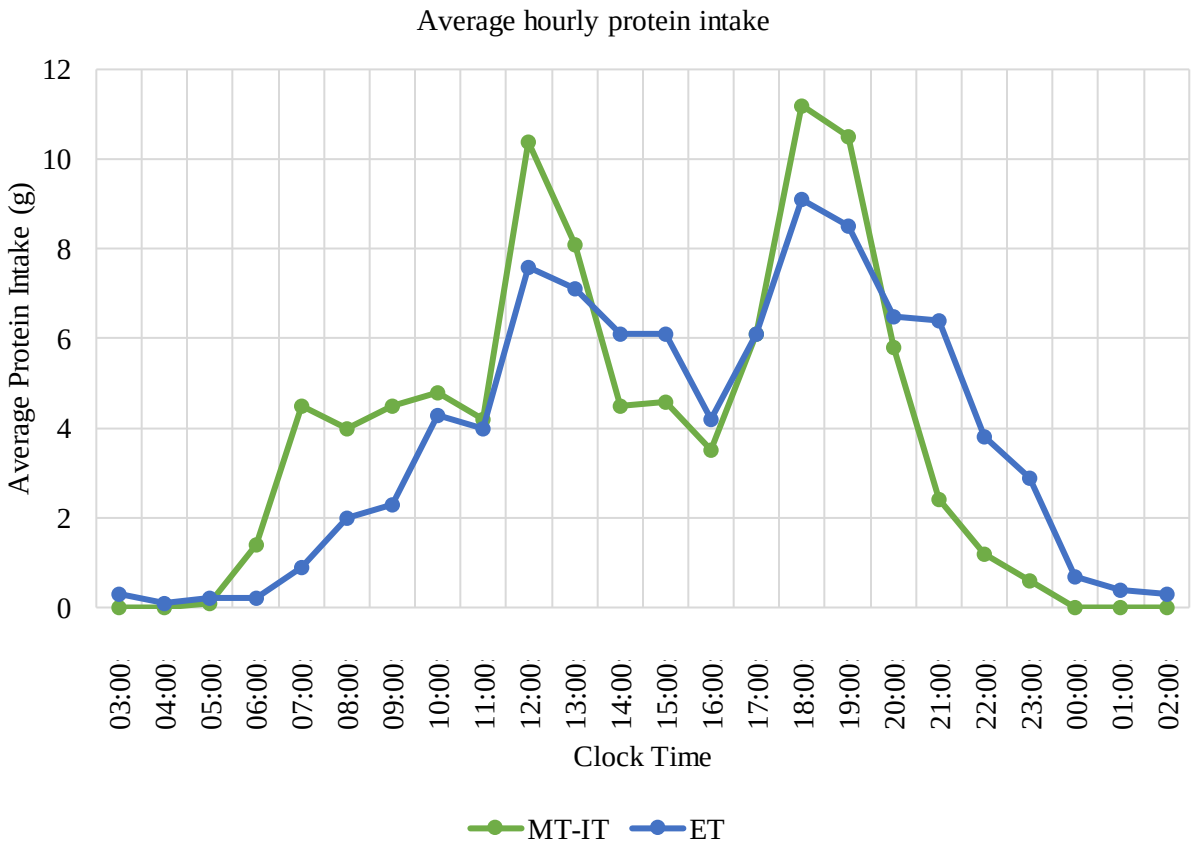
(MT-IT) Morning- and intermediate types combined group; Values are reported as Median (25th ,75th percentiles), means ±SD, or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale. ᶻ*Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data;* ᵣ*Independent T-test for comparing two continuous, normally distributed data.*

Appendix C.2 Sleep characteristics

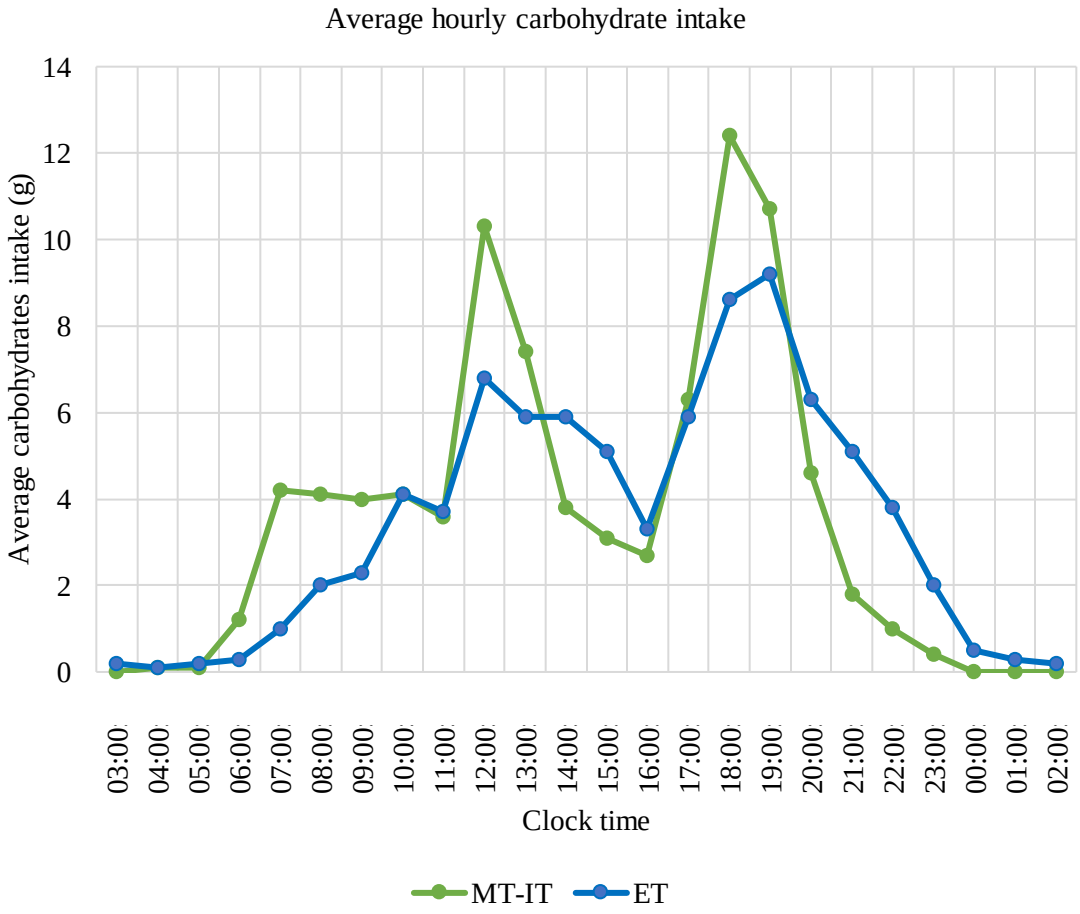
Variable	Morning type (n = 35)	Intermediate type (n = 155)	Evening type (n = 97)	*P*-value [†]	MT-IT[†] (n = 190)	*P*-value[z]
Sleep characteristics						
Average Wake Time (hh:mm)	06:48 (06:24, 07:12)	08:00 (07:30, 08:30) [a]	09:48 (09:00, 10:54) [a, b]	**<0.01**	07:48 (07:18, 08:30)	**<0.01**
Wake Time Free days (hh:mm)	07:30 (07:00, 08:00)	08:30 (08:00, 09:00) [a]	10:30 (10:00, 11:30) [a, b]	**<0.01**	08:00 (08:00, 09:00)	**<0.01**
Wake Time Workdays (hh:mm)	06:00 (05:45, 06:30)	07:30 (07:00, 08:00) [a]	09:23 (08:00, 10:30) [a, b]	**<0.01**	07:15 (06:30, 08:00)	**<0.01**
Average Sleep Onset (hh:mm)	22:18 (21:54, 22:30)	23:00 (22:42, 23:42) [a]	00:48 (00:06, 01:36) [a, b]	**<0.01**	22:54 (22:30, 23:30)	**<0.01**
Sleep Onset Free days (hh:mm)	22:15 (22:00, 22:40)	23:30 (23:04, 00:04) [a]	01:52 (01:10, 02:30) [a, b]	**<0.01**	23:18 (22:43, 00:00)	**<0.01**
Sleep Onset Workdays (hh:mm)	22:04 (21:25, 22:34)	22:37 (22:07, 23:04) [a]	23:42 (22:54, 00:42) [a, b]	**<0.01**	22:30 (22:00, 23:00)	**<0.01**
Social Jetlag	0.77 (0.29, 1.21)	0.92 (0.50, 1.25)	1.75 (1.00, 2.47) [a, b]	**<0.01**	0.88 (0.50, 1.25)	**<0.01**
Average Sleep Duration (hrs)	8.5 (8.1, 9.3)	8.9 (8.4, 9.5)	8.8 (8.1, 9.9)	0.28	8.9 (8.3, 9.5)	0.63

Values are reported as means ±SD, [†]Kruskal- Wallis with multiple comparison rank tests were used to compare between three groups (MT, IT, ET) of continuous, non-normally distributed data [a] compared against MT, [b] compared against IT, 0.05 / 3 = 0.0167, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, [z]Mann-Whitney Test for comparing two groups (ET, MT-IT) continuous non-normally distributed data.

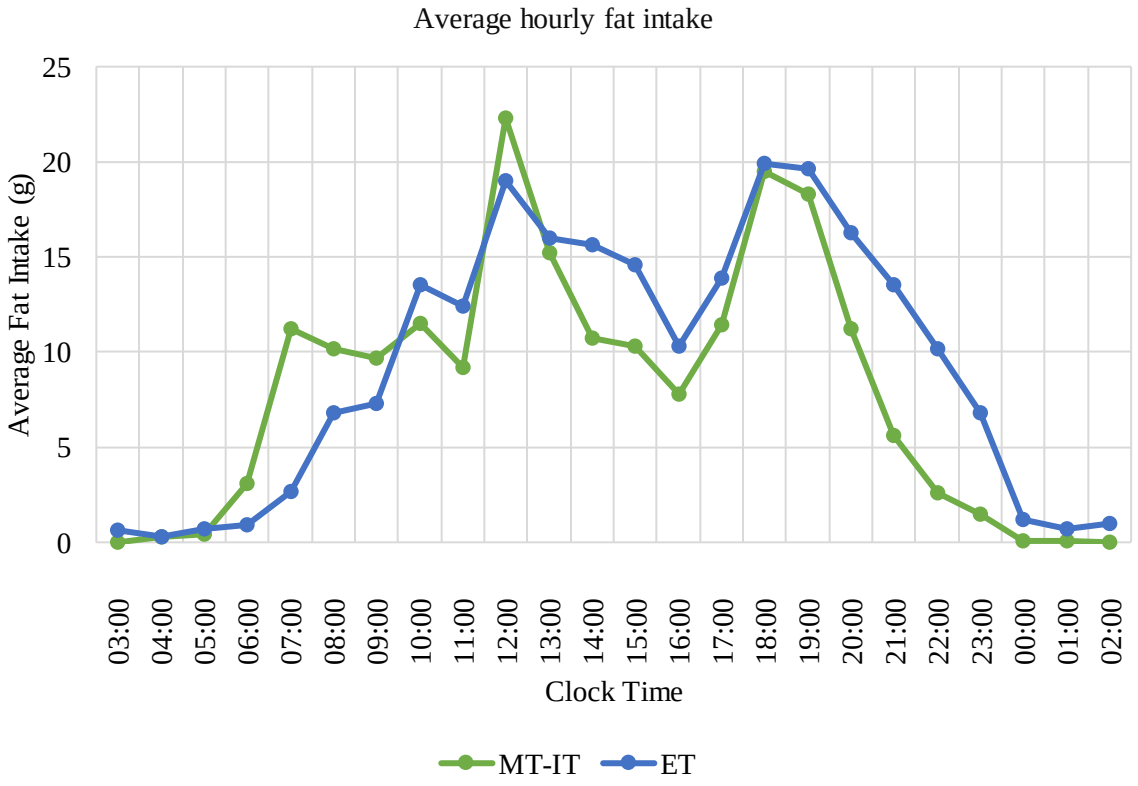
Appendix C.3 Average hourly protein intake



Appendix C.4 Average hourly carbohydrate intake



Appendix C.5 Average hourly fat intake



Appendix C.6 Dietary intake according to dietary windows 2 and 3

Dietary intake	Morning & intermediate type (n = 190)	Evening type (n = 97)	*P*-value [r]	Morning & intermediate type (n = 190)	Evening type (n = 97)	*P*-value [r]
Dietary window	**Dietary intake window 2**			**Dietary intake window 3**		
Energy (kJ)	2929 ± 986	2862 ± 1237	0.65	3257 ± 1049	3225 ± 1332	0.84
Protein (g)	29.2 ± 11.5		0.05	35.2 ± 11.3	32.2 ± 13.6	0.06
Carbohydrate (g)	69.0 ± 30.0	76.2 ± 36.8	0.09	67.2 ± 28.4	78.3 ± 38.4	0.01
Fat (g)	32.1 ± 13.8	28.9 ± 14.0	0.07	35.9 ± 14.5	34.0 ± 15.6	0.30
Sugar (g)	12 ± 6	11 ± 6	0.28	27 ± 14	30 ± 20	0.23
Fibre (g)	8 ± 4	6 ± 3	**<0.01**	9 ± 4	7 ± 4	**<0.01**
MUFA (g)	12 ± 5	11 ± 6	0.09	14 ± 6	13 ± 6	0.31
PUFA (g)	5 ± 3	4 ± 2	**<0.01**	5 ± 3	4 ± 2	0.15
SFA (g)	12 ± 6	11 ± 6	0.32	14 ± 6	13 ± 7	0.51
Cholesterol (mg)	116 ± 85	104 ± 92	0.27	121 ± 65	1025 ± 57	**0.01**
Caffeine (mg)	51 ± 58	30 ± 37	**<0.01**	20 ± 32	20 ± 24	0.80
Alcohol (g)	0 ± 2	0 ± 2	0.74	4 ± 8	2 ± 5	**<0.01**

(SFA) Saturated Fatty Acids, (PUFA) Poly-Unsaturated Fatty Acids, (MUFA) Mono-Unsaturated Fatty Acids,
Values are reported as means ±SD,
[r]Independent T-test for continuous, normally distributed data,
Window 1: 03:00 – 9:59, Window 2: 10:00 – 14:59, Window 3: 15:00 – 19:59, Window 4: 20:00 – 02:59.

Appendix C.7 The association between chronotype (ET vs MT & IT) and nutrient intake according to morning stratified by BMI groups

Nutrient	Normal BMI (<25 kg/m2) n = 111				High BMI (≥25 kg/m2) n = 176			
	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*
Morning Intake (Window 1)								
Energy intake (kJ)	193	-346	-728, 36.3	0.08	152	-286	-585, 14.2	0.06
	Adjusted R^2 = 0.20, $P < 0.01^{\delta}$				Adjusted R^2 = 0.07, $P < 0.01$			
Protein intake (g)	1.86	-3.78	-7.47, -0.09	0.05	1.54	-2.60	-5.65, 0.45	0.09
	Adjusted R^2 = 0.09, $P < 0.01^{\delta}$				Adjusted R^2 = 0.07, $P < 0.01$			
Fat intake (g)	2.48	-3.56	-8.46, 1.36	0.15	1.97	-3.91	-7.79; -0.18	0.05
	Adjusted R^2 = 0.16, $P < 0.01^{\delta}$				Adjusted R^2 = 0.11, $P < 0.01^{\delta}$			
Carbohydrate intake (g)	5.51	-7.74	-18.7, 3.19	0.16	4.13	-7.76	-15.9, 0.40	0.06
	Adjusted R^2 = 0.13, $P < 0.01^{\delta}$				Adjusted R^2 = 0.04, $P < 0.01$			
Evening Intake (Window 4)								
Energy intake (kJ)	259	425	-87.7, 938	0.10	247	722	235, 1209	**<0.01**
	Adjusted R^2 = 0.17, $P < 0.01^{\delta}$				Adjusted R^2 = 0.17, $P < 0.01^{\delta}$			
Protein intake (g)	2.62	3.75	-1.45, 8.94	0.16	2.55	6.21	1.19, 11.2	**0.02**
	Adjusted R^2 = 0.13, $P < 0.01^{\delta}$				Adjusted R^2 = 0.15, $P < 0.01^{\delta}$			
Fat intake (g)	2.88	2.88	-2.81, 8.60	0.32	2.94	8.10	2.30, 13.9	**0.01**
	Adjusted R^2 = 0.22, $P < 0.01^{\delta}$				Adjusted R^2 = 0.19, $P < 0.01^{\delta}$			
Carbohydrate intake (g)	6.08	13.1	1.01, 25.1	**0.03**	6.04	17.2	5.28, 29.1	**0.01**
	Adjusted R^2 = 0.22, $P < 0.01^{\delta}$				Adjusted R^2 = 0.20, $P < 0.01^{\delta}$			
Carbohydrate intake (g)	6.08	13.1	1.01, 25.1	**0.03**	6.04	17.2	5.28, 29.1	**0.01**
	Adjusted R^2 = 0.22, $P < 0.01^{\delta}$				Adjusted R^2 = 0.20, $P < 0.01^{\delta}$			

(BMI) Body Mass Index.
Multiple linear regression models (Enter method) repeated within normal BMI (<25 kg/m^2) and high BMI (≥25 kg/m^2) groups, dependant variable: Nutrient Intake, Independent variable: Chronotype, adjusted for age, deprivation index & ethnicity, $^{\delta}$Covariate ethnicity significant in the model,
Missing deprivation index (n = 3).

Appendix D.1 Principal component food groups description

These food groups were derived from the food group list in Appendix A.3

Food Group	Food Items Included
Dairy milk	Full Fat Dairy Milk: dark blue, purple, silver top milk, lactose free regular fat milk & Low-Fat Dairy Milk: lite and trim milk (green or light blue), lactose free reduced fat milk
Sweetened Dairy & Milk alternatives,	Milk Alternatives: Soy, almond, rice, oat, coconut varieties, milk alternative based drinks from cafés & Sweetened Dairy Milk Products: Flavoured milk, fermented or evaporated milk, breakfast drinks, yakult fermented milk drink, hot chocolates, milk-based smoothies, milk-based drinks from cafés, coffee sachets, coffees made with syrup and cream e.g., caramel macchiato & Dairy Yogurt: all types of cow's milk yoghurt. Soy yoghurt under soy products, coconut yoghurt under coconut fats
Cheese	High Fat Cheese: Cream cheese, goat cheese, haloumi, parmesan, cheddar, processed cheese, blue vein, and mascarpone & Low-Fat Cheese: Brie, bocconcini, edam, feta, mozzarella, camembert, cottage, ricotta, and paneer
Apple, banana & orange	Apple, banana, and orange
Other fruit & Juices	All other fruit (fresh, canned, dried) & Fruit and/or vegetable juice, fruit and/or vegetable smoothies
Coloured Vegetables	Dark yellow vegetables: Dark yellow vegetables & Green vegetables: Lettuce, spinach, cabbage, broccoli, watercress, green beans, brussels sprouts, courgette
Non-Starchy Vegetables	Tomatoes: Fresh, canned, cooked tomatoes & Other: Capsicum, onion, mushrooms, frozen mixed vegetables, beetroot, squash
Starchy vegetables	Kumara, yam, parsnip, turnip, swedes (boiled, mashed, baked) Taro (flesh, roots, stalks), green banana, sweet corn kernels breadfruit, cassava, green banana, Potato (boiled, mashed, baked, salad, scalloped, roasted)
Refined grains	Macaroni and cheese, carbonara, white rice salad, two-minute noodles, white rice, white pasta, noodles (instant, egg, rice), canned spaghetti, cous-cous, including gluten free pasta, plain white bread, wraps, focaccia, bagel, pita, bread, rewena bread, doughboys, breadcrumbs, including gluten free options, naan (plain) & all crackers made from grains, cream crackers, cruskits, rice crackers,
Discretionary snack breads	Crumpets, scone, savoury muffin, plain croissant, pancakes, waffles, iced bun, savoury pin wheels, garlic bread, fruit bread, roti, naan (garlic)

Food Group	Food Items Included
Wholegrains	Tabbouleh, brown rice salad, Quinoa, buckwheat, bulgur wheat, brown rice, wholemeal pasta, wholegrain gluten free pasta e.g., brown rice & Wholegrain Breads: high fibre, wholemeal, wholegrain, including gluten free options & Oats: porridge, rolled oats, oat bran
Breakfast cereals	Sweetened: Sultana bran, light and fruity cereal, chocolate-based cereals, nutrigrain, honey puffs, milo cereals, fruit loops, oat sachets & Unsweetened: Weetbix, bran cereals, rice bubbles, cornflakes, all muesli, and granola
Meats	Red meat: Beef, lamb, venison, mince, patties, (stir fry, curry, stew) & White meat: Chicken, pork, turkey, (stir fry, curry, stew)
Processed meats	Corned beef (canned), corned silverside, smoked chicken, smoked hock and salami, ham, sausages, frankfurters, bacon, chorizo, luncheon meat
Fish & seafood	Canned, fresh, Ota ika, curry, stew
Eggs	Whole eggs (boiled, poached, fried, plain omelette), quiche, frittata, omelette with filling, egg, and banana pancake
Legumes	Baked beans, black beans, dahl, canned or dried legumes, hummus, and legume based vegetarian meals and products, edamame beans tofu, soy yoghurts
Nuts & seeds	Brazil nuts, walnuts, almond, cashew, pistachio, chia, linseed, pumpkin, sesame, peanut butter, and peanuts
Saturated Animal & Coconut Fats	Cream, sour cream, reduced cream, butter, lard, dripping, ghee & coconut products: oil, cream, milk, desiccated, yoghurt, fresh
Unsaturated Plant-Based Fats	Avocado (whole fruit), canola, sunflower, olive, vegetable oil, cooking spray, salad dressings (French/Italian), all margarines
Creamy dressings & sauces	Creamy readymade meal-based sauces, dips, mayonnaise, aioli, tartare sauce, white sauce, cheese sauce
Savoury sauces, spreads & condiments	Curry pastes, herb and spices, stock, vinegar, gravy and garlic sauce, instant and packet soups, oil-based condiments such as sundried tomatoes / olives in oil, pesto, tomato, barbeque, mint, soy, gravy, mustard, chutney, miso, pasta sauce, tomato paste, sweet chilli sauce, vegemite, marmite
Cakes & Puddings	Cakes & Biscuits: Slices, cakes, loaves, muffins, biscuits, doughnuts, sweet pies, pastries, tarts & Puddings & Deserts: Including all milk alternative ice creams, ice cream, custard, milk-based puddings e.g., rice, instant, semolina, pavlova, sticky date, fruit pies and crumbles, jelly, ice blocks
Sweet snack foods	Fruit and nut mixes, bliss balls, chocolate, lollies, muesli bars,
Savoury snack foods	Popcorn, potato crisps, corn chips, twisties, bujha mix,

Food Group	Food Items Included
Fast Foods	Pies, dumpling, burgers, pizzas, curries, noodle-based dishes, Nando's chicken, egg fu yong, Salads, sandwiches, wraps, sushi, vegetable-based stir fry, hot chips/fries, hash browns, and packaged home baked chips, wontons, paraoa bread, schnitzel, nuggets, crumbed fish
Sugar & Artificially Sweetened Beverages	All cordials, flavoured water, sports drinks, soft drinks, fruit drinks, iced tea, energy drinks and all artificially sweetened beverages
Tea	Black, green, herbal, chai, kombucha
Coffee	instant, brewed, espresso, pre-mixed sachet, filter, cold brew
Alcoholic drinks	Beer (standard and low alcohol), Wine (standard and low alcohol), Cider, spirits, ready to drink alcohol products (spirit or wine based with a non-alcoholic mixer), sherry, port, liqueurs
Sugar added to food & drink & Sweet Spreads	All sugar added to food and drink & Sweet Spreads: Jam, honey, marmalade, syrup (maple, golden), Nutella, chocolate peanut butter, chocolate butter
Shakes & Protein Powders	Protein powders, cacao powder, baking powder, baking soda, weight loss shakes, psyllium husk powder, cornflour, vanilla essence, natural sweeteners, yeast, flours
Water	Water (unflavoured, soda and tap)

Appendix D.2 Nutrient distribution between food pattern tertiles (T3 vs T1)

Nutrient[†]	Healthy				Discretionary Food & Beverages				Animal Products				Sugary, Fatty Foods			
	T1	T2	T3	*P*-value [†]	T1	T2	T3	*P*-value [†]	T1	T2	T3	*P*-value [†]	T1	T2	T3	*P*-value [†]
Energy (kJ)	9003 ± 2754	8394 ± 1887	8420 ± 1824	0.10[¥]	8519 ± 2257	8348 ± 2420	8949 ± 1878	0.15[¥]	8740 ± 2090	8167 ± 2133	8901 ± 2337	0.05[¥]	8136 ± 2261	8435 ± 1957	9237 ± 2256[a, b]	<0.01[¥]
Protein (g)	79.9 ± 23.9	84.3 ± 19.3	90.8 ± 21.8[a]	<0.01[¥]	89.1 ± 25.4	83.6 ± 22.3	82.3 ± 17.7	0.08[¥]	82.9 ± 22.0	81.0 ± 21.9	91.0 ± 21.4[a, b]	<0.01[¥]	81.5 ± 23.7	82.4 ± 20.4	90.9 ± 21.2[a, b]	0.01[¥]
Fat (g)	91.7 ± 31.0	89.0 ± 26.8	94.8 ± 30.3	0.40[¥]	85 [79, 92]	84 [7, 89]	92 [86, 98]	0.12[¥]	90.6 ± 27.5	87.0 ± 29.3	97.8 ± 30.5[b]	<0.01[¥]	81.1 ± 27.2	89.7 ± 25.1	105 ± 30.9[a, b]	<0.01[¥]
Carbohydrate (g)	236 ± 78.2	197.4 ± 53.9[a]	175.7 ± 56.9[a]	<0.01[¥]	190 [177, 203]	181 [166, 197]	204 [192, 217]	0.06[¥]	213 [200, 226] [ǂ]	188 [176, 200][a]	176 [161, 191][a]	<0.01[¥]	193 [181, 206]	190 [178, 203]	191 [176, 208]	0.95[¥]
Vitamin A (µg)	481, (297, 653)[b]	750 (555, 938)	923 (740, 1164)[a,b]	<0.01	625 (404, 946)	733 (486, 944)	767 (563, 987)	0.09	672 (493, 997)	653 (410, 920)	821 (553, 969)[b]	0.04	660 (385, 924)	665 (495, 914)	803 (574, 1039)[a]	0.01
Thiamine (mg)	1.2 (0.9, 1.5)	1.2 (0.9, 1.6)	1.2 (1.0, 1.6)	0.53	1.3 (1.0, 1.9)	1.2 (0.9, 1.4)	1.2 (0.9, 1.6)	0.22	1.2 (0.9, 1.6)	1.2 (1.0, 1.5)	1.2 (0.9, 1.6)	0.98	1.3 (1.0, 1.8)[b]	1.2 (0.9, 1.6)	1.2 (0.9, 1.4)[a]	0.01
Riboflavin (mg)	1.6 (1.1, 2.2)	1.7 (1.4, 2.1)	1.9 (1.6, 2.3)[a]	<0.01	1.8 (1.4, 2.1)	1.7 (1.3, 2.1)	1.8 (1.4, 2.1)	0.72	1.7 (1.3, 2.1)	1.6 (1.2, 2.1)	1.9 (1.6, 2.3)[a,b]	0.01	1.9 (1.4, 2.3)	1.7 (1.2, 2.2)	1.8 (1.4, 2.0)	0.22

Nutrient[†]	Healthy				Discretionary Food & Beverages				Animal Products				Sugary, Fatty Foods			
Niacin (mg)	33.9 (27.2, 42.1)	34.5 (29.5, 38.2)	36.4 (31.3, 41.9)	0.06	36.8 (29.8, 44.5)	34.4 (29.1, 39.7)	33.9 (28.6, 39.1)	0.09	34.6 (29.6, 40.8)	33.7 (27.3, 39.7)	36.4 (31.9, 40.3)	0.09	36.0 (29.0, 43)	34 (27.4, 39.5)	34.8 (30.9, 40.3)	0.20
Vitamin B6 (mg)	2.0 (1.3, 2.9)	2.0 (1.6, 2.6)	2.3 (1.8, 2.7)[a]	0.03	2.1 (1.6, 2.9)	2.1 (1.5, 2.6)	2.1 (1.7, 2.8)	0.27	2.2 (1.7, 2.7)	1.9 (1.5, 2.6)	2.3 (1.7, 2.9)	0.15	2.3 (1.5, 3)	2 (1.7, 2.6)	2 (1.5, 2.6)	0.39
Folate (µg)	250 ± 104[b]	324 ± 111	384 ± 123[a,b]	<0.01[¥]	272 [249, 297]	276 [251, 303]	338 [314, 364][a, b]	<0.01[¥]	310 ± 135	297 ± 123	352 ± 112[b]	0.01[¥]	311 ± 125	317 ± 127	331 ± 125	0.53[¥]
Vitamin B12 (µg)	3.7 (2.8, 4.7)	3.6 (2.7, 4.5)	3.6 (2.6, 4.9)	0.85	3.8 (2.6, 5.2)	3.6 (2.9, 4.4)	3.4 (2.7, 4.7)	0.35	3.4 (2.6, 4.3)	3.3 (2.4, 4.3)	4.2 (3.2, 5.3) [a, b]	<0.01	3.5 (2.4, 4.8)	3.5 (2.8, 4.6)	3.8 (2.8, 4.7)	0.42
Vitamin C (mg)	48 (26.2, 80.6)	53.1 (39.4, 84.4)	86.9 (62.8, 121)[a, b]	<0.01	55.3 (36.1, 83.6)	56.4 (39.5, 95.5)	80 (46.6, 115) [a]	0.01	79.8 (47.7, 112) [b]	56.1 (33.6, 94.3)	54.7 (33.4, 85.1) [a]	<0.01	49.2 (29.8, 84.3) [b]	65.5 (41.3, 105)	69.3 (46.4, 102) [a]	0.01
Vitamin E (mg)	7.7 (5.7, 9.7) [b]	8.9 (7.2, 10.6)	10.9 (8.7, 13.9) [a,b]	<0.01	8.5 (6.8, 11.4)	8.5 (6.7, 10.3)	10.4 (8.5, 12.6) [a,b]	<0.01	9 (7.4, 11.4)	8.8 (6.7, 11)	9.2 (7.3, 11.6)	0.66	8.3 (6.1, 10.1)	9.0 (7.4, 11.4)	10 (8.3, 12.6) [a]	<0.01
Sodium (mg)	3020 ± 1155	2795 ± 947	2459 ± 784 [a]	<0.01[¥]	2656 [2452, 2876]	2463 [2279, 2662]	2654 [2500, 2817]	0.25[¥]	2377 [2208, 2558]	2475 [2304, 2660]	2949 [2759, 3153][a, b]	<0.01[¥]	2506 [2320, 2707]	2512 [2347, 2688]	2756 [2562, 2966]	0.11[¥]

Nutrient[†]	Healthy				Discretionary Food & Beverages				Animal Products				Sugary, Fatty Foods			
Potassium (mg)	2510 ± 807[b]	2805 ± 631	3332 ± 778[a,b]	<0.01[¥]	2725 ± 845	2792 ± 806	3137 ± 736[a, b]	<0.01[¥]	2929 ± 767	2718 ± 870	3002 ± 786[b]	0.04[¥]	2729 ± 889	2867 ± 790	3053 ± 733[a]	0.02[¥]
Magnesium (mg)	252 (195, 288)[b]	285 (245, 334)	372 (304, 444)[a, b]	0.00	286 (232, 325)	277 (241, 351)	314 (270, 411)[a, b]	<0.01	287 (247, 357)	278 (233, 361)	309 (262, 371)	0.13	278 (237, 339)	291 (237, 356)	309 (269, 394)[a]	0.01
Calcium (mg)	607 (460, 842)[b]	751 (573, 928)	846 (713, 1121)[a, b]	<0.01	705 (516, 873)	723 (521, 928)	844 (693, 1050) [a, b]	<0.01	762 (551, 922)	730 (515, 916)	792 (641, 1022)	0.08	731 (534, 962)	753 (543, 904)	800 (627, 940)	0.29
Iron (mg)	11 ± 3.6	11.4 ± 3.2	13.1 ± 4.1[a]	<0.01[¥]	12 ± 4	11.1 ± 3.4	12.4 ± 3.7	0.33[¥]	11.3 [10.5, 12.1]	10.5 [9.81, 11.2]	11.9 [11.3, 12.7] [a,b]	<0.01[¥]	11.5 ± 3.9	11.5 ± 3.9	12.4 ± 3.4	1.00[¥]
Zinc (mg)	9.7 (7.7, 11.5)	10 (8.4, 11.7)	10.4 (9, 12.2)	0.09	10.4 (8.4, 13.3)	9.8 (8.6, 11.7)	9.8 (8.5, 11.2)	0.23	9.9 (8, 11.5)	9.5 (8.1, 11)	10.8 (9.4, 13.4)[a, b]	<0.01	9.6 (7.9, 11.5)	9.8 (8.7, 11.4)	10.7 (9.3, 12.6)[a]	0.01
Selenium (µg)	53.5 (38.6, 75)	53.4 (47.1, 69.6)	67.1 (50.4, 84.6)[a, b]	<0.01	56.3 (46.3, 79.1)	56.3 (39.4, 73.1)	59.3 (47.3, 75.8)	0.31	56.5 (43.2, 84.4)	52.8 (42.9, 65.1)	65.9 (50.1, 80)[b]	<0.01	56.6 (42.9, 76.4)	53.5 (43.1, 72.2)	62.6 (49.9, 79.1)	0.07
Iodine (µg)	82.7 (55.4, 110)	96.1 (70, 118)	94.5 (75.9, 136)[a]	<0.01	85.1 (58.8, 110)	87.1 (66.3, 112)	103 (81.9, 136)[a, b]	<0.01	84.2 (64.7, 113)	84.6 (58.8, 113)	105 (83.6, 135)[a, b]	<0.01	86.2 (64.9, 111)	89.9 (66, 122)	97.3 (76.5, 128)	0.12

Values are reported as Median (25th ,75th percentiles), means ±SD, or geometric mean [95% CI] for the log transformed data values, back transformed to the original scale.

[†]Kruskall- Walis with multiple comparison rank tests were used to compare between three groups (T1, T2, T3) of continuous, non-normally distributed data, [a] compared against T1, [b] compared against T2, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167, [¥]One-way ANOVA for comparing three groups continuous, normally distributed data with Tukey HSD as post hoc test.

Appendix D.3 Food distribution between food pattern tertiles (T3 vs T1)

Food Group (g)	Micronutrient-Rich			High Protein-Fat-Fibre			High Vitamin B			High Carbohydrate		
	T1	**T2**	**T3**	**T1**	**T2**	**T3**	**T1**	**T2**	**T3**	**T1**	**T2**	**T3**
Nuts & seeds	3 (0, 15)	1 (0, 10)	4 (0, 13)	0 (0, 8)	0 (0, 10)	10 (0, 27)[a, b]	2 (0, 15)	0 (0, 13)	3 (0, 12)	7 (0, 21)	3 (0, 13)	0 (0, 8)[a]
Coloured vegetables	50 (20, 91)	54 (10, 95)	50 (12, 89)	34 (12, 83)	50 (9, 91)	69 (28, 113)[a]	55 (15, 93)	40 (8, 109)	48 (14, 87)	83 (28, 120)	56 (21, 86)	26 (4, 54)[a, b]
Fast Foods	136 (48, 253)	101 (36, 249)	108 (48, 217)	99 (40, 184)	131 (48, 256)	133 (45, 250)	114 (46, 241)	143 (48, 248)	101 (36, 212)	64 (14, 136)	111 (50, 128)	206 (119, 288)[a, b]
Non-Starchy Vegetables	22 (8, 53)	31 (11, 69)	36 (13, 63)	21 (4, 48)	29 (9, 58)	43 (15, 71)[a]	24 (5, 56)	34 (10, 62)	31 (13, 69)	43 (20, 93)[b]	32 (9, 58)	17 (5, 45)[a]
Sugar & Artificially Sweetened Beverages	77 (0, 251)	83 (0, 236)	67 (0, 189)	95 (0, 227)	95 (0, 275)	50 (0, 194)	76 (0, 118)	88 (0, 254)	67 (0, 246)	23 (0, 137)	66 (0, 162)	195 (26, 345)[a, b]
Coffee	1 (0, 75)	1 (0, 49)	1 (0, 67)	0 (0, 12)	1 (0, 52)	1 (0, 131)	1 (0, 52)	1 (0, 49)	1 (0, 77)	13 (0, 115)[b]	0 (0, 52)	0 (0, 6)[a]
Cheese	11 (0, 23)	10 (0, 26)	13 (2, 25)	5 (0, 18)	12 (1, 23)	17 (4, 36)[a]	10 (0, 30)	11 (0, 21)	12 (1, 23)	17 (3, 31)	13 (1, 26)	6 (0, 20)[a]
Eggs	24 (0, 48)	0 (0, 55)	0 (0, 61)	10 (0, 26)	21 (0, 50)	36 (12, 84)[a, b]	23 (1, 58)	14 (0, 50)	22 (0, 44)	46 (12, 84)[b]	21 (0, 42)	5 (0, 24)[a, b]
Unsaturated Plant-Based fats	7 (2, 19)	9 (2, 23)	6 (2, 19)	5 (1, 13)	7 (2, 18)	14 (4, 32)[a, b]	9 (1, 23)	6 (1, 18)	7 (3, 20)	12 (6, 28)[b]	7 (1, 19)	5 (1, 13)[a]
Tea	40 (0, 223)	102 (0, 306)	41 (0, 237)	30 (0, 242)	50 (0, 223)	117 (0, 306)	0 (0, 204)	56 (0, 266)	101 (0, 275)	113 (0, 284)	50 (0, 285)	0 (0, 190)[a]

Food Group (g)	Micronutrient-Rich			High Protein-Fat-Fibre			High Vitamin B			High Carbohydrate		
Apple, Banana & Oranges	42 (0, 99)	41 (0, 109)	41 (0, 77)	41 (0, 100)	32 (0, 80)	47 (14, 92)	41 (0, 88)	48 (5, 89)	33 (0, 93)	41 (0, 91)	41 (0, 89)	41 (0, 81)
Shakes & Protein Powders	0 (0, 0)	0 (0, 1)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 2)[a]	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 2)	0 (0, 0)	0 (0, 0)[a]
Refined Grains	83 (50, 146)	85 (41, 150)	100 (46, 134)	91 (41, 121)	84 (43, 134)	94 (53, 161)	95 (49, 181)	89 (48, 140)	85 (39, 124)	60 (28, 94)[b]	94 (52, 130)	127 (66, 194)[a, b]
Wholegrains	24 (9, 59)	42 (0, 88)	37 (0, 77)	30 (0, 65)	32 (0, 78)	36 (9, 83)	35 (8, 66)	30 (0, 76)	33 (8, 76)	32 (2, 67)	40 (14, 84)	24 (0, 71)
Savoury Snack Foods	4 (0, 17)	4 (0, 22)	3 (0, 17)	0 (0, 12)	2 (0, 20)	13 (0, 26)[a]	4 (0, 17)	3 (0, 20)	4 (0, 17)	0 (0, 10)[b]	7 (0, 18)	11 (0, 27)[a]
Meat	96 (45, 139)	90 (37, 130)	101 (61, 161)	70 (30, 106)	100 (65,140)	126 (67,183)[a]	98 (62, 139)	105 (55, 164)	84 (31, 128)	106 (65, 152)	94 (56, 142)	84 (30, 127)
Legumes	0 (0, 10)	0 (0, 24)	0 (0, 22)	0 (0, 12)	0 (0, 16)	0 (0, 34)	0 (0, 14)	0 (0, 13)	0 (0, 33)	0 (0, 23)	0 (0, 15)	0 (0, 3)
Sweet Snack Foods	13 (0, 34)	12 (1, 28)	14 (0, 28)	10 (0, 21)	13 (2, 28)	21 (1, 37)[a]	12 (0, 24)	12 (1, 31)	16 (2, 30)	10 (1, 26)	12 (0, 24)	18 (1, 34)
Discretionary Snack Breads	0 (0, 20)	0 (0, 20)	0 (0, 16)	0 (0, 23)	0 (0, 20)	0 (0, 16)	0 (0, 20)	0 (0, 20)	0 (0, 19)	0 (0, 15)	0 (0, 19)	0 (0, 28)
Starchy Vegetables	22 (0, 56)	40 (0, 76)	37 (6, 72)	29 (0, 65)	42 (0, 85)	34 (8, 69)	35 (11, 76)	29 (0, 64)	35 (3, 76)	21 (0, 60)	40 (9, 80)	41 (10, 76)
Processed Meats	7 (0, 21)	11 (0, 24)	17 (0, 46)[a]	4 (0, 22)	12 (0, 35)	15 (0, 33)[a]	12 (0, 24)	12 (0, 36)	9 (0, 32)	12 (0, 40)	11 (0, 28)	8 (0, 26)
Alcoholic drinks	0 (0, 90)	0 (0, 50)	0 (0, 61)	0 (0, 2)	0 (0, 90)[a]	0 (0, 103)	0 (0, 76)	0 (0, 0)	0 (0, 99)[b]	0 (0, 100)	0 (0, 80)	0 (0, 0)[a]
Cakes & Puddings	35 (10, 85)	25 (9, 57)	35 (11, 56)	30 (8, 58)	32 (9, 61)	38 (10, 78)	31 (5, 77)	28 (8, 59)	39 (12, 65)	19 (0, 38)	36 (14, 65)	57 (16, 105)[a]

Food Group (g)	Micronutrient-Rich			High Protein-Fat-Fibre			High Vitamin B			High Carbohydrate		
Sweetened Dairy & Milk Alternatives	54 (5, 108)	26 (0, 125)	36 (2, 103)	25 (0, 85)	30 (0, 108)	54 (6, 131)	39 (2, 120)	32 (0, 104)	53 (0, 106)	33 (1, 78)	32 (0, 103)	53 (1, 157)
Dairy Milk	87 (21, 194)	74 (11, 148)	84 (33, 155)	83 (23, 155)	88 (25, 180)	78 (6, 167)	92 (11, 157)	87 (36, 169)	70 (18, 144)	57 (3, 146)	90 (39, 155)	104 (31, 206)[a]
Other Fruit & Juices	52 (7, 114)	53 (8, 136)	50 (0, 96)	50 (0, 126)	53 (5, 117)	57 (14, 109)	48 (8, 114)	53 (2, 125)	53 (0, 127)	31 (0, 77)	55 (8, 132)	64 (15, 143)[a]
Sugar-Added to Food & Beverages & Sweet Spreads	3 (0, 11)	5 (0, 12)	4 (0, 13)	4 (0, 10)	4 (0, 12)	3 (0, 13)	4 (0, 12)	4 (0, 11)	4 (0, 12)	2 (0, 5)[b]	4 (0, 11)	9 (1, 20)[a, b]
Saturated Animal & Coconut Fats	7 (1, 18)	5 (0, 17)	7 (2, 16)	3 (0, 10)	7 (1, 16)	12 (4, 24)[a, b]	8 (1, 20)	7 (0, 17)	7 (2, 15)	8 (2, 18)	7 (1, 18)	5 (0, 14)
Creamy Dressings & Sauces	2 (0, 9)	1 (0, 7)	1 (0, 13)	0 (0, 4)[b]	3 (0, 13)	3 (0, 10)[a]	2 (0, 8)	1 (0, 9)	1 (0, 7)	1 (0, 10)	1 (0, 9)	2 (0, 7)
Fish & Seafood	3 (0, 36)	4 (0, 35)	4 (0, 31)	0 (0, 37)	3 (0, 30)	10 (0, 39)	0 (0, 26)	1 (0, 36)	11 (0, 40)	7 (0, 35)	0 (0, 30)	6 (0, 34)
Breakfast Cereals	5 (0, 24)	7 (0, 30)	0 (0, 17)	9 (0, 29)	0 (0, 20)	0 (0, 19)	0 (0, 13)[b]	10 (0, 27)	0 (0, 23)	0 (0, 9)[b]	7 (0, 23)	12 (0, 30)[a]
Savoury Sauces Spreads & Condiments	13 (3, 28)	15 (3, 34)	8 (3, 20)	7 (1, 28)	14 (4, 27)	14 (5, 29)	14 (5, 30)	8 (1, 28)	14 (6, 25)	9 (2, 20)[b]	14 (6, 31)	10 (3, 30)

Values are reported as Median (25^{th} ,75^{th} percentiles),
Kruskall- Walis with multiple comparison rank tests were used to compare between three groups (T1, T2, T3) of continuous, non-normally distributed data, [a] compared against T1, [b] compared against T2, adjusted by the Bonferroni correction for multiple comparisons: 0.05 / 3 = 0.0167.

Appendix E.1 The association between chronotype (ET vs MT-IT) & eating behaviours, eating attitudes & dietary intake stratified by BMI groups & BF% groups

Eating behaviours & attitudes	**Normal BMI (<25 kg/m², n = 111)**				**High BMI (≥25 kg/m², n = 176)**				**Normal BF% (<35%)**				**High BF% (≥35%)**			
	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*	SE (B)	B	95% CI	*P*
Restraint	1.12	0.16	-2.05, 2.37	0.88	**0.81**	**-2.01**	**-3.60, -0.42**	**0.01**	1.12	-1.17	-3.39, 1.05	0.30	0.87	-1.03	-2.74, 0.68	0.24
	Adjusted R^2 = 0.07, *p* = 0.01				Adjusted R^2 = 0.02, *p* = 0.07				Adjusted R^2 =0.01, *p* = 0.19				Adjusted R^2 =-0.004, p = 0.48			
Flexible Control	0.43	-0.12	-0.97, 0.73	0.79	**0. 30**	**-0.84**	**-1.43, -0.25**	**0.01**	0.41	-0.58	-1.39, 0.24	0.17	0.33	-0.51	-1.16, 0.13	0.12
	Adjusted R^2 = 0.05, *p* = 0.04				Adjusted R^2 = 0.05, *p* = 0.01				Adjusted R^2 =0.01, *p* = 0.19				Adjusted R^2 =0.01, *p* = 0.19			
Rigid Control	0.42	-0.19	-1.02, 0.65	0.66	0.34	-0.72	-1.39, -0.06	0.34	0.46	-0.52	-1.43, 0.38	0.26	0.35	-0.50	-1.19, 0.19	0.15
	Adjusted R^2 = 0.10, *p* = 0.01				Adjusted R^2 = 0.02, *p* = 0.08				Adjusted R^2 =0.05, *p* = 0.02				Adjusted R^2 =0.003, *p* = 0.34			
Hunger	**0.75**	**-1.72**	**-3.21, -0.22**	**0.03**	**0.63**	**1.60**	**0.36, 2.84**	**0.01**	0.77	-0.76	-2.28, 0.75	0.32	0.68	1.33	-0.02, 2.69	0.05
	Adjusted R^2 = 0.06, *p* = 0.01				Adjusted R^2 = 0.03, *p* = 0.03				Adjusted R^2 =0.02, *p* = 0.11				Adjusted R^2 =0.05, *p* = 0.08			
Internal locus	**0.42**	**-1.10**	**-1.94, -0.27**	**0.01**	**0.34**	**0.93**	**0.26, 1.61**	**0.01**	0.42	-0.41	-1.24, 0.42	0.33	**0.38**	**0.80**	**0.05, 1.55**	**0.04**
	Adjusted R^2 =0.07, p = 0.01				Adjusted R^2 = 0.05, *p* = 0.01				Adjusted R^2 =0.01, *p* = 0.28				Adjusted R^2 =0.05, *p* = 0.01			
External locus	**0.36**	**-0.80**	**-1.52, -0.08**	**0.03**	**0.31**	**0.54**	**-0.07, 1.16**	**0.08**	0.37	-0.59	-1.32, 0.15	0.12	0.34	0.44	-0.22, 1.11	0.19
	Adjusted R^2 = 0.05, p = 0.04				Adjusted R^2 = 0.02, *p* = 0.11				Adjusted R^2 =0.02, *p* = 0.11				Adjusted R^2 =0.02, *p* = 0.16			
Disinhibition	0.82	-1.47	-3.10, 0.15	0.08	0.62	1.01	-0.21, 2.22	0.10	0.83	-1.12	-2.76, 0.53	0.18	0.69	1.00	-0.36, 2.37	0.15
	Adjusted R^2 = 0.03, *p* = 0.08				Adjusted R^2 = 0.14, *p* < 0.001				Adjusted R^2 =0.002, *p* = 0.36				Adjusted R^2 =0.13, *p* <0.001 [@]			
Situational	**0.35**	**-1.15**	**-1.85, -0.46**	**<0.01**	0.26	0.20	-0.30, 0.71	0.43	**0.35**	**-1.15**	**-1.85, -0.46**	**<0.01**	0.28	0.44	-0.11, 1.00	0.12
	Adjusted R^2 = 0.08, *p* =0.01				Adjusted R^2 = 0.05, *p* =0.01				Adjusted R^2 =0.07, *p* = 0.01				Adjusted R^2 =0.06, *p* = 0.01 [@]			
Habitual	0.34	0.07	-0.61, 0.75	0.84	**0.28**	**0.63**	**0.08, 1.18**	**0.02**	0.33	1.75	-0.49, 0.84	0.60	0.31	0.57	-0.05, 1.18	0.07
	Adjusted R^2 = 0.00, *p* = 0.32				Adjusted R^2 = 0.06, *p* = 0.00				Adjusted R^2 =0.01, *p* = 0.31				Adjusted R^2 =0.05, *p* = 0.01 [@]			

Emotional	0.28	-0.26	-0.81, 0.29	0.36	0.20	0.08	-0.33, 0.48	0.71	0.28	-0.09	-0.65, 0.47	0.76	0.22	-0.12	-0.56, 0.32	0.59
	Adjusted R^2 = 0.05, p = 0.04				Adjusted R^2 = 0.18, p <0.001				Adjusted R^2 =-0.01, p = 0.52				Adjusted R^2 =0.20, p <0.001 [ø]			
Total EAT-26	3.26	-1.14	-7.60, 5.32	0.73	3.06	5.04	-1.01, 11.1	0.10	3.45	2.34	-4.48, 9.15	0.50	3.31	3.60	-2.94, 10.1	0.28
	Adjusted R^2 = -0.01, p = 0.63				Adjusted R^2 = 0.03, p =0.05				Adjusted R^2 =-0.001, p = 0.41				Adjusted R^2 =0.02, p = 0.10			
Dieting	1.30	-1.17	-3.74, 1.41	0.37	1.15	0.64	-1.63, 2.91	0.58	1.40	0.18	-2.60, 2.95	0.90	1.21	0.08	-2.31, 2.48	0.95
	Adjusted R^2 = -0.02, p = 0.77				Adjusted R2 = -0.00, p = 0.47				Adjusted R^2 =-0.02, p = 0.99				Adjusted R^2 =-0.002, p = 0.44			
Bulimia & Food Pre-occupation	0.05	0.06	-9.94, 1.05	0.91	**0.52**	**1.43**	**0.40, 2.47**	**0.01**	0.50	0.12	-0.86, 1.10	0.80	**0.58**	**1.74**	**0.59, 2.89**	**0.003**
	Adjusted R^2 = -0.01, p =0.69				Adjusted R^2 = 0.08, p <0.001				Adjusted R^2 =0.01, p = 0.20				Adjusted R^2 =0.08, p = 0.001 [ø]			
Oral Control	0.50	1.08	-0.10, 2.25	0.91	**0.52**	**0.90**	**0.06, 1.73**	**0.01**	**0.58**	**1.74**	**0.60, 2.88**	**<0.01**	0.47	-0.05	-0.97, 0.87	0.92
	Adjusted R^2 = 0.26, p = 0.69				Adjusted R^2 = 0.10, p <0.001				Adjusted R^2 =0.08, p = 0.001				Adjusted R^2 =0.08, p = 0.001 [ø]			

(NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (EAT) Eating Attitudes Test – 26
Multiple linear regression models (Enter method) repeated within normal (<25 kg/m²) and high BMI (≥25 kg/m²) groups and then within normal (<35%) and high (≥35%) BF% groups,
Dependant variable: Eating Behaviour/Attitude, Independent variable: Chronotype groups (MT-IT and ET), adjusted for age, & ethnicity,
Missing TFEQ data (n = 2), Missing deprivation index (n = 3)
P-value of < 0.05 was accepted as statistically significant
[ø] *Ethnicity significant in model*

Appendix E.2 Multinominal regression - BMI groups & BF% groups (adjusted for deprivation index)

Eating behaviour combinations	**Normal BMI (<25 kg/m^2)**		**High BMI (≥25 kg/m^2)**		**Low BF% (<35%)**		**High BF% (≥35%)**	
Eating behaviour combinations of restraint & disinhibition	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
High Restraint – Low Disinhibition	Reference category		Reference category		Reference category		Reference category	
Low Restraint – Low Disinhibition	0.20	1.22 [0.31, 4.82]	1.09	2.98 [0.93, 9.50]	-0.37	0.70 [0.20, 2.42]	-0.90	0.41 [0.11, 1.44]
High Restraint – High Disinhibition	-0.56	0.57 [0.10, 3.41]	0.55	1.73 [0.55, 5.43]	1.04	2.81 [0.59, 13.4]	-0.93	0.40 [0.11, 1.41]
Low Restraint – High Disinhibition	-1.57	0.21 [0.03, 1.68]	**1.37**	**3.93 [1.20, 12.8]***	0.70	2.00 [0.38, 10.6]	**-1.28**	**0.28 [0.80, 0.96]***
Eating behaviour combinations of hunger & disinhibition	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
High Hunger – Low Disinhibition	-0.52	0.59 [0.13, 2.75]	0.75	2.11 [0.64, 7.00]	-0.29	0.75 [0.19, 3.04]	-0.58	0.56 [0.16, 2.00]
Low Hunger – Low Disinhibition	Reference category		Reference category		Reference category		Reference category	
High Hunger – High Disinhibition	**-1.81**	**0.16 [0.03, 0.88] ***	0.94	2.56 [1.00, 6.86] [@]	1.03	2.79 [0.77, 10.2]	-1.02	0.36 [0.12, 1.06]
Low Hunger – High Disinhibition	-0.06	0.94 [0.17, 5.19]	-0.25	0.78 [0.16, 3.94]	0.34	1.41 [0.24, 8.24]	-0.20	0.82 [0.15, 4.48]

ET (Ref. MT-IT)
(BMI) body mass index; (ET) evening type; (MT-IT); morning type & intermediate type combined
Model 1: Multinomial logistic regression for restraint and disinhibition eating behaviours combinations (low restraint – low disinhibition, high restraint – high disinhibition, and low restraint – high disinhibition) relevant to the ideal eating behaviour combination, high restraint – low disinhibition. Model 2: Multinomial logistic regression for hunger and disinhibition eating behaviours combinations (high hunger – low disinhibition, high hunger – high disinhibition, and low hunger – high disinhibition) relevant to the ideal eating behaviour combination, low hunger – low disinhibition.
Missing deprivation index (n = 3), Missing TFEQ (n = 2)
**P-value of < 0.05 was accepted as statistically significant*

Appendix E.3 Multinominal regression - BMI groups & BF% groups (unadjusted for deprivation index)

Eating behaviour combinations	**Normal BMI (<25 kg/m²)**		**High BMI (≥25 kg/m²)**		**Low BF% (<35%)**		**High BF% (≥35%)**	
Eating behaviour combinations of restraint & disinhibition	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
High Restraint – Low Disinhibition	Reference category		Reference category		Reference category		Reference category	
Low Restraint – Low Disinhibition	0.27	1.32 [0.34, 5.09]	0.90	2.47 [0.83, 7.31]	-0.36	0.70 [0.20, 2.40] [a]	-0.84	0.43 [0.13, 1.49] [a]
High Restraint – High Disinhibition	-0.18	0.83 [0.16, 4.38]	0.54	1.72 [0.60, 4.92] [a]	1.04	2.83 [0.60, 13.4]	-0.86	0.42 [0.12, 1.46] [a]
Low Restraint – High Disinhibition	-1.36	0.26 [0.04, 1.60] [a]	**1.30**	**3.66 [1.24, 10.8] * [a]**	0.98	2.65 [0.53, 13.4]	**-1.25**	**0.29 [0.09, 0.96]***
Eating behaviour combinations of hunger & disinhibition	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]	β	OR [95% CI]
High Hunger – Low Disinhibition	-0.30	0.74 [0.18, 3.10]	0.93	2.54 [0.80, 8.04]	-0.34	0.71 [0.18, 2.79]	-0.58	0.56 [0.16, 1.95]
Low Hunger – Low Disinhibition	Reference category		Reference category		Reference category		Reference category	
High Hunger – High Disinhibition	**-1.72**	**0.18 [0.04, 0.83] ***	**1.02**	**2.78 [1.12, 6.90] * [a]**	1.17	3.22 [0.90, 11.5]	-1.04	0.35 [0.12, 1.03]
Low Hunger – High Disinhibition	0.32	1.38 [0.29, 6.67]	0.07	1.07 [0.24, 4.71] [a]	0.36	1.43 [0.24, 8.46]	-0.17	0.84 [0.16, 4.54]

ET (Ref. MT-IT)
(BMI) body mass index; (ET) evening type; (MT-IT); morning type & intermediate type combined
Model 1: Multinomial logistic regression for restraint and disinhibition eating behaviours combinations (low restraint – low disinhibition, high restraint – high disinhibition, and low restraint – high disinhibition) relevant to the ideal eating behaviour combination, high restraint – low disinhibition. Model 2: Multinomial logistic regression for hunger and disinhibition eating behaviours combinations (high hunger – low disinhibition, high hunger – high disinhibition, and low hunger – high disinhibition) relevant to the ideal eating behaviour combination, low hunger – low disinhibition.
Missing deprivation index (n = 3), Missing TFEQ (n = 2)
**P-value of < 0.05 was accepted as statistically significant*
[a]*ethnicity significant in the model*

Appendix E.4 The association between chronotypes and eating behaviours and attitudes in BMI groups (adjusted models)

Chronotype	**Normal BMI (<25 kg/m²) n=111**				**High BMI (≥25 kg/m²) n=176**			
Restraint								
	Low score <7, n (%)	High score ≥7, n (%)	β	OR [95% CI]	Low score <7, n (%)	High score ≥7, n (%)	β	OR [95% CI]
MT-IT	41	44	Reference category		44	61	Reference category	
ET	17	9	-0.08	0.93 [0.30, 2.88]	40	31	**-0.99**	**0.37 [0.17, 0.84] ***
Hunger								
	Low score <5, n (%)	High score ≥5, n (%)	β	OR [95% CI]	Low score <5, n (%)	High score ≥5, n (%)	β	OR [95% CI]
MT-IT	51	34	Reference category		48	57	Reference category	
ET	16	10	-0.97	0.38 [0.11, 1.29]	20	51	**0.98**	**2.67 [1.17, 6.11] ***
Disinhibition								
	Low score <7, n (%)	High score ≥7, n (%)	β	OR [95% CI]	Low score <7, n (%)	High score ≥7, n (%)	β	OR [95% CI]
MT-IT	52	33	Reference category		36	69	Reference category	
ET	21	5	-1.12	0.32 [0.88, 1.15]	35	36	0.51	1.67 [0.71, 3.89]
Total EAT-26								
	Low score <20, n (%)	High score ≥20, n (%)	β	OR [95% CI]	Low score <20, n (%)	High score ≥20, n (%)	β	OR [95% CI]
MT-IT	64	21	Reference category		62	43	Reference category	
ET	20	6	-0.63	0.53 [0.15, 1.95]	36	35	0.24	1.27 [0.59, 2.77]

(NZ) New Zealand; (BMI) Body Mass Index, (BF%) Body Fat Percentage, (EAT) Eating Attitudes Test – 26,
**P-value of < 0.05 was accepted as statistically significant*
Binary Logistic regression (enter method) repeated within low (<25 kg/m²) and high BMI (≥25 kg/m²) groups, Dependant variable: Eating Behaviour/Attitude, Independent variable: Chronotype, adjusted for age, deprivation index & ethnicity
Missing TFEQ data (n = 2), Missing deprivation index (n = 3)