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Sleep characteristics and cardiometabolic disease risk factors in corporate executives

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Declaration of inclusion of publications

I confirm that I have been granted permission by the University of Cape Town's Doctoral Degrees Board to include the following publication(s) in my PhD thesis, and where co-authorships are involved, my co-authors have agreed that I may include the publication(s):

1. Pienaar PR, Kolbe-Alexander TL, van Mechelen W, Boot CR, Roden LC, Lambert EV, Rae DE. Associations between self-reported sleep duration and mortality in employed individuals: systematic review and meta-analysis. *American journal of health promotion*. 2021 Jul;35(6):853-65. Published (Chapter 2)
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5. Pienaar PR, Roden LC, Boot CR, van Mechelen W, Suter JA, Lambert EV, Rae DE. Associations between habitual sleep characteristics and cardiometabolic disease risk in corporate executives. (Chapter 5) Submitted

The tables and figures of these manuscripts have been edited to allow for consecutive numbering throughout the thesis. Likewise, the referencing style has been updated to maintain consistency throughout this thesis and are presented collectively in a reference list at the end of the document.

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Abbreviations

ACM	All-cause Mortality
ATP-III	Adult Treatment Panel III of the National Cholesterol Education Program
BMI	Body Mass Index
BP	Blood Pressure
CI	Confidence Interval
CMD	Cardiometabolic Disease
CVD	Cardiovascular Disease
CVDM	Cardiovascular Disease Mortality
DASS	Depression, Anxiety and Stress Scale
DBP	Diastolic Blood Pressure
DET	Definite Evening-type
DMT	Definite Morning-type
EDS	Excessive Daytime Sleepiness
ESS	Epworth Sleepiness Scale
Glu	Glucose
HDL	High-density Lipoprotein
HÖ-MEQ	Horne-Östberg Morningness-Eveningness Personality Questionnaire
HRA	Health Risk Assessment
ISI	Insomnia Severity Index
IQR	Interquartile Range
LDL	Low-density Lipoprotein
MET	Moderate Evening-type
MMT	Moderate Morning-type
NCD	Non-communicable Disease
NFR	Need For Recovery
NOS	Newcastle-Ottawa Scale

NS	Not Significant
NT	Neither-type
PA	Physical Activity
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
RR	Relative Risk
SBP	Systolic Blood Pressure
SE	Sleep Efficiency
SOL	Sleep Onset Latency
T2DM	Type 2 Diabetes Mellitus
TC	Total Cholesterol
TG	Triglycerides
TiB	Time-in-Bed
TST	Total Sleep Time
WA	Work Ability
WASO	Wake After Sleep Onset
WHO	World Health Organisation
WC	Waist Circumference
WHP	Workplace health promotion

Abstract

Hours spent in work and sleep comprise the majority of time in a typical day of working adults. As a result, the workplace is a key setting for public health action. Among working adults, 71% of deaths globally are related to non-communicable diseases (NCDs), most of which are attributed to cardiometabolic diseases (CMD). While there is clear evidence linking short sleep duration with CMD risk in the general population, similar data in a unique subset of the workforce, namely corporate executives, remains largely unexplored.

The purpose of this thesis was to investigate the associations between sleep health and CMD risk in corporate executives. A systematic review and meta-analysis examined associations between self-reported sleep duration, all-cause mortality (ACM) and cardiovascular disease mortality (CVDM) in employed adults. Sleeping <6 h was associated with a 16% greater risk for ACM and a 26% greater risk for CVDM compared to 7-8 h of sleep, providing strong evidence that adequate sleep mitigates risk of ACM and CVDM (Chapter 2).

Health risk assessment (HRA) data were utilised to investigate cross-sectional (Chapter 3) and longitudinal (Chapter 4) associations between self-reported sleep duration and CMD risk factors. Both studies confirmed that shorter sleep duration was associated with higher CMD risk. Longer work hours and work commute time, depression, anxiety and stress were all associated with shorter sleep duration; and the relationship between sleep duration and CMD risk was mediated by lower physical activity, longer work hours and elevated stress.

To better understand the habitual sleep characteristics of corporate executives, sleep was measured objectively using actigraphy to describe and study the sleep-CMD risk relationship in Chapter 5. Corporate executives had an average actigraphy-derived sleep duration of below 7 h, 52% reported poor sleep quality and 26% displayed catch-up sleep on non-work days. Notably, shorter weekday total sleep time, lower sleep efficiency and being a catch-up sleeper were all associated with increased CMD risk.

Given these findings, it was fundamental to consider workplace sleep health programmes. Chapter 6 qualitatively explored barriers and facilitators of employee participation in sleep health programmes and found that the most common themes that emerged were poor sleep health awareness; work culture; work-family balance and confidentiality. When asked to conceptualise an ideal sleep health programme, three key components were emphasised: identifying the need for such a programme in the workplace; incorporating sleep health screening into HRAs and raising awareness of the importance of sleep through sleep health education.

In summary, the findings from this thesis collectively underpin the adverse cardiometabolic health consequences of shorter sleep duration among corporate executives in both the short and medium term. It further highlights the significance of protecting weekday sleep duration and reveals that occupational and psychological factors may contribute to, and mediate the observed sleep-cardiometabolic health relationship. A workplace sleep health programme designed to mitigate the adverse cardiometabolic health

outcomes of suboptimal sleep should consider raising awareness around sleep health, addressing work culture and work-life balance to promote participation in such programmes.

Chapter 1

General Introduction

1.1 Background

Non-communicable diseases (NCDs), namely cardiovascular diseases, diabetes, cancer and chronic respiratory disease, have a higher morbidity and mortality rate globally than do all other causes combined.¹ Additionally, in adults between the ages of 35 and 70 years they account for approximately 71% of deaths. The traditional risk factors for NCDs, namely tobacco use, physical inactivity, excessive alcohol intake and unhealthy diets are emphasised as targets for NCD prevention. Yet sleep, fundamental to life, is often overlooked.

The undeniable health implications of poor sleep quantity on cardiovascular disease (CVD), type 2 diabetes, mental health and mortality have been well documented in the broader population.^{2,3} When the relationship between sleep duration and NCDs is described in the literature, however, it generally encompasses working age adults exposed to shift work⁴ or does not distinguish between occupational groups (managerial vs non-managerial roles),^{5,6} leaving studies on managerial positions in non-shift workers considerably unexplored. For the purpose of this thesis, employees at senior management and executive level will collectively be defined by the term "corporate executives".

Corporate executive positions are characterised by amplified levels of stress, extended work hours, critical business decision-making, incessant job demands and increased pressure to perform as a leader. It is therefore unsurprising that the demanding corporate workplace culture promotes a host of behaviours that devalue sleep in favour of work time. Consequently, this culture may compromise effective leadership, contributing to poorer, less efficient work performances, as inadequate sleep has been linked to slower information processing, impaired cognition, and reduced task performance.⁷ Given this, corporate executives who meet their sleep needs may demonstrate improved cardiometabolic health profiles, cognitive function, coping skills and work efficiency.⁸ Despite the compelling evidence describing the benefits of good sleep health, the impact it may have on effective leadership and success of organisations is often disregarded. Likewise, the current absence of sleep health screening and management thereof within workplace health promotion programmes further emphasise the urgency to address sleep health within the corporate work environment.

1.2 The corporate work environment and workplace culture

Corporate executives who lead large organisations are subject to stressors which include economic pressures, competition, long working hours and constant decision-making.⁹ Given this, the high-pressure corporate lifestyle can exacerbate an unhealthy work-life balance leaving little time for self-care and leisure activities. The normalisation of corporate work life may also be impacted by the company's workplace culture, which has been defined as "prevailing values, attitudes, beliefs, artifacts, and behaviours that contribute to a company's sense of order, continuity, and commitment".¹⁰ Collectively, the excessive work

demands and business expectations characteristic of the corporate work environment may lead to detrimental outcomes such as occupational burnout, anxiety, depression and cardiovascular disease.¹¹

1.3 Workplace health promotion programmes

Workplace health promotion programmes are designed to protect the health and wellbeing of employees by preventing or delaying the onset of disease, through both screening of relevant risk factors and encouraging lifestyle and behaviour changes aimed at optimising both physical and mental health.

The screening of CMD risk factors is facilitated through health risk assessments (HRAs) and biometric screening, which in turn allow for risk stratification based on the number of health risks identified. Correspondingly, as the number of risk factors increase, so does the risk for developing CMD.

Common clinical measurements assessed during biometric screening include the measurement of body height and body weight (to calculate body mass index), blood pressure, blood glucose and blood lipid levels, whereas self-reported lifestyle-related variables addressed in questionnaires include physical activity, smoking status, alcohol intake, fruit and vegetable intake, and recently some HRAs have included sleep duration. Together, these data are used to create a health risk profile which may guide workplace health management strategies.

While the workplace provides an ideal setting for health promotion, much of the literature in this regard typically involves diverse employee cohorts comprising various occupational levels.^{12,13} Consequently, the health risks of corporate executives whose health may have far reaching consequence such as carrying economic and organisational costs are frequently overlooked.¹⁴ Likewise, workplace health promotion programmes targeted at corporate executives are seldom described in the literature.

1.4 Cardiometabolic disease

Given that raised blood pressure, obesity, high blood glucose levels and dyslipidaemia are shared risk factors for CVD and diabetes, these NCDs have been collectively referred to as CMDs. Further, there is evidence to show that a large proportion of NCD-related deaths in working adults are attributed to CMDs, and that since 2010, the working population has experienced a sharp increase in CMDs as a consequence of increasing obesity prevalence and its metabolic consequences.¹⁵

The clustering of CMD markers which provide an indication of CMD risk, include both objectively measured clinical risk factors and self-reported lifestyle-related factors as summarised in Table 1.1.

1.4.1 Clinical Risk factors for cardiometabolic disease

The criteria for identifying those at increased risk for CMD within this thesis are based on the guidelines proposed by The Adult Treatment Panel III (ATP III).¹⁶ These include waist circumference, fasting blood measurements (HDL-cholesterol, triglycerides, blood glucose) and resting blood pressure. In addition, body mass index (BMI) was analysed as an outcome variable for this thesis as it provides an estimation of body fatness. The defining cut-points for these clinical risk factors for CMD are presented in Table 1.1.

The CMD risk score is another proposed method to assess CMD risk, particularly for research purposes. It is a continuous composite score¹⁷ that sums up the z-standardized scores of multiple clinical CMD risk factors (e.g. HDL cholesterol, fasting insulin, fasting plasma glucose, fasting triglycerides, body mass index, waist circumference, systolic and diastolic blood pressure) such that higher risk scores indicate a higher degree of CMD risk.¹⁸ A more detailed methodology of this validated CMD risk score is described in Chapters 3 to 5 of this thesis.

Table 1.1 Defining criteria for elevated or ‘at risk’ clinical and lifestyle risk factors for cardiometabolic disease.

Clinical	
Body mass index (kg/m²)	≥25.0
Waist circumference (cm)	≥102 (men), ≥88 (women)
High-density lipoprotein cholesterol (mmol/L)	<1.0 (men), <1.3 (women)
Triglycerides (mmol/L)	≥1.7 or on lipid medication
Blood pressure (mmHg)	≥130/85 or on anti-hypertensive medication
Fasting blood glucose (mmol/L)	≥5.6 or on diabetes medication
Lifestyle	
Smoking status	Current smoker
Alcohol intake (drinks/week)	>14
Physical activity (min/week)	<150
Sleep duration (h/night)	<7 or >9

1.4.2 Lifestyle-related risk factors for cardiometabolic disease

Modifiable lifestyle factors, such as smoking, excessive alcohol consumption, physical inactivity and an unhealthy diet, are well-established CMD risk factors.^{19,20} The dietary component of HRAs is more tedious to measure, as one's diet may influence CMD risk indirectly via changes in blood cholesterol, blood pressure, and body weight or directly, through an effect independent of these other factors.²¹ For example, a recent meta-analysis demonstrated that the dietary component of lifestyle-related health risk factors may be reported in various ways such as adherence to the Mediterranean diet, the frequency or number of daily servings of fruits and/or vegetables and measuring the amount of calories consumed.²² Considering this, the dietary health risk factor for CMD risk is beyond the scope of this thesis and therefore omitted from Table 1.1. Nevertheless, the defining criteria for the remaining lifestyle factors are based on the recommended guidelines for adults proposed by respective organisations such as the World Health Organisation (WHO), The American College of Sports Medicine (ACSM), the Centers of Disease Control (CDC) and The National Sleep Foundation (NSF).²³⁻²⁶ Amongst these, sleep duration, which has recently emerged as a novel independent risk factor,²⁷ is often excluded when assessing CMD risk.²⁸

1.5 Sleep

Sleep is a basic human need and is essential for good health and quality of life. A simplified definition of sleep is that it is a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment.²⁹ It is different to other states of altered "consciousness," such as coma, in that it is easily reversible and self-regulating.³⁰

1.5.1 Sleep architecture

Sleep architecture refers to the basic structural organisation of sleep. There are two phases of sleep, non-rapid eye-movement (NREM) and rapid eye-movement (REM) sleep. Each phase has unique characteristics including variations in brain wave patterns, eye movements, and muscle tone. During normal nocturnal sleep in adults, there is an orderly progression from wakefulness to sleep onset, through the three NREM stages (N1 to N3) followed by a bout of REM sleep. This cyclical switching between NREM and REM sleep is referred to as a sleep cycle, which lasts ± 90 –110 minutes.³¹ Healthy sleep typically comprises 4–5 sleep cycles per night,³² with the percentage of REM sleep in each cycle generally increasing and that of N3 decreasing as the night progresses.

Furthermore, NREM sleep accounts for $\pm 75\%$ of sleep time in adults and is divided into three stages. NREM stage 1 (N1) sleep marks the transition from wakefulness to sleep, is regarded as 'light' sleep, and usually comprises 3-5% of total sleep time. NREM stage 2 (N2) sleep comprises the largest proportion of sleep time (45–55%), whereas NREM stage 3 (N3) sleep, also known as 'slow wave' or 'deep' sleep, makes up $\pm 20\%$ of

total sleep time.³³ Much of the body's physical restoration occurs during N3 sleep through the release of hormones (e.g. growth hormone) and promotes tissue growth and repair.^{34,35}

Subsequently, REM sleep accounts for 20–25% of total sleep time with the first bout generally occurring approximately 90 minutes after having fallen asleep. During this stage, the brain is highly active. Typical characteristics of REM sleep include bursts of rapid movements of the eyes, loss of motor tone, increased use of oxygen in the brain, amplified and variable pulse and blood pressure. It is also considered to be the stage in which dreaming predominantly takes place, and the brain completes complex tasks, such as processing and regulating emotions, organising information, processing new learning, and storing long-term memories.³⁶

1.5.2 Sleep regulation

Sleep is thought to be regulated by the interplay of two major processes, one that promotes sleep (Process S), driven by how long one has been awake, and one that maintains wakefulness (Process C), driven by the circadian pacemaker in the brain.³⁷ As such, the interaction between these homeostatic and circadian processes ensures that we sleep at night and maintain wakefulness during the day.³⁸

The homeostatic drive for sleep (Process S) represents the 'need for sleep' or sleep propensity, and is sensed as sleep pressure, which accumulates during wakefulness, peaking just before bedtime at night and dissipating during sleep (Figure 1.1). Additionally, the strength of this process is dependent upon the amount of time awake since the last sleep period. The dashed line in Figure 1.1 represents potential increases in pressure to sleep, which will continue to build if sleep does not occur. For this reason, the longer one stays awake, the stronger the homeostatic drive and one's need to sleep becomes.

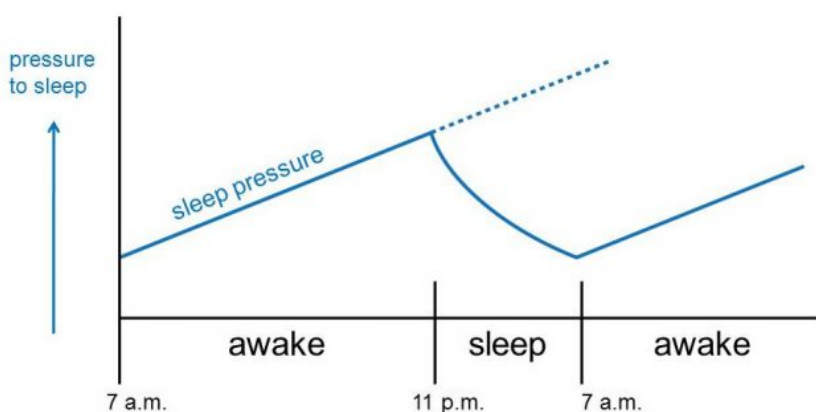


Figure 1.1. Homeostatic drive for sleep (Process S) increases one's pressure to sleep with prolonged wakefulness and declines during sleep. (Source: <https://www.cdc.gov/niosh/>)

Process C is also referred to as the circadian process as it is involved in the timing and organisation of sleep and wakefulness. This is achieved by driving wakefulness as sleep pressure accumulates during the day, and

removing the drive for wakefulness during the natural, nocturnal sleep period (Figure 1.2). Further, there is evidence to suggest that disruptions to both processes are associated with CMD.³⁹ Given this, the impact of sleep regularity and timing on CMD is discussed in Section 1.8.

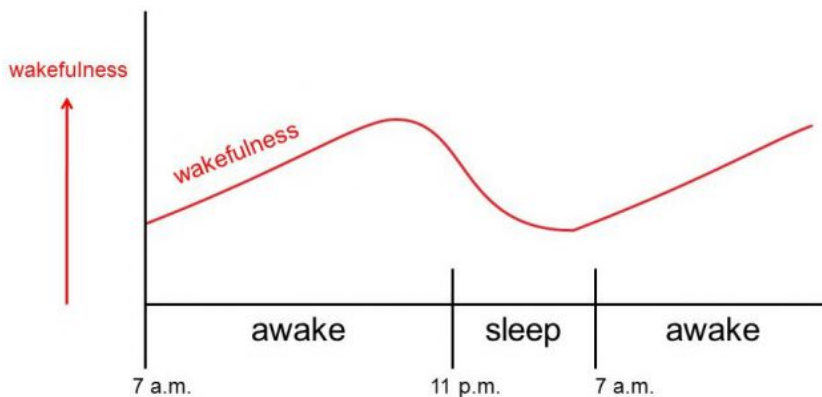


Figure 1.2. Circadian process (Process C) provides a drive for wakefulness at specific phases of the 24 h cycle. (Source: <https://www.cdc.gov/niosh/>)

1.5.3 Circadian rhythms

Circadian rhythms are 24 h patterns in cellular, physiological and behavioural processes that are regulated by the master circadian pacemaker in the suprachiasmatic nuclei (SCN) within the hypothalamus. Since innate circadian rhythms have periods slightly longer than 24 h, daily adjustments are required to ensure these rhythms are synchronised to the external 24 h light/dark cycle via a process referred to as ‘entrainment’.⁴⁰ Entrainment is driven by external time cues, referred to as *zeitgebers* (‘time-givers’) such as light, activity, and meal timing, of which light is the strongest.⁴¹ Owing to this, the SCN is responsible for receiving and integrating information from the *zeitgebers* to ensure alignment of both central and peripheral clocks, which in turn coordinate functioning of all organs and systems to one another as well as the physical environment.

1.5.4 Chronotype

Chronotype is a behavioural reflection of circadian rhythmicity. It is typically defined as individual variation in the preferred timing of the sleep–wake cycle and is dependent on genetic, environmental, and age-related factors.⁴² Morning types (‘morning larks’) have a tendency to go to sleep and wake up early, with a preference for morning cognitive, physical and feeding activities. In contrast, evening types (‘night owls’), who prefer to go to sleep and wake up later, tend to prefer conducting activities in the evening or at night.⁴³ One of the original validated questionnaires that was developed to assess chronotype was created by Horne and Östberg in 1976⁴⁴ and is described briefly in Table 1.2 and more comprehensively in Chapter 5.

1.6 Measuring sleep

1.6.1 Objective measures

Physiologically, the gold standard technique for assessment of sleep and wake states is polysomnography (PSG). To conduct a PSG, numerous non-invasive sensors are attached to a participant to record brain activity (electroencephalogram [EEG]), eye movements, submental muscle tone, leg movements, and heart activity (electrocardiogram [ECG]).⁴⁵ This technique is useful in clinical practice to diagnose some sleep disorders, or in research settings where EEG activity is required, yet it is costly, requires trained sleep technicians, and does not provide information around habitual sleep characteristics. Owing to this, actigraphy was chosen as a means to objectively measure habitual home-based sleep in Chapter 5 of this thesis.

Actigraphy is a method used to monitor habitual sleep-wake patterns using compact, lightweight, computerised wrist-worn accelerometers.⁴⁶ Compared to PSG, actigraphy offers the advantages of assessing habitual sleep patterns in the home environment over longer time periods, being easier to use, more affordable, non-invasive and user-friendly. Accuracy of actigraphy-derived sleep is enhanced through the simultaneous use of a sleep diary, which allows confirmation of nocturnal sleep periods and daytime naps. Further, it is considered the most reliable method for objective assessment of sleep patterns in epidemiologic studies and has been validated in a broad range of clinical and non-clinical adult populations.⁴⁷ The methodology of actigraphy, and the outcome variables it is capable of collecting are described in Chapter 5.

1.6.2 Subjective measures

Preliminary evaluation of sleep health has traditionally been conducted with the help of questionnaires.⁴⁸ The main advantage of this method of data collection is its affordability and accessibility to patients and research participants. The validated questionnaires that were used to describe the sleep profiles of the participants in this thesis are summarised in Table 1.2.

Table 1.2. Summary of subjective measurement tools used in this thesis.

Assessment tool	Description	Score interpretation
Pittsburgh Sleep Quality Index (PSQI)⁴⁹	Assesses sleep quality over a one month time interval. Consists of 19 items which generate seven subcomponent scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction.	The sum of scores for the seven subcomponents yields a global PSQI score ranging from 0-28, with lower scores indicating better sleep quality. A global PSQI score greater than 5 is regarded as poor sleep quality.
Insomnia Severity Index (ISI)⁵⁰	Assesses insomnia symptom severity over a two week time interval. Consists of seven items from which the scores are summed to get a total ISI score. Each item is rated on a 0-4 scale.	ISI scores range from 0-28, with higher scores indicating a greater insomnia symptom severity. ISI scores can also be categorically interpreted as: no clinically significant insomnia (0-7), subthreshold insomnia (8-14), clinical insomnia of moderate severity (15-21) and severe clinical insomnia (22-28).
Epworth Sleepiness Scale (ESS)⁵¹	Assesses daytime sleepiness in recent times and consists of eight questions around the probability of unintentionally falling asleep during the day in various scenarios. Each item is rated on a scale of increasing probability from 0 to 3	Scores for each item are summed to produce an ESS score ranging from 0-24, with higher scores indicating more daytime sleepiness. Excessive daytime sleepiness (EDS) is defined as ESS scores >10.
Morningness Eveningness Questionnaire (HÖ MEQ)⁴⁴	Probes an individual's preferred times for activities such as working, exercising, eating or sleeping based on 19 questions.	Scores range from 16- 86, with lower scores indicating eveningness and higher scores morningness. Accordingly, individuals are placed into one of five chronotype categories: definite evening-type (DET), moderate evening-type (MET), neither-type (NT), moderate morning-type (MMT), and definite morning-type (DMT).

1.7 Sleep Health

The most common sleep characteristic to describe sleep health has been sleep duration. The American Academy of Sleep Medicine (AASM) and the Sleep Research Society (SRS) recommend that adults sleep 7 hours or more per night on a regular basis,⁵² while the National Sleep Foundation (NSF) in the United States recommends 7 to 9 hours of sleep per night for adults²⁶ to promote optimal physical and mental health. In more recent times, the evolving concept of 'sleep health' presents a more holistic understanding of sleep, including multiple domains of sleep characteristics.

In addition to adequate sleep duration, sleep health may be characterised by sleep regularity, appropriate timing, high sleep efficiency, subjective satisfaction, and sustained alertness during the day⁵³ thus making it a multidimensional construct. Owing to this, the study of multidimensional sleep, instead of single sleep characteristics, and its associations with health outcomes, introduced the development of a composite sleep health score.^{54,55} While there is no single derivation for creating this composite score,^{56,57} it remains an emerging prospect for studying multidimensional sleep health and how it impacts on health and well-being.

1.8 Sleep and cardiometabolic disease

In the general population, epidemiological research has reported the association between sleep duration, quality, regularity and timing with CMD risk⁵⁸⁻⁶² yet these studies seldom differentiate between the sleep of retired individuals or the unemployed, which may result in confounding as their sleep patterns differ from that of working adults.

To the best of our knowledge, there are only a few studies exploring the association between sleep duration, sleep quality and CMD in employed adults, but most often focus it drawn to shift work.⁶³ For example, short sleep duration and poor sleep quality has consistently been associated with the cluster of CMD risk factors which include poor glucose, lipid and blood pressure profiles, and obesity, specifically abdominal obesity, resulting in a higher CMD risk score and among shift workers.^{18,64-66}

Adding to this, there is growing evidence that the regularity and timing of the sleep-wake cycle are both associated with CMD risk.⁶⁷ Regularity refers to the degree to which sleep occurs at approximately the same time each night whereas timing refers to when in the 24 h period sleep takes place. Both of these variables relate to circadian rhythmicity, and it is thought that the negative consequences of irregular or mistimed sleep affect CMD risk through circadian rhythm disruption.^{68,69}

For example, in a review examining night-to-night variability in sleep patterns in relation to the metabolic syndrome, type 2 diabetes and their risk factors, sleep variability was associated with increased risk for adiposity, raised glucose levels and the metabolic syndrome.⁶⁷ With respect to sleep timing, a large

observational study of adults found that later sleep–wake timing was associated with greater estimated insulin resistance and higher systolic and diastolic blood pressure measures.⁷⁰

While irregular sleep–wake patterns and timing observed in rotating shift workers has been linked to elevated rates of CMD,⁷¹ this extreme example of sleep irregularity may not apply to corporate executives. Despite having more regular work schedules, corporate executives may still have variability in sleep patterns between working and non-working days.

1.9 The sleep of corporate executives

Given that the sleep of corporate executives may be compromised by high work pressure, and that their workplace culture may de-prioritise the importance of sleep health,^{6,72-74} there is a great need to uncover the sleep-cardiometabolic health relationship in this occupation group. While statistics on the sleep duration of working adults show that more than a third of employees sleep less than 7 h per night,⁷⁵ the heterogeneity of these data limit the ability to elucidate the sleep of employees at corporate executive level.

Nonetheless, a survey exploring the sleep patterns, beliefs and attitudes of corporate executives revealed that they were sleep deprived, reported poor sleep quality, and that their beliefs and attitudes toward sleep were unhealthy.⁷⁴ Specifically, employees who reported an average of 6.6 h of sleep per night believed that getting less sleep related to career success, and that successful leaders slept less than the average worker.⁷⁴ The lack of longitudinal studies and objective sleep measures to support such findings provide the rationale for research to better understand the relationship between habitual sleep and CMD risk among a unique sector of the workforce.

1.10 Sleep in workplace health promotion programmes

Despite the fact that healthy sleep attributes have been shown to be inversely associated with CMD and its risk factors,⁶⁰ sleep remains an underappreciated health metric for the improvement or prevention of CMD.^{76,77} Relative to the traditional risk factors of CMD (smoking, alcohol, physical activity, diet), the inclusion of sleep within workplace health promotion programmes has been the least prominent.

There is therefore a specific opportunity to embed sleep health in workplace health promotion programmes. Achieving this has the potential to not only prevent CMD, but to improve work performance, particularly as there are data suggesting that employees get less sleep than they need to function well at work due to extended working hours, taking work home, working multiple jobs, and having long commute time to work.^{6,78}

When assessed by occupational health service providers, sleep most often is addressed indirectly through fatigue risk assessments and is typically targeted toward manual labourers or shift working employees.^{79,80}

Moreover, in instances where sleep is considered for HRAs, it is limited to a single question related to sleep duration. Consequently, additional components of sleep such as quality and timing are missed.

1.11 Thesis outline

The overall aim of this thesis is to describe the sleep characteristics of corporate executives in relation to CMD risk and further explore longitudinal associations between sleep attributes and CMD risk. Accordingly, this thesis addresses five research questions to support the overall aim:

1. Is there an association between sleep duration and mortality in employed adults?

A systematic review and meta-analysis was conducted to assess the association between sleep duration and all-cause and cardiovascular disease mortality (Chapter 2). The findings of this chapter provide evidence to show the sleep-mortality relationship in employed adults younger than 65 years within the urban workforce. As such, this chapter lays the groundwork and provides rationale to focus attention to the sleep of corporate executives in subsequent chapters.

2. Is there an association between self-reported sleep duration and cardiometabolic disease risk in corporate executives?

Chapter 3 identified correlates of sleep duration in corporate executives, and using mediation analyses, was able to identify the mediators contributing to the sleep duration-CMD risk relationship. Given that this chapter was cross-sectional in nature, it would be valuable to elaborate on the established sleep-CMD risk association by proceeding to a longitudinal research study design.

3. Is there a longitudinal association between sleep duration and cardiometabolic disease risk in corporate executives?

By making use of corporate HRA data collected over a minimum of three years, Chapter 4 used a mixed model analysis to explore the relationships between self-reported sleep duration and CMD risk over time. Having used self-reported sleep data in Chapters 3 and 4, it was necessary to add an objective measure of sleep to gain a better understanding of the sleep profiles of corporate executives.

4. What is the association between objective sleep measures and cardiometabolic disease risk in corporate executives?

Chapter 5 utilised actigraphy in combination with subjective measures of sleep, enabling a wide array of sleep characteristics to be assessed among corporate executives to address this research question. The findings indicating that the sleep of corporate executives may increase their vulnerability to CMD (Chapters 3 – 5) emphasises the opportunity for workplace health promotion programmes to address sleep health.

5. What should be considered when offering a sleep health programme within the workplace?

A qualitative approach was used in Chapter 6 to explore perspectives of both occupational medicine specialists and corporate executives regarding barriers and facilitators to participating in a sleep health programme. Additionally, the key components of an ideal sleep health programme are indicated and collectively offer the evidence to present the recommendations in this chapter.

In summary, Figure 1.3 provides a chapter-specific overview for these research questions illustrating three overarching areas of occupational sleep, namely overall employees' sleep, corporate executives' sleep, and the opportunity for sleep health programmes.

EMPLOYEES' SLEEP	CORPORATE EXECUTIVE SLEEP			OPPORTUNITY FOR SLEEP HEALTH PROGRAMMES
<p>QUESTION 1 <i>(Employed adults)</i></p> <p>Is there an association between sleep duration and mortality in employed adults?</p> <p>CHAPTER 2</p>	<p>QUESTION 2 <i>(Self-reported)</i></p> <p>Is there an association between sleep duration and cardiometabolic disease risk in corporate executives?</p> <p>CHAPTER 3</p>	<p>QUESTION 3 <i>(Self-reported)</i></p> <p>Is there a longitudinal association between sleep duration and cardiometabolic disease risk in corporate executives?</p> <p>CHAPTER 4</p>	<p>QUESTION 4 <i>(Objectively-measured)</i></p> <p>What is the association between objective sleep measures and cardiometabolic disease risk in corporate executives?</p> <p>CHAPTER 5</p>	<p>QUESTION 5 <i>(Qualitative assessment)</i></p> <p>What should be considered when offering a workplace sleep health programme?</p> <p>CHAPTER 6</p>

Figure 1.3 Visual presentation of the research questions presented in each thesis chapter.

Chapter 2

Associations between self-reported sleep duration and mortality in employed cohorts: Systematic review and meta-analysis

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Abstract

Objective

Studies have shown associations between sleep duration and mortality in the general population, which encompasses diversity in employment status, age and community settings. Since sleep patterns of employed individuals may differ to those of their unemployed counterparts, the nature of their sleep-mortality relationship may vary. We therefore investigated the association between self-reported sleep duration and all-cause mortality (ACM) or cardiovascular disease mortality (CVDM) in employed individuals.

Data sources

Based on Preferred Reporting Items for Systematic Reviews and Meta-Analyses, searches between January 1990 and May 2020 were conducted in PubMed, Web of Science and Scopus. Inclusion/exclusion criteria: Included were prospective cohort studies of 18–64-year-old disease-free employed persons with sleep duration measured at baseline, and cause of death recorded prospectively as the outcome. Grey literature, case-control or intervention design studies were excluded.

Data Extraction

Characteristics of the studies, participants, and study outcomes were extracted. The quality and risk of bias were assessed using the Newcastle-Ottawa Scale.

Data synthesis

The pooled relative risks (RR) with 95% confidence intervals (CI) were obtained with a random-effects model and results presented as forest plots. Heterogeneity and sensitivity analysis were assessed.

Results

Shorter sleep duration (≤ 6 h) was associated with a higher risk for (ACM) (RR: 1.16, 95% CI: 1.11-1.22) and CVDM (RR: 1.26, 95% CI: 1.12-1.41), with no significant heterogeneity. The association between longer sleep (≥ 8 h) and ACM (RR: 1.18, 95% CI: 1.12-1.23, $P < 0.001$) needs to be interpreted cautiously owing to high heterogeneity ($I^2 = 86.0\%$, $P < 0.001$).

Conclusion

Interventions and education programs targeting sleep health in the workplace may be warranted, based on our findings that employed individuals who report shorter sleep appear to have a higher risk for ACM and CVDM.

Keywords: sleep quantity; short sleep; heart disease; employed; workplace

2.1 Objective

Non-communicable diseases (NCDs) account for about 71% of deaths globally among working-aged adults (35–70 y), with cardiovascular disease (CVD) being the leading cause of death (31%).¹ Modifiable risk factors for NCDs include an unhealthy diet, physical inactivity, alcohol use and tobacco smoking. The World Health Organisation's (WHO) global action plan aims to manage these risks so that NCD-related deaths can be reduced by 25% by the year 2025.⁸¹ The workplace has been identified as a key setting for public health action, in which diet and physical activity interventions are primarily used to address NCD risk.⁸²

Suboptimal sleep (poor quality, insufficient duration and/or mis-timed) has emerged as an important modifiable risk factor for cardiovascular and metabolic diseases.^{6,82-86} Notwithstanding the importance of sleep timing and quality, we will focus on sleep duration in this study given that much of the prospective work relating sleep to mortality relies on sleep duration as an outcome variable. Guidelines have been published recommending that adults attain 7-9 h²⁶ or at least 7 h⁵² of sleep per night for optimal physical and mental health. Acknowledging that not all studies conclusively observe associations between sleep duration and health outcomes, longitudinal evidence from systematic reviews and meta-analyses indicates that individuals with shorter and longer sleep durations are more likely to experience poorer health outcomes compared to those clustered around the group median (typically 7-8 h).⁸⁷⁻⁹¹ While organisations such as the American Heart Association and the Center for Disease Control and Prevention promote healthy sleep awareness,^{60,92} sleep is yet to be included as one of the WHO's targeted modifiable risk factors that could enhance health and prevent NCDs.⁸¹ Thus it is unsurprising that although workplace health programmes, typically delivered in urban settings, are becoming more commonplace, sleep improvement rarely feature as an intervention.

Working hours, commuting time and even the nature of urban employment may impact sleep. Work has been shown to be the dominant waking activity in employees who sleep ≤ 6 h per night.⁹³ Adults with longer working hours are more likely to have shorter sleep durations^{6,83} such that for every additional hour spent working beyond the standard eight-hour day, sleep is shortened by about two hours.⁸³ Commuters traveling more than 75 minutes may also sleep less compared to those whose commute is under 45 minutes^{85, 86} and one study showed that for every hour of commuting, sleep was shortened by 15 minutes.⁷⁸ Furthermore, employees in the manufacturing sector, routine and semi-routine occupations (e.g. cleaners, receptionists) and in managerial roles have been shown to sleep less than those in other occupational groups.^{85,6} Therefore, extended working hours and fixed work schedules that demand consistent wake-times may largely contribute to shorter sleep duration in employed adults. On the other hand, unemployment has been linked to at least a two-fold increase in longer sleep duration (>9 h) compared to those sleeping 7-8 h. These studies postulated that the sleeping patterns of unemployed individuals are not impinged upon by a fixed work schedule; thus having the opportunity to sleep more than employed individuals.^{94,95}

One might speculate that employees working longer hours might sleep less and be at increased risk for CVD, all-cause mortality (ACM) or CVD mortality (CVDM). In a meta-analysis including data from 24 occupational cohort studies, employees working >54 h per week had a 30% higher risk for stroke and a 13% higher risk for CVD compared to those who worked 35-40 h.⁹⁶ Similarly, the incidence of diabetes was higher among women working >45 h per week compared to those working 35-40 h.⁹⁷ One study has shown a J-shaped relationship between sleep duration and number of health risk factors in employees, with those reporting ≤ 5 h per night likely to present with the most risk factors, and those sleeping 7-8 h per night with the fewest risk factors.⁹⁸ It was interesting to note that employees sleeping ≥ 9 h per night were at greater risk for hypertension, depression, heart disease and cancer compared to those sleeping 7-8 h,⁹⁸ possibly suggesting a link between underlying disease and sleep duration. Since, to the best of our knowledge, no studies have included working hours, sleep duration and NCD, CVDM or ACM risk in their analyses, it is unclear whether shorter sleep associated with employment translates to increased risk for NCDs or mortality.

Although systematic reviews examining the literature pertaining to sleep duration and mortality have been published,⁹⁹⁻¹⁰⁴ none have presented the magnitude of these associations exclusively in employed populations. Since the sleep patterns of employed individuals may well differ from those of unemployed persons, which may in turn alter the nature of the sleep-CMD or sleep-mortality relationship, we see value in focusing exclusively on employed individuals, living in urban areas and younger than 65 y. Even though studies have shown that sleep health can be promoted in the workplace,^{7,105,106} some organisations feel there is insufficient evidence to make sleep a key component of their workplace policies and health promotion programmes. Data emanating from studies like ours may help advocate for the inclusion of sleep health in employee wellness programmes. Therefore, the aim of this systematic review and meta-analysis was to investigate the association between sleep duration and risk for all-cause mortality (ACM) and cardiovascular disease mortality (CVDM) in employed individuals.

2.2 Methods

Data sources

The study was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.¹⁰⁷ A standardised search strategy was developed *a priori* to identify published cohort studies that quantified the relationship between sleep duration and risk for ACM and CVDM in employed populations. Systematic literature searches were conducted in three databases: PubMed, Web of Science and Scopus. Details of the search terms can be found in Supplementary Table S2.1 (Appendix 1). Reference lists of included articles were also searched for eligible studies.

Inclusion and exclusion criteria

The following inclusion criteria were applied: (i) original articles published between January 1990 and May 2020 in English, (ii) prospective cohort study designs, (iii) employed individuals, 18-65 years and disease-free at baseline from urban settings (iv) sleep duration assessed at baseline, measured as either total sleep duration over a 24 h period (nocturnal and daytime sleep) or exclusively during night-time (nocturnal sleep), (v) cause of death recorded prospectively as outcome, (vi) follow-up of at least four years, and (vii) at least three sleep duration categories were reported, e.g. ≤ 6 h, 7-8 h and ≥ 9 h per night. CVDM was defined in accordance with ICD-9 codes 390 - 459 or ICD-10 codes I00–I99. If insufficient data were reported (e.g., absence of relative risk (RR), confidence intervals (CI) or number of cases) additional information was requested from the authors of studies eligible for inclusion. Studies were excluded if (i) a case-control or intervention design was used, (ii) relevant statistical data could not be accessed or (iii) the outcome was not adjusted for potential confounders (examples include: age, ethnicity, gender, marital status, education, social class (i.e. education level and job position,¹⁰⁸ or the individual's own current occupation¹⁰⁹), employment grade (i.e. administrative, professional and executive, or clerical job) lifestyle characteristics (e.g. smoking, physical activity, alcohol consumption), medical conditions (e.g. diabetes, depression, hypertension, cancer, obesity) or medications. Conference abstracts, other published abstracts, and grey literature (e.g., reports, white papers, academic theses or dissertations) were not included in this meta-analysis.

Data extraction

Three authors (DER, TKA and PRP) independently screened titles and abstracts, assessed full-texts for eligibility, and extracted the following data from included studies: (i) study characteristics (author name, publication date, cohort name, design, setting, sleep exposure, start date, end date and duration of follow-up period, inclusion and exclusion criteria, confounders), (ii) participant characteristics (sample size, age range, gender, race and/or ethnicity, disease status at baseline), and (iii) study outcomes (ACM, CVDM, total number of participants and number of deaths at each level of exposure, multivariate adjusted effect estimates, e.g. RR and 95% CIs).

DER, TKA and PRP assessed the quality and risk of bias of included studies using the Newcastle-Ottawa Scale (NOS).¹¹⁰ The NOS has three broad categories (patient selection, comparability of study groups, and assessment of the outcome) and provides a maximum total score of 9. Disagreements were resolved through discussion and consensus and NOS scores for each study are included in Table 2.1.

Table 2.1. Description of studies included in the meta-analysis

Author, Year	Country	Cohort	Year at baseline	Gender	Sample size	Deaths (ACM)	Deaths (CVDM)	Follow-up (y)	Mean age (range, y)	Quality score†	Sleep exposure assessment	Outcome assessment	Adjusted variables
Ferrie, 2007	England	Whitehall II	1985-1988	Men & Women	7729	292	89	4-8	35-55	7	Questionnaire: “How many hours of sleep do you have on an average week night?” Response options: ≤5 h, 6, 7, 8 and ≥9 h.	Death certificate	age, sex, marital status, employment grade, smoking status, PA, alcohol consumption, self-rated health, BMI, SBP, TC, physical illness, modified GHQ score, prevalent CHD
Garde, 2013	The Netherlands	Copenhagen Male Study	1970-1971	Men	4941	2663	587	30	48.9 (40-59)	8	Questionnaire: Participants reported their daily number of hours of sleep Response options: <6 h, 6-7, 8-9, and >9 h.	National Registers	age, BMI, SBP, DBP, diabetes, HT, physical fitness, alcohol use, smoking, LTPA, social class
Heslop, 2002	Scotland	Scottish workplaces	1970-3	Men & Women	5819 978	2303 262	1182 117	25	≤65	7	Questionnaire: “How many hours in 24 do you sleep?” Response option: Hours as a continuous variable	Death certificate	age, marital status, social class, risk

Patel, 2004	USA	Nurses' Health Study	1986	Women	82969	5409	1084	14	53.4 (30-55)	6	Questionnaire: "Indicate total hours of actual sleep in a 24-hour period." Response options: ≤5 h, 6 h, 7 h, 8 h, 9 h, 10 h, or ≥11 h.	National Death Index	age, smoking, alcohol consumption, PA, depression, snoring, BMI, history of cancer, CVD, HT, diabetes, and shift work
Rhee, 2012	South Korea	The Seoul Male Cohort Study	1993	Men	14533	990	-	15	49.5 (49-59)	7	Questionnaire: For sleep hours, respondents were asked to report the usual number of sleep hours, including naps. Response options: Hours as a continuous variable	Death certificate	age, education, smoking, alcohol, self-reported hypertension and diabetes

ACM: all-cause mortality; CVDM: cardiovascular disease mortality; PA: physical activity; BMI: body mass index; SBP: systolic blood pressure; TC: total cholesterol; GHQ: general health questionnaire; CHD: coronary heart disease; DBP: diastolic blood pressure; HT: hypertension; LTPA: leisure time physical activity; CVD: cardiovascular disease; † Newcastle-Ottawa Scale.

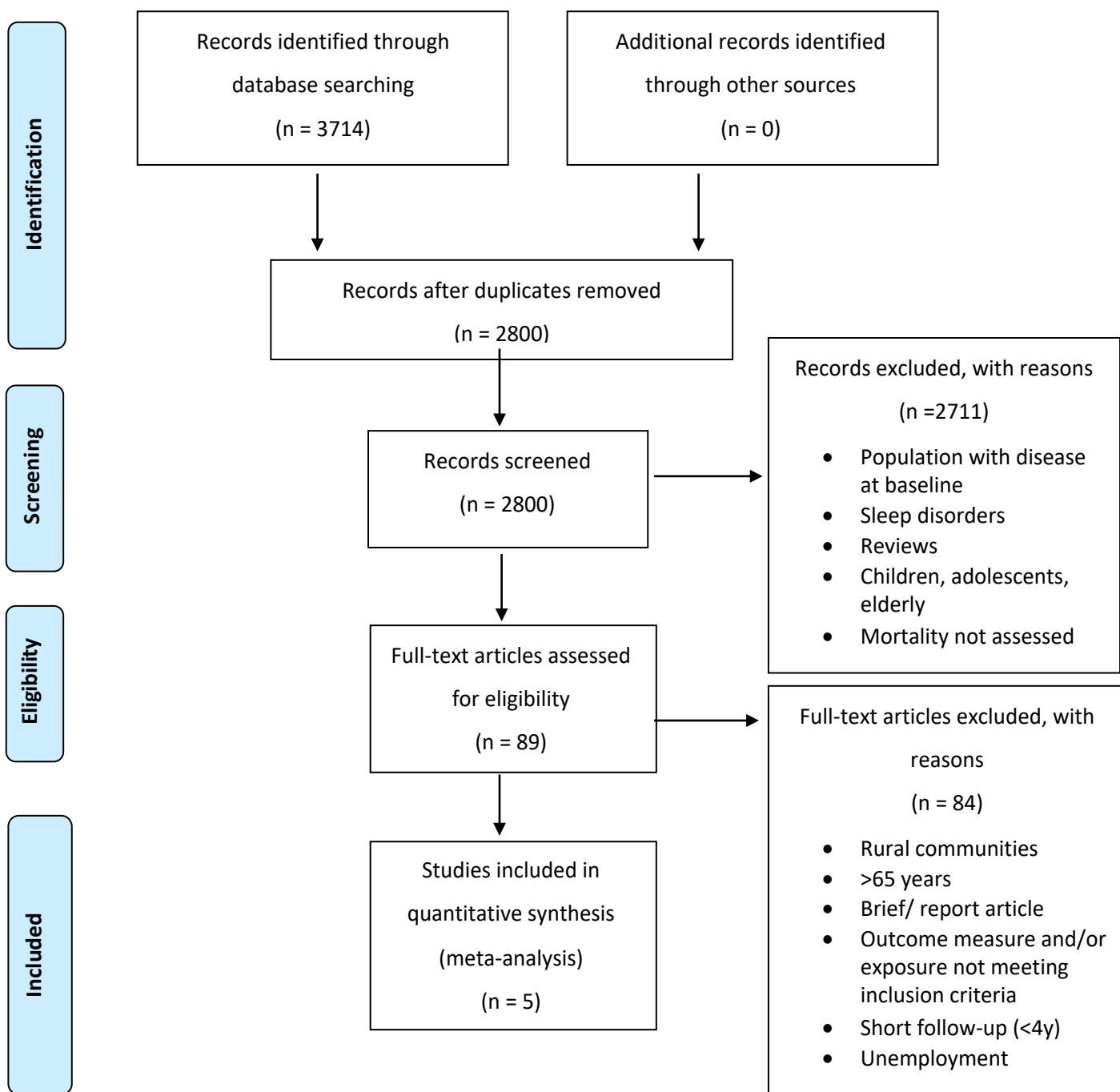


Figure 2.1 Flowchart of the records retrieved, screened and included in the systematic review

Data synthesis

The exposure variable used in this meta-analysis was self-reported sleep duration, measured at baseline of each included study. The manner in which each included study assessed sleep duration is defined in Table 2.1. The included studies varied in their sleep duration categories for which ACM or CVDM statistics were reported (Supplementary Table S2.2, Appendix 1), with three studies presenting statistics on sleep duration in three categories^{108,109,111} and two studies presenting data on five categories.^{112,113} Four of the five studies

used a reference sleep duration category in the range of 6-8 h (Ferrie, Garde, Heslop, Patel) while Rhee et al (2012) originally used their ≥ 8 h group as their reference category. For our meta-analysis, we used the ACM and CVDM statistics in their original form for the three studies reporting on three sleep categories (Garde, Heslop, Rhee), but we re-referenced the data for Rhee et al to their 6-7 h category, to better match the other studies.¹¹¹ For the two studies in which authors reported five sleep duration categories, we collapsed those categories less than the reference group into a single 'shorter sleep' category and those longer than the reference group into a single 'longer sleep' category (Supplementary Table S2.2, Appendix 1). Primary outcome variables were ACM and/or CVDM RR with 95% CIs per exposure. The most covariate-adjusted risk estimates were used in the meta-analysis. Men and women were considered independently among studies that reported gender-specific results. The pooled RRs with 95% CIs were obtained with a random-effects model and results presented as forest plots.

Heterogeneity among studies was assessed by the I^2 statistic, which represents the total variability that is attributed to between-study variability.^{114,115} When heterogeneity was significant, sensitivity analyses was performed to identify the influence of individual studies on the magnitude of the pooled estimate. This was achieved by omitting the studies with the greatest weight, one at a time, and examining the extent to which inferences were dependent on a particular study. Publication bias was not assessed as there were inadequate numbers of included studies to properly measure funnel plot asymmetry. Data were analysed using Stata (v15, StataCorp, Texas, USA). Significance was assumed for $P < 0.05$.

2.3 Results

Search results and study characteristics

After identification, exclusion of duplicates and screening, 89 full text articles were assessed for eligibility. Eighty-four studies failed to meet the inclusion criteria and were excluded, leaving five studies eligible for inclusion and analysis (Figure 2.1). The general characteristics of the included studies are presented in Table 1. The five studies analysed for ACM comprised 116 969 participants from five countries: two studies included only men,^{108,111} one included only women¹¹² and two reported data on men and women combined or separately.^{109,113} The three studies analysed for CVDM comprised 19 467 participants from three countries. One of these studies combined their analysis to include men and women,¹¹³ one study reported only on a male cohort¹⁰⁸ and another analysed men and women separately.¹⁰⁹ The studies used either death certificates or national death registries to determine number of deaths in each cohort. Follow-up years for ACM and CVDM ranged from 4-30 years, and total NOS scores ranged from 6-8 (average of 7) out of a possible score of 9. All studies originated from the USA, Europe or South Korea and used questionnaires to obtain self-reported 24 h^{108,109,111,112} or night-time¹¹³ sleep duration (h). The reference sleep duration ranged from

6-8 h, shorter sleep ranged from <6 to <7 h and longer sleep from ≥ 8 to >8 h (Supplementary Table S2.2, Appendix 1).

Sleep duration and all-cause mortality

In the pooled analysis, shorter sleep duration was associated with a 16% greater risk for ACM relative to the reference sleep duration ($P < 0.001$, Figure 2.2). Although long sleep duration appeared to be associated with an 18% greater risk for ACM compared to the 6-8 h sleep duration ($P < 0.001$, Figure 2.3), high heterogeneity ($I^2 = 86.0\%$, $P < 0.001$) and limited number of studies suggest that this pooled estimate was not robust and therefore unlikely to be associated with ACM.

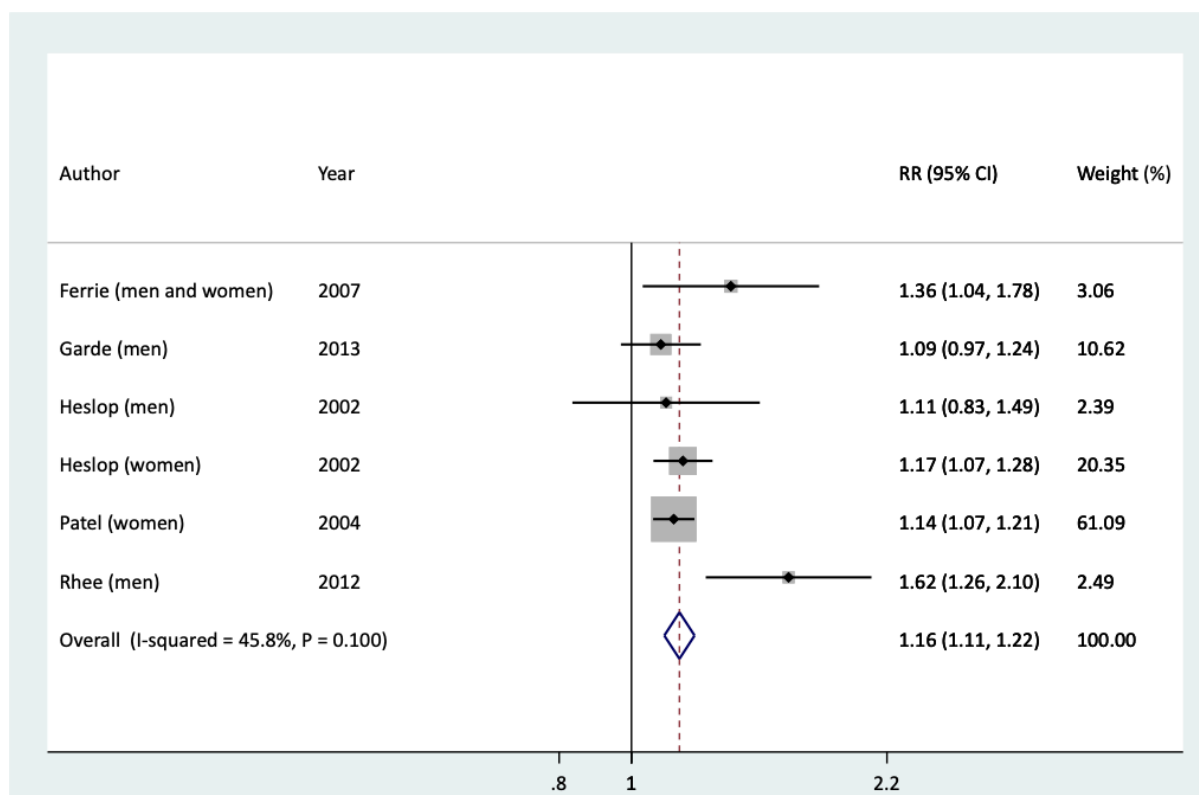


Figure 2.2. Forest plot of the risk of death (all-cause) associated with shorter sleep duration compared to the reference of 6-8 h in five population cohorts from four published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

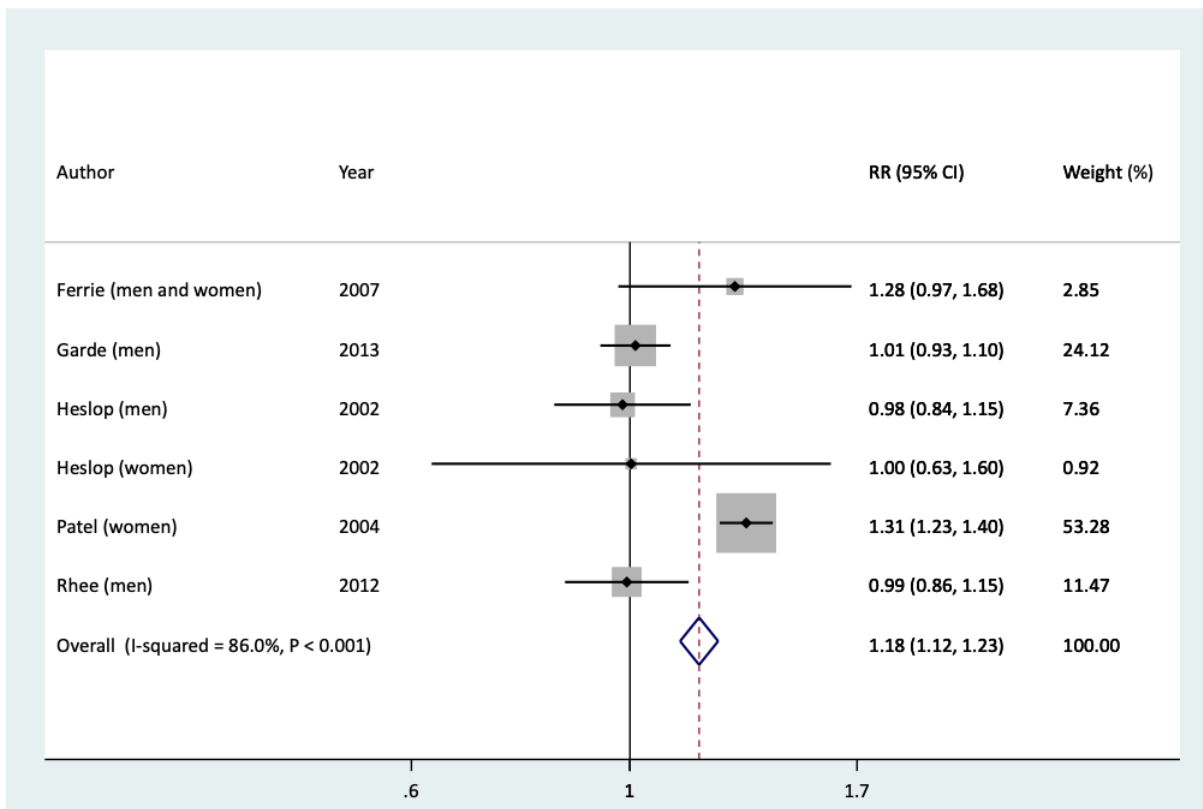


Figure 2.3. Forest plot of the risk of death (all-cause) associated with longer sleep duration compared to the reference of 6-8h in five population cohorts from four published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I² statistic.

Sleep duration and cardiovascular disease mortality

In the pooled analysis, shorter sleep duration was associated with a 26% greater risk for CVDM relative to the reference sleep duration (P<0.001, Figure 2.4) and heterogeneity was non-significant (45.8%, P=0.136). There was no association between longer sleep duration and risk for CVDM (P=0.193, Figure 2.5).

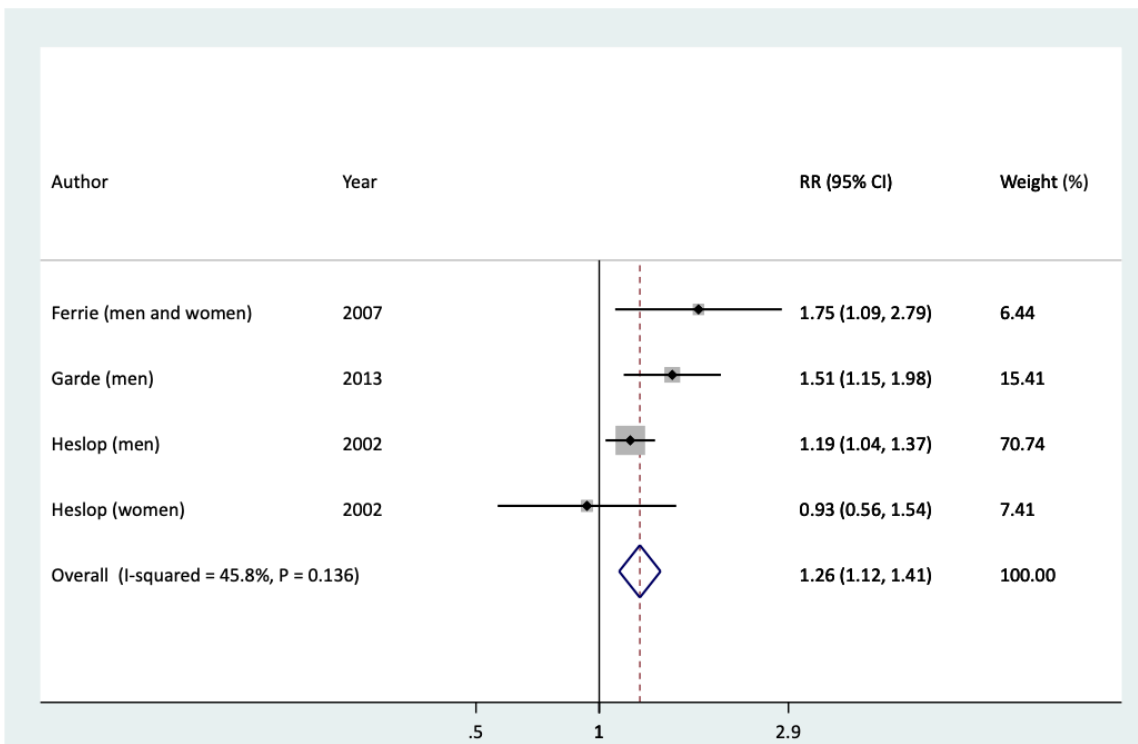


Figure 2.4. Forest plot of the risk of death (cardiovascular disease) associated with shorter sleep duration compared to the reference of 6-8 h in four population cohorts from three published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

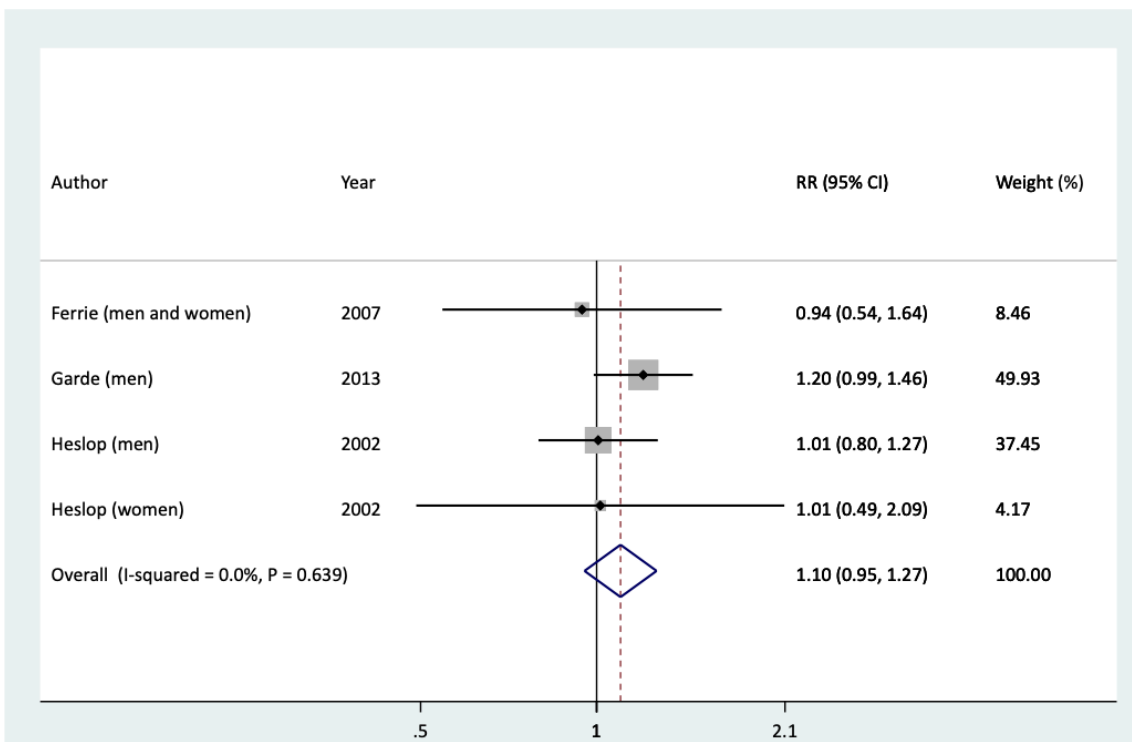


Figure 2.5. Forest plot of the risk of death (cardiovascular disease) associated with longer sleep duration compared to the reference of 6-8 h in four population cohorts from three published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

Sensitivity analyses

All-cause mortality

Sensitivity analyses were conducted by omitting the study with the largest weight to the pooled analyses. Removing Patel et al. (2004) (relative weight: 61.09%) from the analysis between shorter sleep duration and ACM increased the relative risk (RR: 1.19, 95% CI: 1.11-1.27, $P < 0.001$) with no heterogeneity. Patel et al. had the greatest relative weight (53.3%) for the longer sleep duration analysis and when omitted, relative risk for ACM (RR: 1.02, 95% CI: 0.96-1.09, $P = 0.558$) and heterogeneity decreased and were no longer significant.

Cardiovascular disease mortality

Sensitivity analysis was not conducted for the association between shorter sleep duration and CVDM, because heterogeneity was not significant ($I^2 = 45.8\%$, $P = 0.136$). Furthermore, sensitivity analysis was not needed for longer sleep duration as it was not associated with CVDM.

2.4 Discussion

To the best of our knowledge, this is the first systematic review to describe the association between self-reported sleep duration and ACM and CVDM in cohorts of employed persons younger than 65 years of age living in urban settings. The main finding was that compared to the reference sleep duration group, those reporting shorter sleep duration had a 16% greater risk for ACM and a 26% greater risk for CVDM.

The association between short sleep duration and ACM has previously been reported in the general population.^{99-101,103,104} Compared to such systematic reviews in which the same reference of 7-8 h was used, shorter sleep had lower relative risks than the present study. For example, one review reported a RR of 1.13 (95% CI: 1.09-1.17) for short sleep relative to a reference of 7-8 h, but had an extreme definition for short sleep duration (≤ 4 h).¹⁰⁴ In contrast, the present review showed that by sleeping as little as one hour less than the recommended sleep duration of 7-9 h, employees may have a significantly increased risk for ACM. In another review using the same reference range of 7-8 h, shorter sleep duration was significantly associated with ACM, but the RR of 1.07 (95% CI: 1.03-1.11) included only elderly individuals,¹⁰² while the present study excluded all cohorts older than 65 y.

Our finding that shorter, but not longer, sleep duration is associated with higher risk for CVDM contrasts with one previous study¹⁰² that showed a 43% increased risk for long sleep duration and CVDM but no association between short sleep and CVDM. Perhaps age contributes to these disparate findings since we report on working-aged adults (< 65 y) while Da Silva et al. (2016) reported on older individuals (> 60 y). Given the relationship between age and mortality, it is unsurprising that longer sleep duration is more strongly associated with ACM and CVDM in older cohorts.^{103,116,117}

Another study found an association between short sleep and CVDM in women, but not men, although both genders showed an association between long sleep and CVDM compared to 7 h. However, extreme sleep duration criteria of ≤ 4 h and ≥ 10 h were used, and participants were exclusively of Asian ethnicity.¹¹⁸ Socioeconomic status (SES) may affect the sleep duration and CVDM relationship. Studies report that disadvantaged groups who are influenced by factors such as poverty and unemployment are more likely to have shorter or longer sleep compared to the recommended 7-9 h.^{119,120} Grandner et al. (2010) hypothesized that sleep may be the mediator between SES and mortality.¹²¹ We were not able to account for SES in the present study, but future work on this topic would benefit from sub-analyses accounting for SES.

Potential pathways through which short sleep duration may increase risk for ACM include disruption of physiological regulatory pathways,² including those that regulate metabolism,^{122,123} immunity, inflammation, appetite and cardiovascular health.¹²⁴ These disruptions may manifest as CMD risk factors, such as hypertension, hyperglycaemia and obesity, all of which are associated with mortality.^{121,125,126}

The observation that longer sleep duration was associated with an 18% greater risk for ACM compared to an average of 6-8 h must be interpreted cautiously since there was high heterogeneity within the pooled estimate, heavily influenced by the Patel et al. (2004) study. Despite the high heterogeneity, the present findings show that the direction of the associations are predominantly toward increased risk for most of the analysed cohorts. Identifying heterogeneity can be helpful in interpreting and explaining study results, and for the planning of future studies. We therefore view this finding as exploratory, as presently there are insufficient eligible studies to investigate the sources of such wide variation through subgroup analyses.

One might speculate that the high heterogeneity may be attributed to factors such as gender or the definition of long sleep duration. For example, there were four times more women than men in this meta-analysis (women: $n = 83\,947$; men: $n = 19\,474$) for long sleep duration and ACM. We can only speculate that this disproportionate representation of gender may have contributed to the significantly increased risk for ACM among the long sleepers, as previous studies have shown that women sleeping longer than 9 h may have a higher risk for ACM compared to men.¹²⁷ In contrast, three other systematic reviews found no effect for gender on the sleep duration and ACM risk relationship,^{103,116,128} apart from a greater risk for obesity in longer sleeping women.¹¹⁶ Since long sleep duration may impair whole-body metabolism, increasing risk for obesity and T2DM,¹²⁹ the nature of risk may vary between men and women and requires further investigation.

Among the five included studies, longer sleep was defined as either ≥ 8 h or ≥ 9 h per night. Given that current sleep duration guidelines indicate that 7-9 h of sleep are recommended for optimal health,^{26,52} longer sleep defined as ≥ 8 h may not be long enough to adversely affect health. Explanations for why longer sleep may increase risk for ACM are debatable. Long sleep may be a response to poor sleep quality (i.e. fragmented sleep), high daytime sleepiness, elevated inflammatory markers or underlying conditions such as sleep disorders (e.g. obstructive sleep apnoea) or CVD.¹³⁰ One might hypothesise that perhaps individuals with longer sleep (≥ 9 h) unwittingly increased their sleep duration in response to a pre-existing, undiagnosed

underlying health condition predisposing them to premature mortality. In support of this hypothesis, it has been suggested that prolonged sleep might be a consequence of underlying diseases, frailty and worse health status, or be a part of the dying process.¹³¹

The results of this study emphasise the need for sleep health management in the workplace since the sleep health of employees may play as important a role on their future health outcomes as traditional CVD risk factors. Health screenings serve as a first step in workplace health promotion where the awareness of health risks can lead to lifestyle change for the management and prevention of CVD. Of interest is that most workplace health programmes focus on physical activity, smoking and diet, despite the evidence showing that employees averaging ≤ 6 h sleep were found to have a higher average number of health risks compared to those averaging 7-8 h.^{98,132} It is also clear that short sleep duration is associated with decreased workplace and public safety, sickness-related absenteeism,¹³³ lower information processing,¹³⁴ impaired cognition,¹³⁵ reduced task performance¹³⁶ and diminished job performance.¹³⁷

Given that many sequelae of insufficient sleep duration may adversely impact on cardiovascular health, safety and productivity of employees, employers have a vested interest in providing workplace health programmes to support the workforce in achieving healthy sleep. Since our findings, and other recent evidence show a high prevalence of shorter sleep duration in working adults,⁷⁵ there is an urgency to address sleep as a public health issue in workplace settings. Obstacles that prevent employees from having sufficient time to obtain the recommended 7-9 h of sleep, such as the duration and scheduling of work hours, and time taken to commute to work, are important focal points that may aid in the development and implementation of policies to improve employee sleep health. Strategies to promote sleep in the occupational health setting have been proposed, and include sleep education programmes that can be provided at workplaces; behavioural interventions to promote sleep; setting limits on the number of hours worked; where possible, encouraging teleworking to facilitate more time for sleep; and modifying workplace environmental characteristics such as lighting.¹³⁸⁻¹⁴⁰

Strengths

All studies included in this meta-analysis used a prospective study design, thus the misclassification of sleep duration attributable to recall bias was minimized. Most of the included studies were of relatively high quality, based on the NOS quality assessment (average of 7) and only included employed adults from urban settings.

This review provides rationale for the inclusion of further studies representative of employees exclusively, particularly of studies representing the workforce of the African continent, which appear to be absent in the literature. The risk of dying from NCDs is highest in low- and middle-income countries, especially in sub-Saharan Africa,¹⁴¹ therefore the impact of sleep duration in such countries, and its association with mortality are urgently needed to improve our knowledge of sleep health and disease in such populations.

Limitations

Although our findings are internally consistent with previous studies, we acknowledge several limitations. The primary limitation of these analyses is the small number of eligible studies available, which limited our statistical power. However, our study has highlighted a gap in the literature and underscores the importance of more sleep research needed in the field of occupational health. A second limitation is that in all but two studies,^{109,113} sleep duration was assessed at one point in time, and a single measurement of exposure may not capture the sustained effects of sleep duration over time, particularly if the reported period was shortly before death. Third, sleep duration in all studies was based on self-report and all but one study used 24 h sleep, thus making it difficult to distinguish time asleep from time-in-bed, estimating the number and duration of naps or capturing sleep quality data. The consistency of assessments (all self-reported questionnaires) and outcomes (all obtained from official national registries) across studies, however, attenuates heterogeneity which would otherwise occur due to differences in methods. We acknowledge the limitations of self-reported sleep duration as an outcome variable. It has, however, routinely been used in similar studies^{99,102-104,142} and shown to correlate well with more objective measures (sleep diaries, actigraphy, and polysomnography).^{112,143,144} It is also worth considering that from a translational-research and practical perspective, self-reported sleep duration provides a more cost-effective measure to implement in workplace health programmes and can be more widely utilized. Fourth, the included studies all presented mortality data based on different sleep duration categories (Supplementary Table S2) making it difficult to precisely match shorter, reference and longer sleep duration categories between studies. Finally, we recognise that a small number of occupations were represented by the participants in the reviewed studies, and the nature of the employment hours (e.g., night-time or rotational vs daytime shift work) was not recorded, thus limiting the generalisability of our results.

2.5 Conclusion

In this cohort of employed, urban individuals, self-reported shorter sleep was associated with a higher risk for both ACM and CVDM. Whether or not longer sleep is associated with a higher ACM risk, requires further investigations to unravel the mechanisms by which this association may contribute to increased mortality risk. Specifically, studies in which sleep is measured objectively and aspects of sleep quality are required.

In the meantime, interventions such as workplace health programmes which include education, screening and support to improve sleep health and ensure an adequate sleep opportunity on an individual basis are warranted. Conversations around aspects of employment, such as working hours, commuting and industry, which likely limit sleep duration or reduce sleep quality, should be held. Finally, sleep needs to be considered in the management and treatment of comorbid factors such as obesity, hypertension, and T2DM, all of which are known to increase the risk for mortality.

Chapter 3

Association between self-reported sleep duration and cardiometabolic risk in corporate executives

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Purpose

This cross-sectional study aimed to compare the association between self-reported sleep duration and cardiometabolic risk among men and women corporate executives and investigate potential lifestyle, work- and stress-related mediators thereof.

Methods

Self-reported sleep duration and lifestyle, occupational, psychological and measured anthropometrical, blood pressure (BP) and blood marker variables were obtained from health risk assessment data of 3583 corporate executives. Sex-stratified regression analyses investigated the relationships between occupational and psychological variables with self-reported sleep duration, and sleep duration with individual cardiometabolic risk factors. Mediation analyses investigated the effects of work, psychological and lifestyle factors on the relationships between self-reported sleep duration and cardiometabolic risk factors, as well as a continuous cardiometabolic risk score calculated from the sum of sex-stratified z-standardized scores of negative fasting serum HDL, and positive plasma Glu, serum TG, body mass index (BMI), waist circumference, systolic and diastolic BP.

Results

Longer work hours and work commute time, depression, anxiety and stress were associated with shorter sleep duration in both men and women (all $p < 0.05$). Shorter sleep duration was associated with higher BMI, larger waist circumference and greater cardiometabolic risk scores in both men and women (all $p < 0.05$), higher diastolic BP in men ($p < 0.05$) and lower HDL cholesterol in women ($p < 0.05$). Physical activity, working hours and stress significantly mediated the relationships between self-reported sleep duration and BMI, waist circumference, diastolic BP and cardiometabolic risk score in men only.

Conclusion

In these corporate executives, shorter self-reported sleep duration is associated with poorer psychological, occupational and cardiometabolic risk outcomes in both men and women. Given that physical activity, working hours and stress mediate this association among the men, the case for sleep health interventions in workplace health programmes is warranted.

Key words: employees, workplace, health risk assessment, sleep quantity, short sleep

3.1 Introduction

Cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) are among the most common chronic, lifestyle-related diseases worldwide, with the total number of deaths from these diseases combined increasing over the past decade (2007 – 2017) from 21.1% to 43.2%.¹⁴⁵ Conventional risk factors for cardiometabolic diseases (CMD) include lifestyle behaviours, such as lack of physical activity, tobacco smoking, alcohol consumption and unhealthy dietary habits. While sleep has long been associated with mental health,¹⁴⁶ sleep duration has emerged recently as a factor contributing to CMD.

More specifically, habitual short sleep duration (<7 h) has been associated with increased risk for obesity, T2DM, hypertension, CVD, the metabolic syndrome and early mortality.^{3,101,147-149} While current recommendations are 7–9 h sleep per night for optimal physical and mental health,^{26,52} recent findings suggest that time allotted to sleep may have gradually declined over the past decades in the general population.¹⁵⁰

Work and sleep have been identified as the main components of adult time use and longer working hours are associated with shorter sleep duration across all sociodemographic strata in employed adults.⁸³ A growing proportion of the working population has been found to curtail sleep or experience sleep fragmentation in response to factors such as prolonged working hours and job stress.^{63,73,83} This is particularly true among corporate executives who typically have complex work tasks, more cognitively demanding jobs, greater responsibilities and higher work demands, compared to blue collar workers.¹⁵¹ Insufficient sleep occurs when one's sleep need is not met, consequently impacting daytime alertness, performance, and health. Resultant daytime tiredness or fatigue may impair work performance by decreasing high-order cognitive processes required for decision-making pertinent to leadership roles.¹⁵²⁻¹⁵⁵

Self-reported short sleep duration (≤ 6 h per night) has been shown to vary by occupation, with the prevalence of short sleep duration being greatest for employees in managerial (40.5%), followed by those in transportation (37.1%) and manufacturing (34.8%) roles.⁶ Such literature gives insight into the sleep duration of occupational groups, and provides plausible correlates of shortened sleep in the broader population, but not in corporate executives for whom sleep appears to be jeopardised by longer work hours and high levels of stress.^{156,157}

Both working hours and stress contribute to shorter sleep,^{63,73,158} which in turn has been linked to poor cardiometabolic health outcomes. Cardiometabolic health is often studied in shift and blue collar workers, showing an association between poor sleep and the metabolic syndrome,⁷¹ diabetes¹⁵⁹ and obesity,¹⁶⁰ while less is known about the sleep of employees in administrative, professional and managerial positions. In these corporate executives, the focus of the research appears to be directed more towards the work-related and psychosocial impact on sleep. For example, in one study, high job strain has been significantly associated with burnout, but only in the employees experiencing sleeping difficulties defined as insomnia or non-

restorative sleep disorders;¹⁶¹ while in another study, shorter sleeping time was associated with depressive symptoms and anxiety.¹⁶²

Although companies have utilized corporate health risk assessments (HRAs) to measure the health of their executive employee population for more than a decade, sleep is often overlooked, especially in a unique cohort of managerial employees for whom extended work hours, high job stress and sleep deprivation may contribute to suboptimal long-term health. The observed associations between stress,¹⁶³ long working hours¹⁶⁴ and physical inactivity¹⁶⁵ with short sleep duration and consequent impact on cardiometabolic health in employed adults raise the possibility that these factors potentially play an intermediary role in the pathway from sleep duration to cardiometabolic disease risk factors. Identifying whether working, stress and/or physical activity levels mediate this relationship may provide insight into how variables are associated. To the best of our knowledge, this has not been tested in corporate executives.

The first aim was to describe occupational and psychological correlates of self-reported sleep duration in corporate executives, defined as those in senior management or executive positions. The second aim was to explore associations between self-reported sleep duration and cardiometabolic risk factors in these employees. Given the limited research on gender differences with respect to cardiometabolic risk and sleep,^{166,167} particularly in occupational health studies, we attempted to address this gap and stratified our analyses by gender. Further, since it may mediate the magnitude thereof, the third aim was to explore the extent to which physical activity, work- or stress-related factors might mediate the association between sleep duration and cardiometabolic risk factors.

3.2 Methods

Design, setting and participants

This cross-sectional study used corporate health programme data from 3585 managerial employees at 56 companies in South Africa, who underwent health assessments between 2016 and 2019. The companies were from different industrial sectors which included information technology, finance, telecommunication services, health, construction and engineering, consulting, manufacturing and production, retail and wholesale trade, mining, transportation, hospitality.

Participants included in this study were male and female full time employees in senior or executive management positions. Their health assessments comprised a web-based health risk assessment (HRA), followed by a face-to-face comprehensive clinical consultation. Participants were excluded from this study if they were shift workers (n=0), did not have self-reported sleep duration data (n=317), or if there were missing data for gender (n=2). The study was approved by the Human Research Ethics Committee at the University of Cape Town (HREC ref. no: 470/2017) and informed consent was obtained from all participants.

Data collection instruments and measures

Health risk assessment (HRA) questionnaire

The HRA included questions on participant demographics (gender, age), sleep, medical history, occupational, psychological well-being and modifiable lifestyle factors. Sleep duration (h/night) was recorded in response to the question “How many hours, on average, and not including naps, do you usually sleep during the night?”. Response options ranged from <5 h to ≥ 10 h in increments of 30 minutes. Self-reported sleep duration was analysed as a continuous variable throughout this study. Sleep quality was measured with the question “In general, how would you rate your sleep?” with response choices: (1) very good, (2) good, (3) average, (4) poor, and (5) very poor. Fatigue interfering with daytime function was measured with the question ‘How often have you experienced sleepiness or fatigue that interfered with your daily activities (work and/or social)?’ and the response scale was: (1) rarely or never, (2) a few days a month, (3) a few days a week, (4) every or almost every day. This question was introduced in 2018, and consequently there are some missing data for this question.

The occupational variables included hours worked per week; travel time to and from work (min/day); absenteeism and presenteeism. Hours worked per week was stratified into three categories: (1) <40 h/week, (2) 40-60 h/week, and (3) >60 h/week. Absenteeism, defined as absence from work owing to sickness, was measured by a single question “How many days were you absent from work during the last year as a result of illness?”. Responses were: (1) 0 days/year, (2) 1-6 days/year, (3) 7-14 days/year, (4) 15 days/year or more. Presenteeism was measured by the following question: “Over the past 12 months, how often have you gone to work despite feeling that you really should have taken sick leave because of your state of health?” Response options were: (1) never, (2) 1-2 times/year, (3) 3-4 times/year, (4) 5 times/year or more. Since presenteeism was only incorporated into the HRA in 2018, there are some missing data for this variable.

Psychological well-being was assessed using the Depression, Anxiety and Stress Scale (DASS)-21, which consists of 21 statements measuring three subscales: depression, anxiety and stress, as felt over the past week.¹⁶⁸ Each subscale consists of seven items measured on a four-point scale ranging from 0 = Did not apply to me at all, to 3 = Applied to me very much, or most of the time. The DASS-21, which is a shortened version of the DASS-42, has been validated in non-clinical samples,^{169,170} and psychometric properties of this instrument have previously been reported to have Cronbach’s alpha scores of $\alpha = 0.92$ (Mean: 717; standard deviation (SD): 5.39) for stress; $\alpha = 0.90$ (Mean: 4.06; SD: 4.51) for anxiety; and $\alpha = 0.86$ (Mean: 4.85; SD: 4.84) for depression.¹⁷¹ The outcome variables were treated as categorical variables for depression, anxiety and stress symptoms. The sum scores were computed by adding up the scores on the items per subscale and multiplying them by a factor of 2 in order to yield equivalent scores to the full DASS-42. Sum scores for each subscale ranged between 0 and 42 with severity ratings as shown in Supplementary Table S3.1 (Appendix 2). These cut-off scores were derived from a set of severity ratings, proposed by Lovibond and Lovibond.¹⁷²

The lifestyle variables included physical activity (PA, min/week), smoking status and alcohol consumption. Participants reported the number of physical activity (PA) sessions they took part in during a typical week and the duration of each session, from which the average minutes of PA per week were calculated. PA time per week was analysed as a continuous variable for descriptive statistics and incorporated as a dichotomous mediator for mediation analyses (<150 or ≥150 min/week PA). Smoking status and alcohol consumption were analysed as categorical outcome variables. Smoking status was recorded as “current smoker”, “ex-smoker” or “never smoker”, but for the purpose of this study, smoking was analysed as a dichotomous variable (current smoker vs current non-smoker). Employees provided information on alcohol consumption by choosing from the following: (1) never consume alcohol, (2) less than 14 alcoholic drinks/week, (3) 14-21 drinks/week, and (4) more than 21 drinks/week.

Clinical assessment

Cardiometabolic disease risk factors

Standing height (cm) was measured to the nearest cm, using a stadiometer and body weight was measured using a portable calibrated scale and recorded to the nearest 0.1 kg. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared (kg/m²). Waist circumference (WC) was measured around the midpoint of the lowest rib and iliac crest, to the nearest 0.1 cm. Resting systolic blood pressure (SBP, mmHg) and diastolic blood pressure (DBP, mmHg) were measured by the consulting medical doctor using a mercury sphygmomanometer and cuff size appropriate to the participant’s arm circumference. Following a 10 h overnight fast, venous blood samples were drawn and sent to a clinical pathology laboratory (Pathcare, Lancet or Ampath Laboratories) to measure fasting plasma glucose (Glu, mmol/L), high density lipoprotein-cholesterol (HDL, mmol/L) and triglyceride (TG, mmol/L) concentrations.

Cardiometabolic disease risk score

A continuous CMD risk score was calculated by summing the standardized scores for negative fasting serum HDL, and positive plasma Glu, serum TG, BMI, WC, SBP and DBP. This clustered CMD risk score was calculated for each participant as follows:

$$\text{CMD risk score} = -z\text{HDL} + z\text{Glu} + z\text{TG} + [(z\text{BMI} + z\text{WC})/2] + [(z\text{SBP} + z\text{DBP})/2]$$

We used this score to estimate an individual’s overall CMD risk. In contrast to using dichotomous metabolic syndrome characteristics, this approach provides a continuous risk score which increases statistical power, and has been used in previous studies.^{18,66} A higher score indicates a less favourable metabolic profile.

Data and statistical analyses

Data are presented as mean \pm standard deviation (SD), median with the interquartile range (IQR), or count (%). Descriptive comparisons between the men and women were conducted using Mann Whitney U, Chi-Squared or Fisher's Exact tests. Linear regressions were performed to explore occupational and psychological correlates of self-reported sleep duration and to analyse associations between self-reported sleep duration and cardiometabolic risks. For the purpose of this study, the Extremely Severe category of the DASS-21 questionnaire was collapsed into the Severe category. All models were adjusted for age, alcohol consumption, smoking and physical activity.

We used mediation analyses for all significant associations between sleep duration and cardiometabolic characteristics, to explore mediating effects of work hours, stress and physical activity.¹⁷³ Figure 3.1 shows the path diagrams where the product of the a and b coefficients defines the indirect effect of X (independent variable) on Y (dependent variable) through M (mediator). We used 1000 bootstrapping samples to generate 95% confidence intervals (CI) for the different effects being examined. In this study, X refers to self-reported sleep duration, Y refers to cardiometabolic risk and M refers to the mediators: work hours, stress or physical activity. We adjusted for age, smoking, alcohol consumption and physical activity in all models, except for where physical activity was the mediator. Our outcomes are presented (Figure 3.1) by displaying the indirect (effect of M through paths a and b), direct (effect of sleep duration on the outcome variable, controlling for M, path C') and total effects (effect of sleep duration on the outcome variable, path C). Data were analyzed using Stata (v15, StataCorp, Texas, USA) and significance accepted using an alpha value of $P < 0.05$.

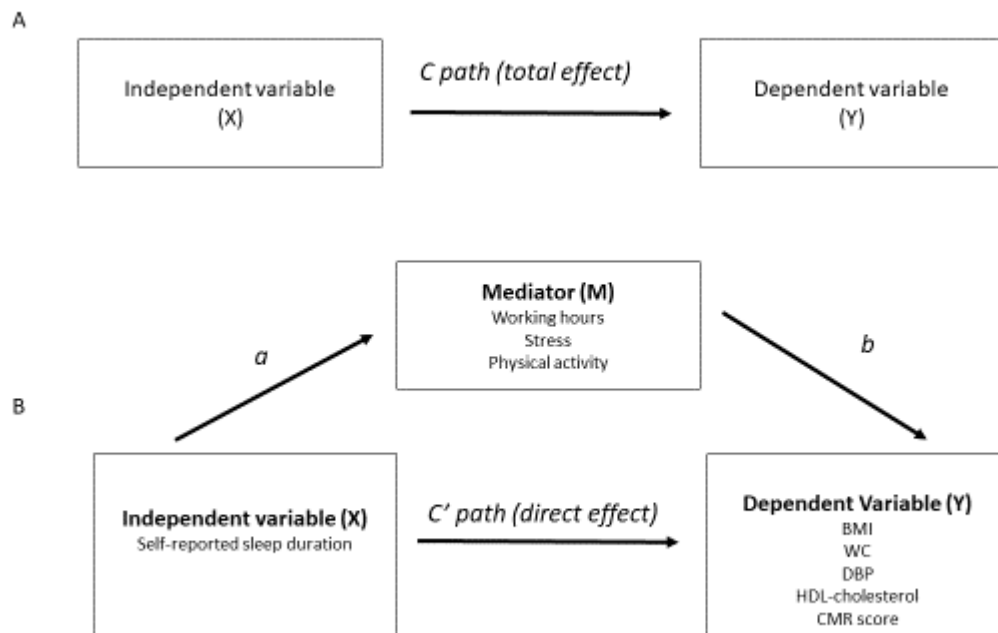


Figure 3.1. Mediation models for the association between self-reported sleep duration and cardiometabolic risk factors: (A) Total-effect model where the C path represents the total effect of the independent variable (self-reported sleep duration) on the dependent variable (cardiometabolic risk factors), without adjusting for mediators. (B) Mediation model where the product of the a and b coefficients define the indirect effect of self-reported sleep duration on cardiometabolic risk factors through the mediators (M). The C' path denotes the direct effect of self-reported sleep duration on cardiometabolic risk factors, controlling for the mediators. BMI, body mass index; WC, waist circumference; DBP, diastolic blood pressure; CMR, cardiometabolic disease risk

3.3 Results

Participant characteristics

Descriptive characteristics of the sample are presented in Table 3.1. Whereas the sleep duration median was 7 h for both men and women, 20.6% of men and 26.5% of women reported a sleep duration <7 h. Sleep duration within the 7-9 h range was reported in 79.5% of the men and 73.5% of the women. No participants reported a sleep duration >9 h.

Table 3.1. Descriptive characteristics of the men and women (n=3585).

	Men (n=2543)	Women (n=1042)	P-value
Age (y)	45.0 (40.0 – 52.0)	42.0 (38.0 – 48.0)	<0.001
Self-reported sleep duration (h)	7.0 (7.0 – 7.0)	7.0 (6.5 – 7.0)	0.090
Sleep quality (count, %)			
Poor	50 (9.5)	30 (9.9)	
Average	181 (34.5)	108 (35.6)	0.911
Good	294 (56.0)	165 (54.5)	
Fatigue interfering with daytime function (count, %)			
Rarely or never	306 (58.5) †	147 (48.7) †	
A few days a month	176 (33.7)	121 (40.1)	0.020
A few days a week	36 (6.9)	26 (8.6)	
Every or almost every day	5 (1.0)	8 (2.7)	
Hours worked (count, %)			
≤40 h/week	88 (3.5) †	54 (5.2) †	
40 – 60 h/week	2149 (84.9)	908 (87.3)	<0.001
≥60 h/week	294 (11.6) ‡	78 (7.5) ‡	
Daily work commute time (count, %)			
1 – 20 min/day	479 (19.6)	186 (18.8)	
21 – 60 min/day	798 (32.6) †	282 (28.5) †	0.006
>60 min/day	1169 (47.8)	521 (52.7)	
Absenteeism (count, %)			
0 days/year	1123 (45.7) ‡	335 (33.6) ‡	
1 – 6 days/year	1233 (50.1) ‡	578 (58.0) ‡	<0.001
7 – 14 days/year	78 (3.2) ‡	59 (5.9) ‡	
≥15 days/year	26 (1.1) †	24 (2.4) †	
Presenteeism (count, %)			
Never	194 (37.1) †	90 (30.0) †	
1 -2 times/year	210 (40.2)	109 (36.3)	
3 -4 times/year	76 (14.5)	57 (19.0)	0.004
≥5 times/year	43 (8.2) †	44 (14.7) †	
Normal	2231 (91.6) †	864 (87.7) †	
Mild	103 (4.2)	47 (4.8)	
Moderate	70 (2.9)	41 (4.2)	<0.001
Severe	31 (1.3) ‡	33 (3.4) ‡	
Normal	2199 (90.4) ‡	831 (84.4) ‡	

Mild	74 (3.0)	35 (3.6)	<0.001
Moderate	112 (4.6) †	70 (7.1) †	
Severe	49 (2.0) ‡	49 (5.0) ‡	
Normal	2056 (84.4) ‡	776 (78.8) ‡	
Mild	189 (7.8)	90 (9.1)	<0.001
Moderate	133 (5.5) †	78 (7.9) †	
Severe	57 (2.3) †	41 (4.2) †	
Physical activity (min/week)	135.0 (20.0 – 270.0)	90.0 (0.0 – 180.0)	<0.001
Current Smoker (count, %)	267 (10.5)	98 (9.4)	0.325
Alcohol intake/week (count, %)			
0 units	488 (19.2) ‡	299 (28.8) ‡	
<14 units	297 (11.7) ‡	33 (3.2) ‡	<0.001
14-21 units	1717(67.7)	704 (67.7)	
>21 units	35 (1.4) †	4 (0.4) †	

Data are presented as median (IQR) or count (%). Sleep quality: n=828 (all), n=525 (men), n=303 (women); Work hours: n=3571 (all), n=2531 (men), n=1041 (women); Daily work commute time: n=3435 (all), n=2446 (men), n=989 (women); Absenteeism: n=3456 (all), n=2460 (men), n=996 (women); Presenteeism: n=823 (all), n=523 (men), n=300 (women); Daytime function: n=825, n=523 (men), n=302 (women). DASS-21: Depression, Anxiety and Stress Scale 21. Depression: n=3420 (all), n=2435 (men), n=985 (women); Anxiety: n=3419 (all), n=2434 (men), n=985 (women); Stress: n=3420 (all), n=2435 (men), n=985 (women); Physical activity: n=3196 (all), n=2264 (men), n=932 (women); Smoker: n=365 (all), n=267 (men), n=98 (women); Alcohol: n=3577, n= 2537 (men), n=1040 (women). P-values represent between group comparisons using Mann-Whitney or Chi-squared tests. Posthoc analyses were conducted using Fisher's Exact test and the † symbol indicates where the posthoc differences lie: † P<0.05 and ‡P<0.001

Significant differences were observed between men and women for all factors except sleep duration, sleep quality and smoking status. Compared to women, men were older (P<0.01), reported less fatigue interfering with their daytime function (P=0.020), longer working hours (P<0.001), more commuting time to work (in the 21-60 min category, P=0.006), less absenteeism (P<0.001), less frequent presenteeism (P=0.004), less depression (P<0.001), anxiety (P<0.001) and stress (P<0.001), were more physically active (P<0.001) and consumed more alcohol per week (P<0.001).

CMD risk factors of the men and women are presented in Table 3.2. In general, 29.3% and 24.1% of the men and women were obese; 45% of men and 17.9% of women had hypertension or elevated blood pressure; and elevated blood glucose or diabetes was prevalent in 23% of the men and 7.8% of the women in this cohort. Men had a larger WC (P<0.001), higher BMI (P<0.001), Glu (P<0.001), TG (P<0.001), SBP and DBP (both P<0.001) and lower HDL (P<0.001) concentrations compared to women. Similarly, a greater proportion of the men were obese (P=0.002) and they had a higher cardiometabolic risk score than women (P<0.001).

Table 3.2. Cardiometabolic disease risks of the men and women (n=3585).

	Men (n=2543)	Women (n=1042)	P-value
BMI (kg/m²)	27.4 (25.1 – 30.6)	25.7 (22.7 – 29.7)	<0.001
WC (cm)	94.0 (87.0 – 102.0)	80.0 (73.0 – 90.0)	<0.001
Glucose (mmol/L)	5.1 (4.8 – 5.5)	4.8 (4.5 – 5.1)	<0.001
TG (mmol/L)	1.2 (0.9 – 1.8)	0.9 (0.6 – 1.2)	<0.001
HDL (mmol/L)	1.2 (1.0 – 1.4)	1.6 (1.3 – 1.8)	<0.001
SBP (mmHg)	122.5 (120.0 – 130.0)	120.0 (110.0 – 120.0)	<0.001
DBP (mmHg)	80.0 (72.0 – 84.0)	70.0 (70.0 – 80.0)	<0.001
Obese (count, %) (BMI ≥30 kg/m ²)	744 (29.3)	251 (24.1)	0.002
High WC (count, %) (≥102 cm for men; ≥88 cm for women)	680 (27.0)	313 (30.5)	0.035
Elevated TG (count, %) (≥1.7)	682 (27.9)	104 (10.3)	<0.001
Low HDL (count, %) (<1.0 mmol/L for men and <1.3 mmol/L for women)	462 (18.4)	28 (2.7)	<0.001
Elevated BP (count, %) (systolic ≥130 and/or ≥85 diastolic mmHg or antihypertensive drug treatment)	1137 (45.1)	184 (17.9)	<0.001
Elevated glucose (count, %) (≥5.6 mmol/L or drug treatment for diabetes)	574 (23.0)	80 (7.8)	<0.001
Cardiometabolic disease risk score	0.15 (-1.53 – 2.18)	-1.62 (-2.99 – 0.30)	<0.001

Data are presented as median (IQR) or count (%). BMI: body mass index, n=3582 (all), n=2542 (men), n=1040 (women); WC: waist circumference, n=3548 (all), n=2521 (men), n=1027 (women); Glucose: n=3520 (all), n=2498 (men), n=1022 (women); TG: triglycerides, n=3457 (all), n=2449 (men), n=1008 (women); HDL: high density lipoprotein, n=3545 (all), n=2516 (men), n=1029 (women); SBP: systolic blood pressure, n=3553 (all), n=2524 (men), n=1029 (women); DBP: diastolic blood pressure, n=3552 (all), n=2524 (men), n=1028 (women); Cardiometabolic risk score: n=3371 (all), n=2391 (men), n=980 (women). P-values represent between group comparisons using Mann-Whitney or Chi-squared tests.

Occupational and psychological correlates of self-reported sleep duration

The occupational and psychological correlates of self-reported sleep duration are presented in Table 3.3. In the fully adjusted models, men who reported longer work hours (>60 vs 40-60 h/week; $P<0.001$) and a longer travel time to work (>60 vs <20 min/day; $P=0.012$) had shorter sleep durations. Similarly, in women, working more than 60 h/week (vs 40-60 h/week, $P=0.001$), and travelling 21-60 min/day ($P=0.041$) or >60 min/day ($P=0.009$) vs <20 min/day to and from work were associated with shorter sleep durations. Among the men, mild ($P=0.002$) and moderate ($P=0.002$) depression scores, moderate ($P<0.001$) and severe ($P<0.001$) anxiety scores and mild ($P=0.001$), moderate ($P<0.001$) and severe ($P<0.001$) stress scores were associated with shorter sleep durations compared to those with normal symptom levels. Among the women, mild depression ($P<0.01$), severe anxiety ($P=0.008$) and mild ($P=0.022$), moderate ($P=0.028$) and severe ($P=0.002$) stress scores were associated with shorter sleep duration.

Table 3.3 Occupational and psychological correlates of self-reported sleep duration in men and women.

	Unadjusted			Adjusted		
	N	β (95% CI)	P-value	N	β (95% CI)	P-value
Men						
Hours worked/week	2531			2257		
<40 v 40-60		0.05 (-0.12; 0.22)	0.558		0.09 (-0.004; 0.004)	0.323
>60 v 40-60		-0.51 (-0.61; -0.41)	<0.001		-0.52 (-0.62; -0.41)	<0.001
Daily work commute time (min/day)	2446			2221		
21-60 v <20		-0.01 (-0.10; 0.08)	0.853		0.02 (-0.07; 0.12)	0.460
>60 v <20		-0.14 (-0.23; -0.06)	0.001		-0.12 (-0.21; -0.03)	0.012
Depression symptoms	2435			2227		
Mild v Normal		-0.20 (-0.36; -0.04)	0.013		-0.62 (-0.43; -0.10)	0.002
Moderate v Normal		-0.33 (-0.53; -0.14)	0.001		-0.31 (-0.51; -0.12)	0.002
Severe v Normal		-0.43 (-0.72; -0.14)	0.003		-0.25 (-0.55; 0.05)	0.102
Anxiety symptoms	2434			2227		
Mild v Normal		-0.23 (-0.42; -0.04)	0.015		-0.16 (-0.35; 0.03)	0.104
Moderate v Normal		-0.44 (-0.59; -0.29)	<0.001		-0.44 (-0.60; -0.28)	<0.001
Severe v Normal		-0.48 (-0.71; -0.25)	<0.001		-0.46 (-0.70; -0.22)	<0.001
Stress symptoms	2435			2227		
Mild v Normal		-0.29 (-0.35; -0.12)	<0.001		-0.22 (-0.34; -0.09)	0.001
Moderate v Normal		-0.28 (-0.42; -0.13)	<0.001		-0.29 (-0.44; -0.15)	<0.001
Severe v Normal		-0.39 (-0.60; -0.18)	<0.001		-0.40 (-0.62; -0.18)	<0.001
Women						
Hours worked/week	1040			929		
<40 v 40-60		0.27 (0.03; 0.51)	0.029		0.26 (-0.0008; 0.51)	0.051
>60 v 40-60		-0.40 (-0.60; -0.19)	<0.001		-0.38 (-0.61; -0.16)	0.001
Daily work commute time (min/day)	989			912		
21-60 v <20		-0.20 (-0.36; -0.03)	0.020		-0.18 (-0.36; -0.007)	0.041

>60 v <20		-0.24 (-0.39; -0.09)	0.002		-0.22 (-0.38; -0.06)	0.009
Depression symptoms	985			913		
Mild v Normal		-0.47 (-0.73; -0.21)	<0.001		-0.51 (-0.78; -0.24)	<0.001
Moderate v Normal		-0.02 (-0.30; 0.26)	-0.120		0.04 (-0.26; 0.33)	0.240
Severe v Normal		-0.03 (-0.34; 0.28)	0.848		-0.04 (-0.37; 0.28)	0.798
Anxiety symptoms	985			913		
Mild v Normal		-0.04 (-0.35; 0.26)	0.778		-0.09 (-0.42; 0.23)	0.579
Moderate v Normal		-0.18 (-0.40; 0.04)	0.100		-0.16 (-0.39; 0.07)	0.169
Severe v Normal		-0.32 (-0.58; -0.07)	0.014		-0.37; (-0.64; -0.09)	0.008
Stress symptoms	985			913		
Mild v Normal		-0.24 (-0.44; -0.05)	0.014		-0.23 (-0.43; -0.03)	0.022
Moderate v Normal		-0.22 (-0.43; -0.01)	0.037		-0.24 (-0.45; -0.03)	0.028
Severe v Normal		-0.45 (-0.73; -0.17)	0.001		-0.49 (-0.80; -0.19)	0.002

Models were determined using linear regression. β : beta coefficient, CI: confidence interval. Models were adjusted for physical activity, alcohol consumption and smoking status.

Associations between self-reported sleep duration and cardiometabolic risk factors

Table 3.4 presents unadjusted and adjusted models of the associations between self-reported sleep duration and cardiometabolic risk factors. In the fully adjusted models, higher BMI ($P=0.002$), larger WC ($P=0.001$), higher DBP ($P=0.027$) and a greater CMD risk score ($P<0.001$) were associated with shorter self-reported sleep duration in men. Among the women, higher BMI ($P=0.040$), larger WC ($P=0.028$), lower HDL ($P=0.027$) and a greater CMD risk score ($P=0.036$) were associated with shorter self-reported sleep durations. The association between self-reported sleep duration and cardiometabolic risk score is shown in Figure 3.2.

Table 3.4. Associations between self-reported sleep duration and cardiometabolic risk factors in men and women.

	Unadjusted			Adjusted		
	N	β (95% CI)	P-value	N	β (95% CI)	P-value
Men						
BMI (kg/m ²)	2542	-0.40 (-0.61; -0.18)	<0.001	2260	-0.36 (-0.59; -0.13)	0.002
WC (cm)	2521	-1.20 (-1.78; -0.61)	<0.001	2245	-1.07 (-1.67; -0.47)	0.001
Glucose (mmol/L)	2498	-0.01 (-0.06; 0.03)	0.580	2221	-0.015 (-0.07; 0.03)	0.546
TG (mmol/L)	2449	-0.04 (-0.9; 0.02)	0.179	2177	-0.03 (-0.08; 0.03)	0.404
HDL (mmol/L)	2516	0.01 (-0.01; 0.03)	0.230	2238	0.001 (-0.02; 0.02)	0.850
SBP (mmHg)	2524	-0.57 (-1.17; 0.02)	0.060	2243	-0.47 (-1.12; 0.16)	0.142
DBP (mmHg)	2524	-0.54 (-0.99; -0.09)	0.018	2243	-0.53 (-1.01; -0.06)	0.027
Cardiometabolic risk score	2391	-0.10 (-0.14; -0.05)	<0.001	2125	-0.09 (-0.13; -0.04)	<0.001
Women						
BMI (kg/m ²)	1040	-0.56 (-0.96; -0.15)	0.007	929	-0.44 (-0.86; -0.02)	0.040
WC (cm)	1027	-1.32 (-2.26; -0.38)	0.006	920	-1.10 (-2.07; -0.12)	0.028
Glucose (mmol/L)	1022	-0.01 (-0.07; 0.04)	0.661	914	-0.02 (-0.07; 0.03)	0.437
TG (mmol/L)	1008	-0.001 (-0.05 ;0.05)	0.957	902	0.01 (-0.04; 0.06)	0.671
HDL (mmol/L)	1029	0.04 (0.01; 0.07)	0.003	920	0.03 (0.004; 0.06)	0.027
SBP (mmHg)	1029	-0.44 (-1.29; 0.40)	0.301	920	-0.38 (-1.24; 0.49)	0.394
DBP (mmHg)	1028	-0.41 (-1.05; 0.23)	0.211	919	-0.40 (-1.06; 0.26)	0.239
Cardiometabolic risk score	980	-0.08 (-0.15; -0.01)	0.019	879	-0.07 (-0.14; -0.005)	0.036

Models were determined using linear regression. β : beta coefficient, CI: confidence interval, BMI: body mass index, WC: waist circumference, TG: triglycerides, HDL: high density lipoprotein, SBP: systolic blood pressure, DBP: diastolic blood pressure. All unadjusted models include age as a covariate. Adjusted models include physical activity, alcohol consumption and smoking status as covariates.

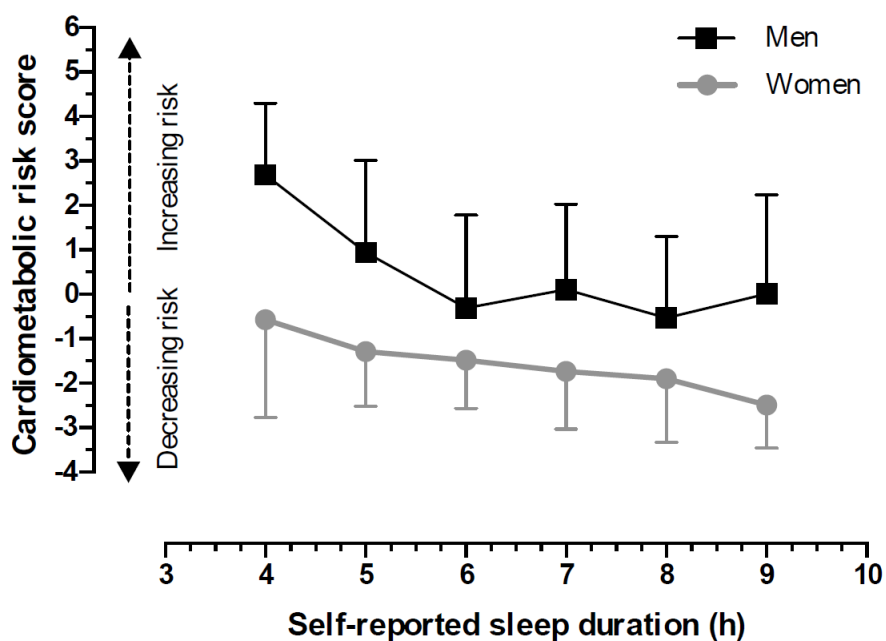


Figure 3.2. Relationship between self-reported sleep duration and cardiometabolic disease risk score in men (n=2391) and women (n=980). Data are presented as median cardiometabolic disease risk score with interquartile range. Shorter self-reported sleep duration was associated with increasing cardiometabolic disease risk in men ($P<0.001$) and women ($P=0.036$).

Mediation analyses

Table 3.5 shows the results of the mediation analyses for men, in whom working hours, stress symptoms and physical activity mediated the main associations between sleep duration and BMI, WC, DBP and cardiometabolic risk score. The magnitudes were small but significant for the indirect effects of self-reported sleep duration and (1) BMI through working hours ($P=0.015$); stress ($P=0.033$) and physical activity ($P=0.006$). Similarly, the magnitudes of the indirect effects were significant for the relationship between sleep duration and (2) WC, through working hours ($P=0.005$), stress ($P=0.044$) and physical activity ($P=0.004$); (3) DBP through working hours ($P=0.026$), stress ($P=0.044$) and physical activity ($P=0.009$) and (4) cardiometabolic risk score through working hours ($P=0.019$), stress ($P=0.024$) and physical activity ($P=0.004$). No mediation effects were observed for women (Supplementary Table S3.2, Appendix 2)

Table 3.5. Mediation analyses of the association between sleep duration and body mass index, waist circumference, cardiometabolic disease risk score and diastolic blood pressure using the mediators working hours, stress and physical activity in men.

Independent variables	Mediators						
	Effect	Working hours ^a		Stress ^a		Physical activity ^b	
		β (95%CI)	P value	β (95%CI)	P value	β (95%CI)	P value
BMI (kg/m²)		N=2227		N=2227		N=2261	
	Indirect	-0.06 (-0.12; -0.01)	0.015	-0.04 (-0.08; -0.01)	0.033	-0.06 (-0.10; -0.02)	0.006
	Direct	-0.29 (-0.54; -0.03)	0.032	-0.30 (-0.57; -0.05)	0.020	-0.34 (-0.58; -0.09)	0.008
	Total	-0.36 (-0.62; -0.09)	0.007	-0.34 (-0.62; -0.08)	0.010	-0.40 (-0.65; -0.14)	0.002
Waist circumference (cm)		N=2227		N=2227		N=2261	
	Indirect	-0.20 (-0.35; -0.06)	0.005	-0.10 (-0.20; -0.02)	0.044	-0.22 (-0.63; -0.07)	0.004
	Direct	-0.89 (-1.57; -0.17)	0.014	-1.0 (-1.73; -0.32)	0.005	-1.0 (-1.66; -0.33)	0.004
	Total	-1.09 (-1.76; -0.35)	0.002	-1.1 (-1.84; -0.40)	0.002	-1.22 (-1.90; -0.53)	<0.001
Cardiometabolic risk score		N=2257		N=2227		N=2261	
	Indirect	-0.01 (-0.02; -0.003)	0.019	-0.008 (-0.02; -0.002)	0.024	-0.01 (-0.02; -0.004)	0.004
	Direct	-0.07 (0.02; -0.12)	0.002	-0.08 (-0.13; -0.03)	0.001	-0.08 (-0.13; -0.04)	<0.001
	Total	-0.09 (-0.13; -0.04)	<0.001	-0.09 (-0.13; -0.04)	<0.001	-0.09 (-0.14; -0.05)	<0.001
DBP (mmHg)		N=2257		N=2227		N= 2261	
	Indirect	-0.12 (-0.22; -0.01)	0.026	-0.07 (-0.15; -0.004)	0.044	-0.08 (-0.13; -0.02)	0.009
	Direct	-0.42 (-0.89; 0.06)	0.089	-0.47 (-0.93; -0.02)	0.052	-0.50 (-0.98; -0.02)	0.042
	Total	-0.53 (-1.0; -0.06)	0.027	-0.54 (-1.02; -0.09)	0.024	-0.57 (-1.06; -0.09)	0.020

BMI: body mass index; DBP: diastolic blood pressure; β: beta co-efficient; CI: confidence interval. ^aThe effects were adjusted for age, smoking status, alcohol consumption and physical activity. ^bThe effects were adjusted for age, smoking status and alcohol consumption.

3.4 Discussion

Working hours, work-related commute time and stress were found to be the occupational and psychological factors associated with shorter sleep duration in this cohort of corporate executives. Our findings further suggest that compared to their longer sleeping colleagues, the men with shorter sleep durations were more likely to have higher BMI, WC, DBP and overall CMD risk scores, while shorter sleeping women were more likely to have higher BMI, WC and CMD risk scores, but lower HDL. Working hours, stress and physical activity appear to mediate these relationships among the male but not the female corporate executives.

Female corporate executives had a lower mean CMD risk score, compared to their male counterparts,¹⁸ and when comparing the associations between sleep and individual CMD risk factors, it is evident that men and women presented with distinctly different outcomes which may be explained by variances in body composition (adiposity) and hormonal regulation.¹⁷⁴ Specifically, gender differences were observed in the relationships between sleep duration, DBP and HDL cholesterol. Only among the male executives was sleep duration inversely associated with DBP, which is in contrast to previous literature reporting that sleep-deprived women were at a greater risk of developing hypertension.¹⁷⁵ However, the average age of women in studies investigating sleep and hypertension was approximately 55 years,^{176,177} which may correspond to the decrease in oestrogen occurring during menopause. Moreover, the same studies showed that the greatest risk for high blood pressure were in women sleeping <5 h.^{176,177} In our sample of 1042 women, only 262 reported <6 h sleep per day and their mean age was 43 years. Therefore, we could speculate that our contrasting finding may partly be due to the younger age of our participants, and the smaller proportion reporting on short sleep duration.

In women, longer sleep duration was associated with higher HDL cholesterol concentration and is in line with findings reported by Kaneita et al., that sleep duration of ≤ 6 h/day was linked to low serum HDL cholesterol in women specifically.¹⁷⁸ While the mechanisms behind these gender-specific associations are unclear, the most consistent findings seem to be that short sleep is associated with adverse effects on lipid metabolism, including lower HDL cholesterol levels.¹⁷⁹

Mediators of the association between sleep duration and CMD risk was only observed in men. More specifically, longer work hours, physical activity and higher stress scores were found to mediate the associations between sleep duration and BMI, WC, DBP and CMD risk score. Since this mediating effect was small, it is plausible that had we had a greater sample of women in our study, similar findings may have been observed. Alternatively, we speculate that other factors that were not measured in this study, such as family responsibilities or childcare, may have been more suitable mediators in women. For example, research conducted by Nemoto et al. 2012 exploring the association between long work hours and the gender divide in corporate Japan showed that women managers were still being expected to fulfil a 'dual-role' of being a care-taker and ideal worker.¹⁸⁰ Similarly, Cho et al. 2015 described a 'double burden' of work and family

responsibilities in white collar women employees.¹⁸¹ Additionally, the men in this study reported significantly longer work hours and higher physical activity levels relative to the women which may have further strengthened the mediating effect.

In a study corroborating the interrelationship between sleep, work, physical activity and BMI, Magee et al. (2011) found a link between working hours, sleep duration and obesity, such that short sleep partially mediated the association between long work hours and increased BMI. Their study made no differentiation between shift or non-shift workers, and their BMI was self-reported. Since shift work is associated with many adverse health outcomes including obesity, CVD and CMD,⁴ and self-reporting weight and height could be prone to reporting biases, our study that excluded shift workers and utilized objective anthropometric measurements, would potentially have reduced such bias.

The odds of higher perceived stress in short sleepers (≤ 7 h/day) have been reported in employees with specialized work and in office workers, when compared to those in manual labour;¹⁶³ and in another study, the primary predictor of short sleep in obese employees was stress.¹⁸² Insufficient sleep may evoke a stress response, contributing to fat deposition and raised blood pressure, both of which are features of the shorter sleeping men in this cohort.

While poor sleep may reduce motivation to be physically active¹⁸³ and increase daytime sleepiness, lower levels of physical activity have also been associated with increased daytime fatigue,¹⁸⁴ which over time, could promote weight gain and obesity via reduced energy expenditure. Taken together, a concomitant decrease in sleep duration and increase in stress may thus play an important role in linking stress with CMD risk, while lower physical activity levels may partly explain the relationship between sleep and obesity.

Our findings suggest that extended work hours increase the likelihood for obesity, and corroborate the work of Di Milia and Mummery, who found that long daily working hours (12 h/day), and short sleep (≤ 7 h/day), were predictors of obesity.¹⁸⁵ Moreover, older executives who worked ≥ 50 h/week and slept ≤ 47 h/week in their midlife reported poorer physical health and function (i.e. health-related quality of life) in old age.¹⁸⁶ Since our overall cohort had a mean age of 45 years of which many reported sleeping < 7 h and working ≥ 60 h/week, our results are important in implementing a pro-active approach to workplace health, mitigating future adverse CMD risks, and promoting a better health-related quality of life. The combined effect of long work hours and short sleep duration may therefore amplify the risk for CMD, such as CVD and T2DM.

Strengths of this study include the well-characterized sample of corporate executives who came from a similar working background and who participated in a standardized routine data collection procedure, which included a HRA and face-to-face clinical assessments. Seldom are men and women described separately in employee cohorts, and to our knowledge this is the first to do so regarding sleep duration and CMD risk in corporate executives.

Self-reported data that were used to describe the sleep and work-related characteristics (e.g., absenteeism, presenteeism, travel time) of the cohort may have suffered from recall biases, which is a limitation to this study. Future work would therefore benefit by incorporating objective measures of sleep such as actigraphy or polysomnography. However, the data obtained were from standardised HRAs, which were most convenient for a cohort of corporate executives with time constraints and high job demands. Secondly, as this was a cross-sectional study in a unique subset of the working population, causal relationships could not be determined and the findings cannot be generalized. However, it provides some insight into correlates of short sleep duration, and the relationship of sleep with CMD risk factors in an exclusive sample of business executives and managers, and future studies may extend into different levels of employment allowing for a greater understanding of these relationships in working adults. Thirdly, we only accounted for blood pressure, diabetes and cholesterol treatment to analyse CMD risk, and therefore there is potential for confounding by medications that affect sleep, mental health or that may interact to affect blood pressure or lipid profiles.

3.5 Conclusion

These findings provide insight into the relationship between sleep duration and CMD risk in corporate executives, and help to fill the gap in the literature by describing gender differences in this unique cohort. Long work hours, daily work commute time, elevated stress, anxiety and depression scores were apparent correlates of shorter sleep duration, which in turn was associated with CMD risk. These data suggest that corporate executives exhibiting elevated stress, anxiety and depression symptoms, those who work long hours, and have a long daily commute time to work may be vulnerable to poor cardiometabolic health outcomes by compromising sleep duration. This knowledge is useful since managers, executives and entrepreneurs are continually faced with long working hours, coupled with high pressures experienced in their job responsibilities. The potential to modify sleep and lower long term risk for CMD through interventions may differ depending on the correlates of sleep duration, and the mediators of this relationship with CMD. Our findings may thus assist companies to underpin underlying occupational and psychological factors such as working hours and stress, and provide a starting point for targeted sleep interventions to inform the workforce and its management about the potential health consequences of poor sleep. Such findings may provide additional evidence in this unique cohort, emphasizing workplace health programmes that promote a balance between work hours and sleep, combined with stress management in order to mitigate the development of CMD and its comorbidities in corporate executives.

Chapter 4

Longitudinal association between self-reported sleep duration and cardiometabolic disease risk in corporate executives

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Abstract

This study aimed to determine the longitudinal associations between self-reported sleep duration and cardiometabolic disease (CMD) risk in corporate executives. Self-reported sleep duration and lifestyle, occupational, psychological, and measured anthropometrical, blood pressure and blood marker variables were obtained from 1512 employees at annual health risk assessments between 2016 and 2019. Gender-stratified linear mixed models, adjusting for age, lifestyle, occupational and psychological covariates were used to explore these longitudinal associations. Among women, shorter sleep duration was longitudinally associated with higher body mass index (BMI) covarying for age only (β with 95% confidence intervals: -0.19 [-0.36, -0.03]), age and occupational factors (-0.20 [-0.36, -0.03]) and age and psychological factors (-0.20 [-0.37, -0.03]). Among men, shorter sleep was longitudinally associated with both BMI and waist circumference (WC) covarying for age only (BMI: -0.15 [-0.22; -0.08]; WC: -0.62 [-0.88; -0.37]); age and lifestyle factors (BMI: -0.12 [-0.21; -0.04]); WC: -0.016 [-0.92; -0.29], age and occupational factors (BMI: -0.20 [-0.22; 0.08]; WC: -0.62 [-0.88; -0.36]), and age and psychological factors (BMI: -0.15 [-0.22; -0.07]; WC: -0.59 [-0.86; -0.33]). Among men, shorter sleep was also longitudinally associated with higher CMD risk scores in models adjusted for age and lifestyle factors (CMD: -0.12 [-0.20; -0.04]) and age and psychological factors (CMD: -0.08 [-0.15; -0.01]). Corporate executives who report shorter sleep durations are more likely to present with poorer CMD risk profiles, independent of age, lifestyle, occupational and psychological factors. Addressing sleep health in workplace health programmes may help mitigate the development of CMD in such employees.

Keywords: employees; health risk assessment; longitudinal study; sleep quantity

4.1 Introduction

Cardiometabolic disease (CMD) risk has been defined as a cluster of adverse metabolic and cardiovascular factors, including obesity, hypertension, dyslipidaemia and hyperglycaemia, which predispose individuals to cardiovascular disease (CVD) and type 2 diabetes.^{3,125,148} Existing evidence shows that sleep which is both longer (>9 h) and shorter (<7 h) than the 7-9 h recommended duration guidelines²⁶ increases this risk. Furthermore, there is evidence that decreasing sleep duration below the required optimum time may lead to a number of adverse mental health outcomes, which include stress, anxiety and depression.^{187,188}

In the workplace specifically, shorter sleep (<7 h) contributes largely to absenteeism (e.g. workplace absence due to illnesses) and presenteeism (e.g. sub-optimal work performance due to working while ill).⁹⁸ When compared to workers sleeping the recommended 7–9 h per night, sleeping <6 h has been shown to equate to losing approximately six working days per year due to absenteeism and a 2.4% productivity loss due to presenteeism.¹³⁷ Predictors of employee sleep duration include working hours, work demands, and job complexity, such that longer work hours (>50 h/week) and high work demands contribute to shorter sleep duration.^{83,93,189} Adequate sleep is critical for workplace performance¹³⁹ and strongly influences the work quality and output of a subgroup of employees, namely corporate executives who are exposed to considerable performance pressure in a competitive environment. A study by Ganesh et al. (2018) examined predictors of stress in executives and found that they struggle most with poor quality sleep, anxiety and lack of physical activity.¹⁵⁷ This suggests that executives may compromise their sleep and physical activity to meet challenging performance objectives in response to work pressures. Moreover, corporate executives drive the strategic and tactical direction of a business, and when sleep deprived, may exhibit increasingly distorted risk processing, leading to irrational business decisions, ultimately compromising large corporations.¹⁹⁰

Another consideration in understanding the sleep–CMD risk relationship within this subgroup is that both sleep and risk factors for CMD may present differently between men and women.^{167,191} Such evidence in corporate executives has been reported by a recent cross-sectional study investigating the association between sleep and CMD risks, which shows that there may indeed be gender differences for risk associations in this group.¹⁹² In addition, the disproportionate representation of men and women in top tiers of management further support the decision to explore sleep and health relationships separately in these cohorts.

Many corporations offer employee health risk assessments (HRAs), which typically measure factors related to physical activity, diet, smoking and alcohol consumption. Despite the evident impact of poor sleep on health and productivity,⁹⁸ sleep has only recently been explored more extensively in the workplace. For example, when HRA data from employees of a Fortune 100 corporation were used to identify whether or not changes in sleep duration over two years were associated with changes in health risk factors, medical conditions, or workplace economic outcomes, those sleeping <6 h or >9 h per night had more CMD risk

factors, medical conditions and lower work productivity, compared to those sleeping 7-9 h. Additionally, employees who restored their sleep to 7-8 h over the two years, showed a significant improvement in on-the-job-productivity as measured by presenteeism.¹³²

While there is growing evidence of the impact of employee sleep duration on CMD health, previous studies have tended to focus on shift work rather than the sleep of non-shift workers, and often non-managerial occupations as opposed to employees in senior and executive management positions. Further, the predominance of cross-sectional occupational sleep-related studies limit the ability to account for complex and reciprocal relationships between sleep and CMD risk within a corporate population across follow-up time. Longitudinal studies that have investigated employees' sleep have focussed predominantly on psychosocial relationships and/or were conducted in shift workers.^{193,194} There is therefore an opportunity to expand on such work to better understand the factors that influence the longitudinal relationship between sleep and CMD risk in the non-shift working adult corporate executives. Such data could help establish sleep health as one of the pillars of workplace health programmes, which aim to mitigate and manage disease in a highly demanding work environment. Therefore, the aim of this study was to describe the longitudinal association between self-reported sleep duration and CMD risk, over a four-year period in corporate executives, *a priori* stratified for gender. It was hypothesized that shorter sleep duration would be longitudinally associated with increased CMD risk.

4.2 Methods

Study design and population

This study is a secondary, longitudinal analysis of HRA data collected annually from January 2016 to December 2019 (n=1840), for which corporate executives were able to consent to their data being used for research purposes. The study was approved by the Faculty of Health Sciences' Human Research Ethics Committee at the University of Cape Town (HREC ref. no: 470/2017) and informed consent was obtained from all participants allowing for data inclusion. Briefly, employees completed a web-based HRA prior to a comprehensive clinical consultation and were full-time employees in senior or executive management positions. The companies were from different business sectors which included information technology, finance, telecommunication services, health, construction and engineering, consulting, manufacturing and production, retail and wholesale trade, mining, transportation and hospitality. All participants were eligible for this study unless they had only one measurement over the four-year period, did not respond to sleep questions, or were shift workers. In total, data from 1160 men and 352 women were included in the analyses giving a response rate of 82%.

Measures

The following measures were taken at each annual HRA (2016, 2017, 2018, 2019): self-reported data related to occupation, sleep duration, mental health and lifestyle-related CMD risk factors (physical activity, alcohol intake, smoking). The clinical assessment provided quantitative data such as anthropometric measurements, fasting blood parameters and blood pressure. These measures are described in more detail below.

Occupational factors included: (1) hours worked per week (<40 h/week; 40–60 h/wk or >60 h/wk); (2) absenteeism, (3) presenteeism and (4) commute time to work (travel time to and from work, min/day). Absenteeism was measured by a single question “How many days were you absent from work during the last year as a result of illness?”. Responses were: (1) 0 days, (2) 1-6 days, (3) 7-14 days, (4) 15 days or more. Presenteeism was measured by the single question: “Over the past 12 months, how often have you gone to work despite feeling that you really should have taken sick leave because of your state of health?”. The response options were: (1) never, (2) one to two times, (3) three to four times, (4) five times or more. Presenteeism was incorporated into the HRAs in the year 2018. Therefore, participants whose first year of participation was in 2016 or 2017 are missing presenteeism data (n=1387).

Sleep duration: Self-reported sleep duration was recorded as the number of hours slept per night in response to the question “How many hours, on average, and not including naps, do you usually sleep during the night?” with the option to choose from ≤ 5 h to ≥ 10 h in increments of 30 minutes.

Mental health: The Depression, Anxiety and Stress Scale (DASS)-21 comprises 21 statements that measured three subscales, namely depression, anxiety and stress, as felt over the past week.¹⁶⁸ Each subscale consists of seven items measured on a four-point scale, ranging from 0 = Did not apply to me at all, to 3 = Applied to me very much, or most of the time. The psychometric properties of this instrument have previously been applied to both healthy and psychiatric populations, as well as in an occupational health care setting.^{170,195,196} The Cronbach’s alpha for each scale for the DASS normative sample are depression: 0.91; anxiety: 0.84; and stress: 0.90.¹⁷¹ Outcome variables for the present study were represented as continuous variables for depression, anxiety and stress scores, where higher scores denoted greater severity.

Lifestyle factors: Participants reported the number of physical activity sessions they performed in a typical week and the duration of each session, from which the average minutes of physical activity per week was calculated. Physical activity time per week was analysed as a continuous variable. Alcohol consumption and smoking status were analysed as categorical variables. Employees provided information on alcohol consumption by choosing from the following: (1) never consume alcohol; (2) less than 14 alcoholic drinks per week, (3) 14–21 alcoholic drinks per week, and (4) more than 21 alcoholic drinks per week. Smoking status was recorded as: (1) current smoker, (2) ex-smoker or (3) never smoked. For the purpose of this study, smoking was analysed as a dichotomous variable (current smoker vs. current non-smoker).

CMD risk factors: Biometric measurements were conducted by trained healthcare professionals and included height (cm); body weight (kg); waist circumference (WC, cm) and blood pressure (BP, mmHg). Following a 10 h overnight fast, blood samples were sent to a clinical pathology laboratory to measure fasting plasma glucose (Glu mmol/L), high density lipoprotein-cholesterol (HDL, mmol/L) and triglyceride (TG, mmol/L) concentrations. Each of these factors were analysed as continuous variables and were also dichotomized according to the defined criteria by the Adult Treatment Panel III of the National Cholesterol Education Program (ATP-III)¹⁹⁷ (Supplementary Table S4.1, Appendix 3).

A continuous CMD risk score was calculated by summing the standardized z-scores for key variable as follows: $-z\text{HDL} + z\text{Glu} + z\text{TG} + [(z\text{BMI} + z\text{WC})/2] + [(z\text{SBP} + z\text{DBP})/2]$. Since HDL and waist circumference vary by gender, this study stratified them by gender before standardizing them. This approach provides a continuous risk score that increases statistical power, and has been used in previous studies.^{18,66} A higher score indicates a less favourable CMD profile.

Data and statistical analyses

Data are presented as median (interquartile range, IQR), count (%) or beta coefficients with 95% confidence intervals (CI). Longitudinal associations between self-reported sleep duration and CMD risk factors were analysed using linear mixed model analyses: model 1 adjusted for age; model 2 adjusted for age and lifestyle factors, i.e., physical activity, alcohol consumption and smoking status; model 3 adjusted for age and occupational factors, i.e., work hours and travel time, and model 4 adjusted for age and psychological factors, i.e., DASS scores for depression, anxiety and stress. Regression coefficients of these longitudinal analyses reflect the relationship between sleep duration and CMD risk factors on average over time and include both within- and between subject relationships.¹⁹⁸ Linear mixed model analyses were used to (1) take into account the correlated observations within the participants by adding a random intercept on subject level to all models and (2) because the method has demonstrated to be highly suitable for the analysis of longitudinal data with missing values.^{199,200} Given that findings from our cross-sectional study in the same cohort showed significantly different health risk profiles between men and women,¹⁹² all longitudinal analyses were performed separately for men and women. Data were analysed using Stata (v.15, StataCorp, Texas, USA) and significance accepted for $P < 0.050$.

4.3 Results

Description of the cohort

Data on occupational, psychological and lifestyle factors were available for 1512 corporate executives (men: $n=1160$ [76.7%], women: $n=352$ [23.3%]) and are presented in Table 4.1. The CMD risk factors of the cohort

illustrated in Table 4.2 indicate that 25% had high waist circumference measurements, 19% had elevated glucose concentrations, 28% elevated TG concentrations, one in four had low HDL-concentrations and 42% presented with elevated BP based on the respective ATP-III criteria. Tables 4.1 and 4.2 represent all data collected for the cohort's first HRA.

Table 4.1. Descriptive characteristics of corporate executives from 56 companies at first health risk assessment in South Africa between 2016 and 2019.

	N	All (n=1512)	Men (n=1160)	Women (n=352)
Age (y)	1512	45.0 (40.0 – 51.0)	46.0 (41.0 – 52.0)	44.0 (39.0 – 48.0)
Self-reported sleep duration (h)	1512	7.0 (7.0 – 7.0)	7.0 (7.0 – 7.0)	7.0 (6.5 – 7.0)
Hours worked (n, %)	1438			
≤40 h/week		54 (3.8)	39 (3.5)	15 (4.5)
40 – 60 h/week		1230 (85.5)	936 (84.7)	294 (87.8)
≥60 h/week		154 (10.7)	128 (11.6)	26 (7.8)
Daily work commute time (min/day)	1440	50.0 (30.0 – 90.0)	50.0 (30.0 – 90.0)	50.0 (30.0 – 90.0)
Absenteeism (n, %)	1385			
0 days/year		647 (46.7)	531 (49.5)	116 (37.0)
1 – 6 days/year		683 (49.3)	502 (46.8)	181 (57.8)
7 – 14 days/year		55 (4.0)	39 (3.6)	16 (5.1)
≥15 days/year		0 (0.0)	0 (0.0)	0 (0.0)
Presenteeism (n, %)	125			
Never		40 (32.0)	28 (32.9)	12 (30.0)
1 – 2 times/year		50 (40.0)	37 (43.5)	13 (32.5)
3 – 4 times/year		23 (18.4)	14 (16.5)	9 (22.5)
≥5 times/year		12 (9.6)	6 (7.1)	6 (15.0)

Depression anxiety stress scale	1397			
Depression score		1.0 (0.0 -3.0)	1.0 (0.0 -3.0)	1.0 (0.0 – 4.0)
Anxiety score		1.0 (0.0 – 3.0)	1.0 (0.0 – 3.0)	1.0 (0.0 – 4.0)
Stress score		6.0 (3.0 – 11.0)	6.0 (3.0 – 11.0)	7.0 (3.0 – 13.0)
Physical activity (min/week)	978	180.0 (120.0 – 300.0)	182.0 (120.0 – 305.0)	150.0 (90.0 – 240.0)
Current smoker (n, %)	1440	134 (9.3)	106 (9.6)	28 (8.4)
Alcohol intake/week (n, %)	1437			
0 units		305 (21.2)	215 (19.5)	90 (27.0)
<14 units		964 (67.1)	732 (66.4)	232 (69.5)
14-21 units		149 (10.4)	138 (12.5)	11 (3.3)
>21 units		19 (1.3)	18 (1.6)	1 (0.3)

Data are presented as median (IQR) or count (%).

Table 4.2. Cardiometabolic disease risk factors of corporate executives from 56 companies at first health risk assessment in South Africa between 2016 and 2019.

	All (n=1512)	Men (n=1160)	Women (n=352)
BMI (kg/m²)	26.8 (24.5 – 30.0)	27.1 (24.9 – 30.1)	25.5 (22.7 – 29.9)
WC (cm)	91 (83 – 100)	93 (86 – 101)	80 (74 – 90)
Glucose (mmol/L)	5.0 (4.7 – 5.4)	5.1 (4.8 – 5.5)	4.8 (4.5 – 5.1)
TG (mmol/L)	1.1 (0.8 – 1.7)	1.2 (0.9 – 1.8)	0.9 (0.6 – 1.3)
HDL-cholesterol (mmol/L)	1.3 (1.1 – 1.5)	1.2 (1.0 – 1.4)	1.6 (1.3 – 1.8)
SBP (mmHg)	120 (118 – 130)	120.0 (120 – 130)	120.0 (110 – 120)
DBP (mmHg)	80 (70 – 82)	80 (70 – 82)	70 (70 – 80)
High WC (n, %)	378 (25.2)	277 (24.0)	101 (28.9)
Elevated glucose (n, %)	294 (19.4)	267 (23.0)	27 (7.7)
Elevated TG (n, %)	426 (28.2)	365 (31.5)	61 (17.3)
Low HDL-cholesterol (n, %)	387 (25.6)	326 (28.1)	61 (17.3)
Elevated BP (n, %)	641 (42.4)	559 (48.2)	82 (23.3)
CMD risk score	-0.28 (-1.30 – 0.89)	0.02 (-0.91– 1.11)	-1.35 (-2.29 - -0.02)

Data are presented as median (IQR) or count (%). BMI, body mass index; WC, waist circumference; TG, triglycerides; HDL, high density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CMD, cardiometabolic disease.

Longitudinal analyses

Table 4.3 illustrates the results of the linear mixed model analyses in women. Over the four-year period, shorter self-reported sleep duration was consistently associated with a higher BMI when adjusting for age ($\beta = -0.19$, 95%CI: -0.36, -0.03, $P=0.019$); age and occupational factors ($\beta = -0.20$, 95%CI: -0.36, -0.03, $P=0.012$); and age and psychological factors ($\beta = -0.20$, 95%CI: -0.37, -0.03, $P=0.020$); but not when adjusting for age and lifestyle factors. It is possible that the smaller sample size in the latter model limited the power of this analysis. Moreover, the regression coefficient of -0.19 (model 1) represents the weighted average of within – and between – subject relationships such that the within-subject interpretation indicates that a self-reported increase of one hour in sleep duration within participants is associated with a decrease of 0.19 units in BMI over the four-year period, and the between-subject interpretation indicates that a one hour longer sleep duration between participants is associated with a 0.19 unit lower BMI. A shorter self-reported sleep duration was also weakly associated with a lower HDL-cholesterol concentration in the age-adjusted model over the four-year period ($\beta = 0.03$, 95%CI: 0.00, 0.06, $P=0.045$).

Among the men (Table 4.4), a shorter self-reported sleep duration was associated with a higher BMI when adjusting for age ($\beta = -0.15$, 95%CI: -0.22, -0.08, $P < 0.001$); age and lifestyle factors ($\beta = -0.12$, 95%CI: -0.21, -0.04, $P = 0.004$); age and occupational factors ($\beta = -0.20$, 95%CI: -0.22, -0.08, $P < 0.001$); and age and psychological factors ($\beta = -0.15$, 95%CI: -0.22, -0.07, $P < 0.001$) over the four-year period. Similarly, a shorter sleep duration was associated with a higher WC in all four models (all $P < 0.001$). A shorter sleep duration was also associated with a higher CMD risk score when adjusting for age and lifestyle factors ($\beta = -0.12$, 95%CI: -0.20, -0.04, $P = 0.002$) and age and psychological factors ($\beta = -0.08$, 95%CI: -0.15, -0.01, $P = 0.030$).

Table 4.3. Longitudinal associations between self-reported sleep duration and cardiometabolic disease risk in women from 56 companies in South Africa between 2016 and 2019.

	Model 1 (n=361)		Model 2 (n=298)		Model 3 (n=361)		Model 4 (n=356)	
	β (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value
BMI (kg/m²)	-0.19 (-0.36; -0.03)	0.019	-0.13 (-0.32; 0.06)	0.169	-0.20 (-0.36; -0.03)	0.012	-0.20 (-0.37; -0.03)	0.020
WC (cm)	-0.37 (-0.88; 0.15)	0.166	-0.14 (-0.85; 0.56)	0.693	-0.36 (-0.87; 0.16)	0.179	-0.36 (-0.89; 0.17)	0.185
Glucose (mmol/L)	0.01 (-0.04; 0.06)	0.765	0.01 (-0.06; 0.07)	0.801	0.00 (-0.05; 0.06)	0.881	0.00 (-0.05; 0.06)	0.944
TG (mmol/L)	-0.02 (-0.05; 0.02)	0.405	0.03 (-0.02; 0.07)	0.275	-0.02 (-0.06; 0.02)	0.282	-0.01 (-0.05; 0.02)	0.477
HDL-cholesterol (mmol/L)	0.03 (0.00; 0.06)	0.045	0.02 (-0.001; 0.06)	0.160	0.03 (-0.001; 0.05)	0.056	0.02 (-0.00; 0.05)	0.094
SBP (mmHg)	-0.25 (-1.10; 0.60)	0.561	-0.80 (-1.81; 0.22)	0.126	-0.29 (-1.15; 0.57)	0.511	-0.26 (-1.12; 0.61)	0.558
DBP (mmHg)	-0.25 (-0.90; 0.39)	0.444	-0.25 (-1.03; 0.54)	0.538	-0.33 (-0.99; 0.32)	0.318	-0.27 (-0.92; 0.39)	0.425
CMD risk score	-0.07 (-0.18; 0.05)	0.254	-0.06 (0.20; 0.08)	0.388	-0.07 (-0.18; 0.05)	0.255	-0.07 (-0.19; 0.04)	0.218

Data are presented as linear mixed effects regression coefficients (β) and 95% confidence intervals (CI). Model 1: adjusted for age; Model 2: adjusted for age and lifestyle factors (physical activity, alcohol consumption, smoking status); Model 3: adjusted for age and occupational factors (work hours, travel time); Model 4: adjusted for age and psychological factors (depression, anxiety, stress). BMI, body mass index; WC, waist circumference; TG, triglycerides; HDL, high density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; CMD, cardiometabolic disease.

Table 4.4. Longitudinal associations between self-reported sleep duration and cardiometabolic disease risk in men from 56 companies in South Africa between 2016 and 2019.

	Model 1 (n=1168)		Model 2 (n=1014)		Model 3 (n=1169)		Model 4 (n=1156)	
	β (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value
BMI (kg/m²)	-0.15 (-0.22; -0.08)	<0.001	-0.12 (-0.21; -0.04)	0.004	-0.20 (-0.22; -0.08)	<0.001	-0.15 (-0.22; -0.07)	<0.001
WC (cm)	-0.62 (-0.88; -0.37)	<0.001	-0.06 (-0.92; -0.29)	<0.001	-0.62 (-0.88; -0.36)	<0.001	-0.59 (-0.86; -0.33)	<0.001
Glucose (mmol/L)	-0.04 (-0.09; 0.00)	0.074	-0.05 (-0.09; 0.00)	0.053	-0.04 (-0.09; 0.01)	0.078	-0.03 (-0.08; -0.10)	0.179
TG (mmol/L)	-0.07 (-0.15; 0.02)	0.151	-0.06 (-0.18; 0.05)	0.257	-0.05 (-0.14; 0.04)	0.239	-0.05 (-0.14; 0.04)	0.305
HDL-cholesterol	0.00 (-0.01; 0.02)	0.548	-0.00 (-0.02; 0.02)	0.891	0.00 (-0.01; 0.02)	0.751	0.00 (-0.01; 0.02)	0.771
SBP (mmHg)	-0.31 (-0.85; 0.22)	0.255	-0.12 (-0.74; 0.51)	0.720	-0.31 (-0.84; 0.23)	0.265	-0.34 (-0.89; 0.21)	0.224
DBP (mmHg)	0.33 (-0.42; 1.08)	0.382	-0.08 (-0.56; 0.40)	0.746	0.37 (-0.39; 1.13)	0.336	-0.25 (-0.67; 0.17)	0.237
CMD risk score	-0.04 (-0.12; 0.04)	0.290	-0.12 (-0.20; -0.04)	0.002	-0.04 (-0.11; 0.04)	0.368	-0.08 (-0.15; -0.01)	0.030

Data are presented as regression coefficients (β) and 95% confidence intervals (CI). Model 1: adjusted for age; Model 2: adjusted for age and lifestyle factors (physical activity, alcohol consumption, smoking status); Model 3: adjusted for age and occupational factors (work hours, travel time); Model 4: adjusted for age and psychological factors (depression, anxiety, stress). BMI, body mass index; WC, waist circumference; TG, triglycerides; HDL, high density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; CMD, cardiometabolic disease.

4.4 Discussion

To the best of our knowledge, we present some of the first longitudinal data describing gender-specific associations between self-reported sleep duration and CMD risk in corporate executive employees over a four-year follow-up period. We build on existing longitudinal studies that have described this relationship in shift workers²⁰¹ or children and adolescents.²⁰² Our findings suggest that corporate executives whose self-reported sleep is shorter than the recommended 7-9 h may be more vulnerable to obesity and CMD over time, and that this relationship differs between men and women. While both men and women reporting a shorter sleep duration had higher BMIs over the study period, the men reporting shorter sleep also had higher waist circumference measures and CMD risk scores. Conversely, women reporting shorter sleep presented with lower HDL concentrations.

The gender-specific differences in longitudinal associations between shorter sleep and higher BMI appeared to be sensitive to various lifestyle factors. While men appeared to exhibit a more robust relationship, the sleep-BMI relationship did not persist in women when adjusting for lifestyle factors (physical activity, alcohol, smoking), suggesting that these factors contribute more to the association than sleep duration alone. A negative association between sleep duration and BMI, covarying for similar lifestyle factors was shown in a cross-sectional study by Thomas et al. (2009). In contrast to our longitudinal study, however, the majority of their cohort were blue-collar workers, and no gender-specific differences were accounted for. Given the scarcity of data in this area, future studies with a larger sample of women are needed to shed light on the potential gender-specific differences, particularly related to lifestyle factors, that account for the association between sleep duration and obesity.

Several studies have observed that a shorter sleep duration (<7 h per night) is associated with a higher BMI.²⁰³ For example, one study demonstrated that in the general population, BMI was on average 0.35 kg/m² lower for every additional hour of sleep reported.¹²⁵ In another similar cohort to the present study, the association between shorter sleep duration and higher BMI was mediated by longer work hours, stress and physical activity.^{125,192}

Plausible mechanisms to consider for the inverse association between sleep duration and BMI over time have previously been suggested to include stress and behavioural changes in that shorter sleep, results in more stress, less physical activity and poor dietary choices, that can contribute to weight gain. Even after adjusting for psychological factors in the present study, the relationship between sleep and BMI persisted, suggesting that shorter sleep may independently contribute to weight gain over time in both men and women. Shorter sleep duration could lead to the development of obesity through elevated ghrelin levels (a stomach-derived peptide that stimulates appetite) and decreased leptin levels (an adipocyte-derived hormone that suppresses food intake), which together may promote appetite and food intake, ultimately increasing BMI.²⁰⁴ More research is required to assess whether habitual insufficient sleep is truly associated with greater appetite and

greater food intake. While dietary intake was not included in the present analyses, other lifestyle factors, which include physical activity and alcohol consumption, did not appear to impact the sleep – BMI relationship in men

Gender-specific differences in HDL cholesterol levels have previously been attributed to the effect of sex hormones (oestrogen, in particular) on lipoprotein metabolism by increasing lipoprotein transport in women and consequently yielding a greater generation of HDL compared to men.¹⁷⁸ Indeed, the women in this cohort showed a significant, but weak longitudinal association between shorter sleep duration and lower HDL concentrations; one might therefore speculate that longer sleep duration may provide some CMD protection in women over time. Since this association disappeared when adjusting for lifestyle, occupational and stress factors, however, the sleep-HDL cholesterol relationship may be mediated by other external factors beyond the scope of this study.

An inverse association between self-reported sleep duration and the composite CMD risk score has previously been reported in the general public.^{18,66} This association in our male cohort was independent of lifestyle and psychological factors, but not age, or age and occupational factors (i.e. work hours (≥ 60 h/week), commute time to work). As such, it may be that older age, longer work hours or work commute time may be more directly linked to CMD risk in men. Investigating CMD risk in employees has also been studied by Buxton et al. (2018), in which objective and subjective sleep health indicators were found to be associated with lower CMD risk, but only in low- to middle-wage workers.²⁰⁵ While the Buxton et al. study extrapolated CMD risk from the Framingham Risk score and was cross-sectional in nature, the present study with its longitudinal design and use of a composite cardiometabolic disease risk score in a unique subgroup of employees further contributes to our understanding of the role of sleep in the context of CMD health in the context of the workplace.

Longer work hours may impact sleep opportunity. There is evidence to suggest that working more than 55 h/week, compared to 35–40 h/week, is associated with 1.98 times higher odds for shorter sleep duration (<7 h per day), and that repeated exposure to longer work hours is associated with up to 3.24 times higher odds for shorter sleep duration over time.²⁰⁶ Moreover, results from a meta-analysis on long work hours and occupational health showed the strongest association with short sleep duration (<6 h/day).²⁰⁷ Further, in a previous cross-sectional, we showed that among corporate executive men, long work hours (≥ 60 h/ week) mediated the relationship between self-reported sleep duration and CMD risk.¹⁹² Similarly, when the association between being a manager and CVD risk factors were examined by Ikesu et al. (2021), it was found that over a four-year follow-up, managers were less likely to report sufficient sleep, compared to non-managers.²⁰⁸ Our longitudinal findings suggest that over time, longer work hours may have a more direct relationship with CMD risk than shorter sleep duration in this subset of the workforce.

Finally, one may also consider the work culture within corporate settings, specifically the attitude towards sleep health. This has been demonstrated by Soprovich et al. (2021), where working men's perspectives of

sleep health indicated that they accustomed themselves to sleep deprivation, and that their work culture perpetuated that working more, and thus sleeping less, showed commitment and dedication.^{209,210} Additionally, there is also evidence to suggest that many workplaces have adapted to working overtime,²¹¹ resulting in an obligation to work additional hours in order to demonstrate leadership, potentially at the expense of healthy sleep habits. Our findings demonstrate the need to enable engagement around a healthy workplace culture and attitude toward sleep.

This study offers preliminary evidence for practical implications around sleep behaviour to protect the long term health of corporate employees. Specifically, employee HRAs should include robust assessments of sleep habits to flag those at risk for short, poor quality sleep. Education around the risks of displacing sleep opportunity with work hours should be included in workplace health programmes. Given the conflict between the corporate workplace culture and healthy sleep habits, efforts to promote healthy attitudes towards sleep at the leadership level may result in trickle down behaviour change in sleep behaviours among employees.

A limitation of the study is that the data collected to describe the sleep, occupational, lifestyle and psychological characteristics of the cohort were self-reported and obtained from pre-existing HRA questions which meant that certain variables were limited to categorical instead of continuous variables (e.g., absenteeism, presenteeism, work hours). Future work would therefore benefit from incorporating objective measures of habitual sleep and physical activity, such as accelerometry, and using questionnaires that are less restricted to categories and ranges. Sedentary behaviour and dietary intake measures should be included in future studies given their associations with CMD risk, while noise and light pollution also warrant consideration since this too may disturb sleep. Nevertheless, the data obtained for this study were from standardised corporate executive HRAs, which may serve in measuring health changes over time and enable companies to benchmark comparable data.

Secondly, as this was a study intentionally conducted in a unique subset of the workforce, findings cannot be generalized to the broader population. The findings do, however, provide insight into the relationship between sleep with CMD risk over a time period in a specific sample of non-shift working corporate executives, and future studies may extend into different employment levels allowing for a greater understanding of these relationships in working adults. Thirdly, we did not account for underlying chronic diseases other than CMD, nor symptoms of depression and anxiety. Consequently, there is potential for confounding by stimulants, depressants or psychoactive medications that affect sleep, mental health, or that may interact to affect blood pressure or lipid profiles.

A strength of this study is that the analysis was based on a relatively large sample of well-characterized corporate executives, who came from similar working backgrounds and who participated in at least two identical annual HRAs and face-to-face clinical assessments. This provides subjective and objective measures, coupled with a standardized routine data collection procedure, which increases the validity of our results.

Moreover, results were robust to adjustments for multiple potential confounders, such as lifestyle, occupational and psychological factors.

4.5 Conclusion

These longitudinal data suggest that over time corporate executives who report shorter sleep are more likely to present with worse CMD risk profiles, even when accounting for covariates such as age, physical activity, alcohol consumption, smoking, work and commuting hours, depression, stress and anxiety. Incorporating workplace sleep health programmes may mitigate risk for CMD.

Chapter 5

Associations between habitual sleep characteristics
and cardiometabolic disease risk in corporate
executives

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Submitted

Objectives

Corporate executive job demands may lead to poor sleep habits, increasing their risk for cardiometabolic disease (CMD). The aim of this study was to describe and explore associations between objectively-measured habitual sleep characteristics and CMD risk of corporate executives, while accounting for occupational, psychological and lifestyle factors.

Methods

Habitual sleep was measured using wrist-worn actigraphy and a sleep diary over seven consecutive days in 61 (68.3% men) corporate executives aged 46.4 ± 8.7 y. A composite CMD risk score was determined using body mass index (BMI), waist circumference, blood pressure and fasting glucose and lipid concentrations. Linear regression models were built using a backward stepwise selection approach to explore associations between sleep characteristics and CMD risk factors adjusting for occupational, psychological and lifestyle covariates.

Results

Average total sleep time (TST) was 6.60 ± 0.75 h, with 51.7% of participants reporting poor sleep quality and 26.2% being classified as catch-up sleepers. Lower sleep efficiency (β with 95% confidence intervals: -0.25 [-0.43; -0.08, $P=0.006$]) and shorter weekday TST (-1.40, -2.48, -0.31; $P=0.013$) were associated with higher CMD risk scores. Shorter average time-in-bed (2.00, -3.76; -0.18, $P=0.031$), average TST (1.98, -3.70; -0.25, $P=0.025$) and weekday TST (-2.63, -4.47; -0.80, $P=0.006$) were associated with higher BMIs. Being a catch-up sleeper was associated with both a higher CMD risk scores (2.40, 0.46; 4.35, $P=0.002$) and BMI (5.16, 1.54; 8.78, $P=0.006$).

Conclusion

Corporate executives with shorter weekday TST may not be meeting their sleep need, as evident by catch-up sleep behaviour, thereby increasing their risk for obesity and future CMD.

Keywords: employees; cardiometabolic disease; health risk assessment; actigraphy; sleep quantity

5.1 Introduction

It has been well established that regularly obtaining fewer than 7 h or more than 9 h of sleep per night is associated with detrimental cardiometabolic disease outcomes.^{89,212} More recently, indicators of sleep variability such as social jet-lag, catch-up sleep and week-to-weekend variability in sleep duration have also become recognised as important sleep health factors.^{213,214} For example, in a recent systematic review, daily intraindividual variability in sleep duration and timing in the general population was associated with poorer physical and mental health outcomes, such as higher body mass index, body weight gain, depression symptomology and stress.²¹⁵ When prospectively investigating the association between irregular sleep patterns and metabolic abnormalities, Huang et al. (2019) showed that irregular sleep duration and timing were associated with higher risk of cardiometabolic disease.²¹⁶ Additionally, a recent study in adults investigated sleep duration and wake-up time on work days compared to free or weekend days and found that sleep duration was being extended on weekends along with a delay of wake-times,²¹⁷ suggesting an attempt to recover from insufficient sleep experienced during the week. A proposed driver of variations in sleep duration may be attributed to different work and social obligations experienced during the week and weekend,²¹⁸ thus making working adults particularly vulnerable to the consequences of poor sleep health relative to the general population.

Social norms at work may promote a demanding workplace culture whereby meeting one's sleep need is portrayed as being 'for the weak', and sleeping less as a 'badge of honour'.^{139,219} This attitude toward sleep, often present at managerial levels of businesses, has allowed studies to shed light on leadership effectiveness and organisational performance in the context of sleep health. After surveying sleep patterns, beliefs, attitudes, and problems among business leaders and professionals across different leadership levels, it was evident that respondents believed that successful executives sleep less than the average employee, and that they borrow time from sleep to meet work demands.⁷⁴ This behaviour is contrary to that which would support key elements of effective leadership, as it is well established that good sleep health optimizes brain function and human performance, and plays a crucial role in attention, decision-making, problem-solving, and emotion regulation.¹³⁹ In combination with evidence to suggest that self-reported short sleep duration (<7 h) in corporate executives is associated with increased CMD risk mediated by long work hours and stress,¹⁹² it is necessary to explore the sleep habits of such individuals, and to investigate whether their objectively measured sleep characteristics may be exposing them to an increased risk for poor health outcomes. Furthermore, stress and burnout predominate in jobs characterised by high workloads and in office employees who work at high pace and under pressure.²²⁰ Given this high job demand, time pressures and elevated stress, it is unsurprising that corporate executives often extend work hours at the expense of sleep. Whether these business leaders compensate for lost sleep during weekends remains unclear, as to our knowledge, there are limited data about the objectively measured sleep habits of corporate executives.

The cognitively demanding work typically characteristic of corporate executive jobs may contribute to daytime fatigue, consequently affecting work performance over time. Therefore, another concept to be examined among this group is the need for recovery, which refers to the need to recuperate from a day's work and unwind from work-induced effort.²²¹ While it is recognised that having adequate sleep is associated with reduced daytime fatigue,⁸⁰ it is also an important component of cognitive and mental recovery.^{222,223} Further, a higher need for recovery has been associated with sleep complaints, work-stress and an increased risk for developing burnout, cardiovascular disease and musculoskeletal disorders.^{221,224} To date, only a few studies have explored the relationship between sleep characteristics and the need for recovery from work,^{224,225} and these studies have been conducted in shift workers. Thus, how the need for recovery relates to sleep among corporate executives remains to be examined.

It is known that the sleep health of working adults may be influenced by occupational (e.g. work hours, travel time) and psychological (e.g. depression, anxiety, stress) factors.^{83,170} While research conducted in the general population, in shift workers and within laboratory settings has helped us appreciate the impact of poor sleep on cardiometabolic health, obesity, work productivity and occupational health,^{4,80,226} there is a distinct paucity in similar research available on business leaders.⁷⁴ For this reason, the aim of this study was to describe and explore associations between objectively-measured habitual sleep characteristics and cardiometabolic disease risk of corporate executives, while accounting for occupational, psychological and lifestyle factors.

5.2 Methods

Participants

Sixty-one corporate employees who participated in their workplace's annual health risk assessment (HRA) in 2018 or 2019 were recruited for this study. All were from senior and executive management positions from different industry sectors, namely retail, manufacturing and production, and health. Participants were excluded if they were shift workers. The study was approved by the Human Research Ethics Committee at the University of Cape Town (HREC ref. no: 470/2017) and informed consent was obtained from all participants.

Study design and overview

This cross-sectional study made use of information collected from participants' workplace HRAs, which comprised both a web-based questionnaire and a clinical consultation. Participants completed additional questionnaires specific to this study to assess work ability, need for recovery, sleep quality, daytime

sleepiness, insomnia symptom severity and chronotype. They also wore a wrist-worn accelerometer for seven consecutive days to objectively assess their habitual sleep characteristics, while keeping a sleep diary.

Measures

Measures of daytime fatigue, occupational, psychological and lifestyle factors

These data were collected from the employees' HRA questionnaires. The HRA questions reported on for this study include participant's gender and age and factors describing their daytime fatigue, occupational, psychological and modifiable lifestyle factors. The extent to which participants felt that daytime fatigue interfered with daytime function was assessed with the question 'How often have you experienced sleepiness or fatigue that interfered with your daily activities (work and/or social)?' and the response scale was: (1) rarely or never, (2) a few days a month, (3) a few days a week, (4) every or almost every day.

The occupational factors included hours worked per week, travel time to and from work (min/day), absenteeism and presenteeism. Hours worked per week was stratified into three categories: (1) <40 h/week, (2) 40-60 h/week, and (3) >60 h/week. Absenteeism, defined as absence from work owing to sickness, was measured by a single question "How many days were you absent from work during the last year as a result of illness?". Responses were: (1) None, (2) 1-6 days/year, (3) 7-14 days/year, (4) ≥15 days/year. Presenteeism was measured by the following question: "Over the past 12 months, how often have you gone to work despite feeling that you really should have taken sick leave because of your state of health?" Response options were: (1) Never, (2) 1-2 times/year, (3) 3-4 times/year, (4) ≥5 times/year.

In addition to the occupational factors within the HRA, two additional scales assessed work ability and need for recovery. Work ability refers to a worker's self-rated capacity to meet work demands, including work attitudes and performance. The present study used a valid, reliable single item from the original Work Ability Index tool ("Please rate your present work ability" on a scale ranging from 0 (unable to work) to 10 (work ability at its best), to assess participants perceived present work ability compared to their lifetime best.²²⁷ Perceived work ability was dichotomized into good (8–10 points) and reduced (0–7 points), as done in previous studies.²²⁸⁻²³⁰

The Need for Recovery (NFR) after work scale is a short, valid and reliable (Chronbach's $\alpha = 0.86$)²³¹ measure of early symptoms of work-related fatigue, used in both health surveillance and research settings, at individual, departmental, organisational and national levels. It contains eleven dichotomous items (yes/no) representing short-term effects of a day at work, with questions such as "I find it hard to relax at the end of a working day" and "When I get home, people should leave me alone for some time". The NFR score was analysed as a dichotomous variable (high NFR: scores >6), as previous studies have shown that having a score above this cut-off point places individuals at a higher risk for developing psychological complaints compared to those with a lower score.²³¹

Psychological factors were assessed using the Depression, Anxiety and Stress Scale (DASS)-21, which consists of 21 statements measuring three subscales (depression, anxiety and stress) as experienced over the past week.¹⁶⁸ Each subscale comprises seven items measured on a four-point scale, ranging from (0) Did not apply to me at all, to (3) Applied to me very much, or most of the time. The DASS-21, which is a shortened version of the DASS-42, has been validated in non-clinical samples,^{169,170} and psychometric properties of this instrument have previously been reported to have Cronbach's α scores of 0.86 for depression, 0.90 for anxiety; and 0.92 for stress.¹⁷¹ Each subscale score was the sum of its seven items multiplied by a factor of two to yield equivalent scores to the full DASS-42, with scores ranging from 0 and 42 and severity ratings as shown in Supplementary Table S5.1 (Appendix 4). Outcome variables included continuous scores for depression, anxiety and stress as well as binary variables for raised depression (≥ 10), anxiety (≥ 8) and stress (≥ 15) scores based on cut-off scores proposed the authors who developed the scale.¹⁷²

The modifiable lifestyle factors included physical activity (min/week), smoking status and alcohol consumption. Participants reported the number of physical activity sessions they took part in during a typical week and the duration of each session, from which the average minutes of physical activity per week were calculated. Weekly physical activity duration was analysed as both a continuous and dichotomous variable (low physical activity: < 150 min/week). Smoking status response options were "current smoker", "ex-smoker" or "never smoked", but dichotomised for the purpose of this study (current smoker vs. current non-smoker). Employees provided information on alcohol consumption by choosing from the following: (1) never consume alcohol, (2) < 14 alcoholic drinks/week, (3) 14-21 drinks/week, and (4) > 21 drinks/week. As nobody reported on consuming more than 21 drinks per week, alcohol consumption was analysed using the first three categories.

Cardiometabolic disease risk factors

Standing height was measured to the nearest cm, and body weight to the nearest 0.1 kg. Body mass index (BMI) was calculated as body weight divided by height squared (kg/m^2) and participants were classified as obese if $\text{BMI} \geq 30 \text{ kg/m}^2$. Waist circumference (WC) was measured around the midpoint of the lowest rib and iliac crest, to the nearest 0.1 cm. Resting systolic blood pressure (SBP, mmHg) and diastolic blood pressure (DBP, mmHg) were measured using a mercury sphygmomanometer and cuff size appropriate to the participant's arm circumference. Following a 10 h overnight fast, venous blood samples were drawn and sent to a clinical pathology laboratory (Pathcare, Lancet or Ampath Laboratories) to measure fasting plasma glucose (Glu, mmol/L), high density lipoprotein-cholesterol (HDL, mmol/L) and triglyceride (TG, mmol/L) concentrations. Binary CMD risk factor variables were categorized using the following cut-off values proposed by the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III):²³² high WC: women > 88 cm, men > 102 cm; high TG: ≥ 1.7 ; low HDL: women < 1.3 , men < 1.0 ; high BP: ≥ 130 mmHg

SBP or ≥ 85 mmHg DBP or drug treatment for hypertension; and high fasting Glu: ≥ 5.6 or drug treatment for diabetes.

A continuous CMD risk score to estimate overall CMD risk was calculated for each participant, using the sum of standardized z-scores for fasting serum HDL, plasma Glu, serum TG, BMI, WC, SBP and DBP as follows:

$$\text{CMD risk score} = -z\text{HDL} + z\text{Glu} + z\text{TG} + [(z\text{BMI} + z\text{WC})/2] + [(z\text{SBP} + z\text{DBP})/2]$$

In contrast to using dichotomous metabolic syndrome characteristics, this approach provides a continuous risk score which increases statistical power, and has been used in previous studies.^{18,66} A higher score indicates a less favourable CMD risk profile.

Measures of sleep

Subjective measures of sleep

Self-reported sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). It is a validated and widely used tool comprising 19 questions, which give a global sleep quality score.⁴⁹ In this study scores of ≤ 5 were categorised as good sleep quality in accordance with the original PSQI report, wherein the authors concluded that a global PSQI score of ≥ 6 yielded a diagnostic sensitivity of 89.6% and specificity of 86.5% in distinguishing good versus poor sleepers.^{49,233,234} The Epworth Sleepiness Scale (ESS) measures an individual's general level of daytime sleepiness.⁵¹ Based on eight questions, an individual is assigned a daytime sleepiness score of between 0 and 24, with higher scores indicating greater levels of daytime sleepiness. Excessive daytime sleepiness was defined as an ESS score of >10 . The Insomnia Severity Index (ISI) is a validated, brief, self-report instrument measuring one's perception of insomnia symptoms. It comprises seven items rated on a 0–4 scale (4 indicates greater severity), the sum of which yields a total ISI score ranging from 0 to 28, with scores of 0–7 categorized as no clinically significant insomnia; 8–14 as sub-threshold insomnia; 15–21 as moderate insomnia; and 22–28 as severe insomnia.⁵⁰ ISI scores were presented as continuous and binary (clinical insomnia: ISI score ≥ 15) outcome variables.

Chronotype was assessed using the Horne-Östberg Morningness-Eveningness Personality questionnaire.⁴⁴ This widely used tool consists of 19 questions addressing preferred rising and bed times and preferred times of physical and mental performances. Lower scores indicate eveningness and higher scores indicate morningness. In addition to the continuous score, individuals are also categorized as definite evening-type (DET, scores of 16-30), moderate evening-type (MET, 31-41), neither-type (NT, 42-58), moderate morning-type (MMT, 59-69), and definite morning-type (DMT, 70-86). For the purpose of this study, chronotype was also analysed as a binary variable (being a morning-type or not) as only one participant had a score representative of being an evening-type and the remainder that were not morning-types, were in the neither-type category.

Objective measures of sleep

Habitual sleep characteristics were measured objectively using wrist-worn actigraphy. Participants wore an Actiwatch Spectrum Plus (Philips Respironics, Bend Oregon, USA) on their non-dominant wrist for seven consecutive days and were instructed to press the marker button to indicate the start and end of each nocturnal sleep period. Simultaneously, participants kept a sleep diary to assist with manually marking sleep periods. For a dataset to be valid, a minimum of three weekday nights and one weekend night each with 24 h of uninterrupted activity recording was required. Data were analysed using Philips Respironics Actiware software (v5.61, Bend Oregon, USA). Sleep periods were manually determined using the guidelines published by Chow et al.²³⁵ Outcome variables include average (i.e. seven-day) bedtime, wake-up time, time-in-bed (TiB, h), total sleep time (TST, h), sleep onset latency (SOL, min), sleep efficiency, defined as the proportion of time spent asleep relative to the amount of time spent in bed (SE, %), wake after sleep onset (WASO, min), arousal index (no. arousals/h of TST), midpoint of sleep, bedtime regularity, wake-up time regularity, TiB regularity, and catch-up sleep duration (h). These variables are also presented for weekday nights (Sunday through Thursday) and weekend nights (Friday and Saturday). Midpoint of sleep was determined by adding half of the TiB value to bedtime. To assess regularity in sleep timing (bedtime and wake-up time) and TiB, we calculated the standard deviation for each variable across all nights for each participant, with smaller values reflecting more consistent sleep habits. Catch-up sleep was determined as the difference between weekend and weekday TiB, with larger values (i.e. longer sleep on weekends) indicating more catch-up sleep. Participants were categorized as catch-up sleepers if their catch-up sleep duration was >1.5 h.

Data and statistical analyses

Descriptive data are presented as mean \pm standard deviation, median (interquartile range) or count (percentage). The Shapiro-Wilks test was used to assess normality of data. Comparisons between weekday and weekend nights were tested using dependent t-tests or Wilcoxon matched pairs tests. Additional analyses to describe the actigraphy-derived sleep of participants included comparisons between men and women using independent t-tests. Owing to the large number of candidate sleep variables and possible covariates, we used a prediction modelling approach to explore associations between sleep and CMD risk factors. First, simple linear regression analyses were used to identify candidate independent variables (sleep factors; age, gender, occupational, psychological and lifestyle covariates) associated with CMD risk score or BMI (step 1). All sleep factors with a P value of less than 0.150 were selected as independent variables for step 2 of the analyses.^{236,237} In step 2, a backward stepwise selection approach was used in which models first included all candidate variables identified in step 1. While the candidate sleep factor was always included in the next models, the weakest covariates were removed one at a time until the final model contained only the sleep factor and any covariates with a P value <0.150. Data were analysed using Stata (v.15, StataCorp, Texas, USA) and significance accepted for P<0.050.

5.3 Results

Descriptive results

Descriptive characteristics of the 61 corporate executives are presented in Table 5.1. Just over half of all participants reported poor sleep quality, with one in ten reporting excessive daytime sleepiness, but only 7% reporting clinically significant (moderate and severe) insomnia symptoms. The chronotype of participants was also skewed towards a preference for mornings. The majority of participants worked 40-60 h/week, reporting minimal absenteeism, but some presenteeism. While nearly one third of participants reported reduced work ability and one quarter a high need for recovery score, half of the participants reported some interference of fatigue on their daytime function. One in five of the participants had raised stress scores and close to 15% of participants had raised depression and anxiety scores. Nearly half of participants were insufficiently physically active but only a few reported smoking (5.3%) or exceeding the 14 unit/week alcohol intake threshold (10.5%).

Table 5.1. Descriptive characteristics of the corporate executives.

	Mean (SD), Median (IQR) or n (%)	n
Age (y)	46.4 ± 8.7	61
No. men	41 (68.3)	61
<u>Self-reported sleep variables</u>		
PSQI score	5.8 ± 2.8	58
Poor sleep quality ^a	30 (51.7)	
ESS score	5.5 (3-8)	61
Excessive daytime sleepiness ^b	6 (10.3)	
ISI score	6 (2-10)	57
Clinically significant insomnia symptoms	4 (7.0)	
HO-MEQ score	59.9 ± 9.1	57
Evening- or Neither-type ^d	24 (42.1)	
Morning-type ^e	33 (57.9)	
<u>Occupational factors</u>		
Work hours per week		57
<40 h	2 (3.5)	
40 – 60 h	50 (87.7)	
>60 h	5 (8.8)	
Daily work commute time (min/day)	45.0 (25.0-77.5)	56
Days absent in the past year		57
None	27 (47.4)	
1–6	27 (47.4)	
7–14	2 (3.5)	
≥15	1 (1.8)	
Presenteeism in the past year		54
Never	19 (35.2)	
1-2 times	22 (40.7)	
3-4 times	9 (16.7)	
≥5 times	4 (7.4)	
Reduced work ability^f	18 (30.5)	59
High NFR^g	15 (25.9)	58
Fatigue interfering with daytime function		56
Rarely or never	28 (50.0)	
A few days a month	16 (28.6)	
A few days a week	8 (14.3)	
Every or almost every day	4 (7.1)	

Psychological factors

DASS-21 depression score	2 (0-6)	57
Raised depression score	9 (15.8)	
DASS-21 anxiety score	2 (0-4)	57
Raised anxiety score	8 (14.0)	
DASS-21 stress score	8 (4-13)	57
Raised stress score	12 (21.1)	

Lifestyle factors

Physical activity (min/week)	150.0 (80.0-270.0)	47
Low physical activity ^h	23 (48.9)	
Current smoker	3 (5.3)	57
Alcohol intake		57
None	13 (22.8)	
<14 units/week	38 (66.7)	
≥14 units/week	6 (10.5)	

Data are presented as mean ± standard deviation, median (interquartile range) or count (%). PSQI: Pittsburgh Sleep Quality Index, ESS: Epworth Sleepiness Scale; ISI: Insomnia Severity Index; HÖ-MEQ: Horne-Östberg Morningness-Eveningness Personality Questionnaire, NFR: Need for recovery, DASS-21: Depression, Anxiety and Stress Scale-2, ^aPSQI ≥6. ^bESS >10. ^cISI >14. ^d Evening-type: HO-MEQ score 16 – 41 and Neither-type: HO-MEQ score 42-58; ^eMorning-type: HO-MEQ score ≥59. ^fReduced work ability: >7. ^gHigh need for recovery: ≥6. ^hLow PA: <150 min/week. Categories for depression, anxiety and stress scores are presented in Supplementary Table S5.1 (Appendix 4).

Table 5.2 displays the CMD risk factors of participants. Just less than half (43.9%) of the group was categorized as being obese and having high WC values, the latter indicative of abdominal obesity. Approximately one quarter of participants was classified as having elevated blood glucose levels (26.8%) and blood pressure (25.4%), and nearly one third had elevated TG levels (29.3%).

Actigraphy-derived nocturnal sleep habits of the participants are presented in Table 5.3. Forty three (70.5%) of participants had TSTs shorter than the 7-9 h recommended guidelines. On the weekends, participants went to bed later ($P=0.002$), woke up later ($P<0.001$), spent more time in bed ($P<0.001$), accumulated a longer TST ($P=0.002$), fell asleep more quickly ($P=0.042$), had a longer WASO time ($P=0.017$) and a delayed midpoint of sleep ($P<0.001$) compared to weekdays. Sixteen (26.23%) participants were classified as catch-up sleepers, with median catch-up sleep being 0.6 (IQR: -0.19, 1.51) h in length. Additional analyses showed that women had a longer average TST (7.0 ± 0.61) h and catch-up sleep duration (1.0 ± 1.10 h) compared to men (TST: 6.4 ± 0.75 h; catch-up sleep duration: 0.33 ± 1.14 h, $P<0.05$). Similarly, women had a longer weekend TST (7.7 ± 0.94 h) compared to men (6.6 ± 1.02 h, $P<0.001$). Median bedtime regularity was 0.8 (0.57-0.99) h and that for wake-up time was 0.7 (0.56-0.95) h, with average TiB regularity being 1.0 ± 0.34 h.

Table 5.2. Cardiometabolic disease risk factors of the corporate executives.

	Mean (SD), Median (IQR) or n (%)	n
BMI (kg/m²)	28.7 (26.3-33.1)	57
Obese^a	25 (43.9)	
WC (cm)	94.9 (87.0-106.0)	58
High WC ^b	25 (43.9)	
Glucose (mmol/L)	5.1 (4.8-5.6)	56
Elevated glucose ^c	15 (26.8)	
TG (mmol/L)	1.2 (0.8-1.8)	58
Elevated TG ^d	17 (29.3)	
HDL (mmol/L)	1.2 (1.0-1.5)	58
Low HDL ^e	7 (12.1)	
SBP (mmHg)	120 (115-128)	59
DBP (mmHg)	80 (71-82)	59
Elevated BP ^f	15 (25.4)	
CMD risk score	-0.6 (-2.5-2.0)	55

Data are presented as median (interquartile range) or count (%). BMI: body mass index, WC: waist circumference, TG: triglycerides, HDL: high density lipoprotein, SBP: systolic blood pressure, DBP: diastolic blood pressure, CMD: cardiometabolic disease. ^aBMI ≥ 30 kg/m²; ^bWC >102 cm for men and >88 cm for women; ^c Glucose ≥ 5.6 mmol/L or drug treatment for diabetes. ^dTG >1.7 mmol/L; ^eHDL <1.0 mmol/L for men and <1.3 mmol/L for women; ^fSBP ≥ 130 and/or ≥ 85 DBP mmHg or antihypertensive drug treatment.

Table 5.3. Actigraphy-derived habitual sleep characteristics of corporate executives (N=61).

	Average sleep	Weekday sleep	Weekend sleep	P value
Bedtime (hh:mm)	22:41 ± 00:46	22:34 ± 00:49	22:59 ± 01:04	0.002
Wake-up time (hh:mm)	06:14 ± 00:41	06:03 ± 00:42	06:39 ± 00:47	<0.001
TiB (h)	7.64 ± 0.72	7.42 ± 0.85	7.95 ± 1.02	<0.001
TST (h)	6.60 ± 0.75	6.46 ± 0.85	6.91 ± 1.11	0.002
SOL (min)	8.3 (4.5-15.4)	7.6 (4.3 – 14.1)	5.6 (1.6 – 11.8)	0.042
SE (%)	87.7 (83.4-90.4)	87.4 (84.6 – 90.8)	88.2 (83.5 – 91.5)	0.872
WASO (min)	27.8 (22.8-37.3)	27.0 (21.5 – 32.4)	30.5 (21.6 – 40.3)	0.017
Arousal index (arousals/h)	6.5 (3.9-11.5)	6.4 (5.2 – 8.9)	6.4 (5.2 – 8.8)	0.908
Midpoint of sleep (hh:mm)	02:24 ± 00:38	02:15 ± 00:36	02:55 ± 00:58	<0.001

Data are presented as mean ± standard deviation or median (interquartile range). TiB: time-in-bed; TST: total sleep time; SOL: sleep onset latency; SE: sleep efficiency; WASO: wake after sleep onset. P values represent comparisons between weekday and weekend nights using dependent t-tests or Wilcoxon matched pairs tests.

Associations between sleep characteristics and CMD risk

Using simple linear regression analyses, 13 candidate independent variables (age, gender, seven sleep, one occupational and three psychological factors) met our inclusion criteria for prediction modelling of CMD risk ($P < 0.150$): average TiB; average TST; average SE; average arousal index; weekday TST; chronotype category; being a catch-up sleeper; high NFR score and raised depression, anxiety and stress scores (Supplementary Table S5.2, Appendix 4).

In step 2 (Table 5.4), final models indicate that a lower average SE (Model 3, $\beta = -0.25$, 95%CI: -0.43; -0.08, $P = 0.006$), shorter weekday TST (Model 5, $\beta = -1.37$, 95%CI: -2.41; -0.32, $P = 0.011$) and being a catch-up sleeper (Model 7, $\beta = 2.57$, 95%CI: 0.65; 4.50, $P = 0.010$) were all associated with a higher CMD risk score. Age contributed significantly to models 2 to 5, gender to models 1, 6 and 7, and raised stress scores to model 7. High NFR, raised depression and raised anxiety scores did not contribute significantly to any of the final models, and were therefore not retained as covariates. Model 7 explained most of the variance in CMD risk score (25%).

Associations between sleep characteristics and body mass index

Simple linear regression analyses indicate that the following eight independent variables (four sleep, two psychological and two lifestyle factors) met our inclusion criteria for prediction modelling of BMI: average TiB; average TST; weekday TST; being classified as a catch-up sleeper; raised anxiety and stress scores; low physical activity level; and alcohol consumption (Supplementary Table S5.2, Appendix 4).

In step 2 (Table 5.5), final models indicate that shorter average TiB (Model 1, $\beta = -2.00$, 95%CI: -3.76; -0.18, $P = 0.031$), shorter average TST (Model 2, $\beta = -1.98$, 95%CI: -3.70; -0.25, $P = 0.025$), shorter weekday TST (Model 3, $\beta = -2.13$, 95%CI: -3.56; -0.69, $P = 0.004$) and being a catch-up sleeper (Model 4, $\beta = 4.12$, 95%CI: 1.20; 7.10,

P=0.006) were all associated with a higher BMI. Of the covariates identified in step 1, having a raised stress score contributed significantly to all the models, while raised anxiety scores, low physical activity levels and alcohol consumption were not retained in the final models. The strongest among these models was model 3, which explained 19% of the variance in BMI.

Table 5.4. Final linear regression models assessing associations between sleep factors and cardiometabolic disease risk score in corporate executives (Step 2).

	<u>Model 1</u>	<u>Model 2</u>	<u>Model 3</u>	<u>Model 4</u>	<u>Model 5</u>	<u>Model 6</u>	<u>Model 7</u>
	Average TiB	Average TST	Average SE	Arousal index	Weekday TST	Chronotype	Catch-up sleeper
	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
Sleep factor	-0.63 (-2.00; 0.70)	-1.24 (-2.51; 0.04)	-0.25 (-0.43; -0.08) **	0.32 (-0.04; 0.69)	-1.37 (-2.41; -0.32) *	1.37 (-0.56; 3.30)	2.57 (0.65; 4.50) *
Age		0.11 (0.01; 0.22) *	0.16 (0.06; 0.27) **	0.12 (0.01; 0.23) *	0.12 (0.02; 0.23) *	0.10 (-0.01; 0.21)	
Gender	2.46 (0.37; 4.54) *	1.70 (-0.45; 3.84)		1.74 (-0.42; 3.90)	1.65 (-0.33; 3.63)	2.23 (0.12; 4.34) *	2.78 (0.90; 4.65) **
Raised stress score	-2.2 (-4.6; 0.09)						-2.53 (-4.62; -0.44) *
Model statistics	N=53, F=4.19, P=0.010, r ² =0.155	N=53, F=5.16, P=0.004, r ² =0.194	N=53, F=7.17, P=0.002, r ² =0.192	N=53, F=4.89, P=0.005, r ² =0.183	N=54, F=6.61, P<0.001, r ² =0.241	N=50, F=4.34, P=0.009, r ² =0.170	N=54, F=6.84, P<0.001, r ² =0.248

TiB: time in bed, TST: total sleep time, SE: sleep efficiency, β : beta coefficient; CI: 95% confidence intervals. Parameters shown are independent variables that retained significance using a backward stepwise selection approach. *P<0.05, **P<0.01

Table 5.5. Final linear regression models assessing associations between sleep factors and body mass index in corporate executives (Step 2).

	Model 1	Model 2	Model 3	Model 4
	Average TiB	Average TST	Weekday TST	Catch-up sleeper
	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
Sleep factor	-2.0 (-3.76; -0.18) *	-1.98 (-3.70; -0.25) *	-2.13 (-3.56; -0.69) **	4.12 (1.20; 7.01) **
Raised stress score	-3.56 (-6.96; -0.16) *	-3.72 (-7.10; -0.35) *	-3.52 (-6.64; -0.39) *	-4.39 (-7.56; -1.22) **
Model statistics	N=55, F=5.25, P=0.008, r ² =0.136	N=55, F=5.48, P=0.007, r ² =0.142	N=56, F=7.40, P=0.002, r ² =0.189	N=56, F=6.97, P=0.002, r ² =0.178

β: beta coefficient; CI: 95% confidence intervals. Parameters shown are independent variables that remained significant in the model. *P<0.05, **P<0.01.

5.4 Discussion

This study sought to characterise the sleep, occupational, psychological and lifestyle characteristics of corporate executives and specifically explore the relationships between both subjective and objective sleep factors with CMD risk. Descriptively, it was evident that the majority (71%) of the participants sleep less the recommended sleep duration guidelines of 7-9 h per night,²⁶ with at least half of the group (52%) reporting poor sleep quality. Further, we may speculate that the average nocturnal weekday TST of 6.5 h did not meet the personal sleep need during the work week of many participants, since one quarter of the group extended their sleep duration by at least 1.5 h on weekends, suggestive of catch-up sleep, and one quarter reported a high need for recovery.

Our main findings pertaining to the associations between sleep characteristics and CMD risk suggest that shorter actigraphy-derived weekday TST and displaying catch-up sleep on weekends are associated with both a higher CMD risk score and BMI. Although the adverse effects of self-reported short sleep on cardiometabolic health are increasingly recognized, we show that specifically objectively measured a potential sleep deficit during the working week of corporate executives is associated with higher risk for CMD and greater adiposity.

While insufficient sleep in employed adults has previously been shown to negatively impact cardiometabolic health,²³⁸ studies describing these adverse outcomes have usually been carried out in a shift working cohort. For example, relative to daytime workers, shift workers may experience chronic circadian misalignment and are more likely to develop metabolic syndrome.²³⁹ One review has shown that night shift work was significantly associated with an increased risk for metabolic syndrome along with a positive dose–response relationship with duration of exposure.⁷¹ Accordingly, these studies report on circadian misalignment which results from the variation in sleep timing experienced by shift workers. This contrasts with the present study where in non-shift working adults, the variation observed was due to changes in week to weekend sleep duration as opposed to differences in sleep timing. Also, in studies of non-shift working adults, social jetlag is commonly used as the sleep characteristic to describe the sleep – cardiometabolic health relationship.²⁴⁰ As such, this too refers to variation in sleep timing and not sleep duration differences between week and weekend nights.

The present study utilized both sleep characteristics, namely sleep timing (difference in midpoint of sleep) and sleep duration variation. While we observed that corporate executives had a later midpoint of sleep on weekends compared to the week, this was not associated with CMD risk nor BMI. Instead, the association was unique to shorter weekday TST suggesting that their sleep habits during the working week may be predisposing them to poorer health outcomes. Given that it is often presumed that insufficient weekly TST may lead to “catching up” on sleep during weekends, we also provide evidence that corporate executive

demonstrating catch-up sleep of at least 1.5 h on weekends may be at risk for poorer cardiometabolic health, as in both instances, shorter weekday TST and being a catch-up sleeper was associated with increased CMD risk scores and BMI.

According to the National Sleep Foundation in the US, a sleep efficiency measurement of 85% or more is ideal for optimal health.²⁴¹ Despite the group average sleep efficiency of 87.7%, we found that lower efficiency was associated with an increased CMD risk score. Since sleep efficiency may be regarded as one indicator of sleep quality,²⁴¹ our findings contribute to the literature describing an association between poor sleep quality and adverse cardiometabolic health outcomes^{242,243} and illustrate the importance of considering both sleep quality and duration as key factors when addressing the sleep health of corporate executives.

Psychological factors such as stress and anxiety may interfere with sleep quality of employees with a demanding work schedule,²⁴⁴ and given that a fifth of our corporate participants had high stress scores, and nearly one third reported elevated anxiety, we ensured that these psychological factors were taken into account in our models. Additionally, long work hours may also interfere with sleep duration during the week. This was seen in previous work where we recruited a similar cohort and found that long work hours and stress mediated the significant association between self-reported short sleep duration (<7 h) and CMD risk.¹⁹² The findings of the present study therefore support and strengthen our previous work by using actigraphy-derived sleep duration to show that shorter sleep duration, specifically during the week, is associated with both a higher CMD risk profile and a higher BMI. It is also in line with work by Wong et al. (2015) where it was found that a sleep debt (difference in TST between weekend and weekday) of 1.2 h in non-shift workers was associated with more insulin resistance and higher adiposity levels.²⁴⁵

Shorter average (7-day) sleep duration in these corporate executives was also associated with a higher BMI. Many studies have shown a link between short sleep duration and obesity in the general population, as made evident in a meta-analysis where shorter duration of sleep, irrespective of definitions used in the individual studies, was associated with a significantly increased odds of obesity.²⁴⁶ Similar results have been reported in employed cohorts to show that when compared to 7-8 h of sleep, those reporting less than 6 h were more likely to be obese,²⁴⁷ and within a corporate executive group, self-reported sleep duration of less than 7-9 h was associated with a higher BMI compared to a sleep duration of 7-9 h.¹⁹² While evidence for the association between short sleep duration and obesity is now abundant, to the best of our knowledge, this is one of the first studies among corporate executives to examine associations between objective sleep measures and obesity, taking into account occupational, psychological and lifestyle factors.

While poor sleep is hypothesized to affect each CMD risk component on its own through several distinct biological pathways,^{248,249} the relationship between sleep duration variation between weekdays and weekends, and CMD risk or obesity remain largely unknown. Although a full accounting of potential mechanisms linking short weekday sleep duration and cardiometabolic health outcomes is beyond the scope of our study, it is important to note several possibilities. It has been proposed that the mechanism linking

inconsistent weekday-weekend sleep duration to CMD and obesity risk may be due to the internal desynchrony of the circadian rhythms. Since the circadian clock helps maintain energy homeostasis and coordinates a hierarchy of circadian rhythms, sleep schedule variability may predispose individuals to obesity and adverse CMD outcomes.⁶⁷ Since we did not find significant differences in the association between sleep timing (midpoint of sleep or social jetlag) and either CMD risk or BMI within our participants, the proposition of circadian misalignment with external time does not fit our present findings. Instead, our findings emphasised that it is the shorter sleep duration during the week relative to the weekend that make corporate executives more vulnerable to poor health outcomes. Perhaps a more plausible explanation to consider may relate to the underlying genetic pathways linking insufficient sleep and adverse health outcomes. This was demonstrated by Moller et al. (2013) where in as little as one week, an average sleep duration of 5.7 h (compared to 8.5 h) was sufficient to alter the temporal organization of the human blood transcriptome with 711 genes being up- or down-regulated, leading to changes in the expression of genes linked to metabolism and oxidation, amongst other factors.²⁵⁰ A potential explanation to how insufficient sleep may be associated with an increase in BMI may be related to how sleep impacts energy metabolism. For example, when healthy participants were given only a five-hour sleep opportunity over five days, Markwald et al. (2013) showed that compared to a 9 h sleep opportunity, short sleep increased their energy needs and food intake such that intake was in excess of energy needed, ultimately leading to weight gain in just five days. The participants in our study curtailed their sleep over the five working days of the week; thus one may hypothesise that over time, chronic shorter weekday TST may contribute to the observed association with higher BMI.

Whether recovery sleep or catch-up sleep on free-days or weekends may offset these effects remain to be clarified. To emphasise this, a recent systematic review on sleep timing and consistency, and how it relates to health, reported that catch-up sleep was favourably associated with health outcomes. It should be noted, however, that the review only identified three studies that referred to catch-up sleep²⁵¹ and all three studies made use of self-reported sleep duration. One study used self-reported BMI and another self-reported hypertension as outcome measures. Furthermore, in the studies defining catch-up sleep, one defined it as one or more hours added sleep duration on weekends, and another defined catch-up sleep as sleeping longer on weekends compared to weekdays with no pre-defined cut off. As such, these studies were also deemed a lower quality by the authors of the systematic review. In another study making use of self-reported sleep duration within the general population, authors suggested that engaging in 'catch-up sleep' on the weekend may help ameliorate some of the effects of insufficient sleep during the week.²⁵² Within the population studied, many were older adults beyond the 65-year retirement age, and employment status was dichotomised into 'employed' or 'unemployed' with no distinction between shift or non-shift work. This therefore renders it unclear as to whether the beneficial findings on catch-up sleep are representative within a corporate setting.

Opposing the benefits of catch-up sleep, there is evidence to show that the attempted recovery sleep over weekends does not offset the harmful effects of insufficient weekday sleep.²⁵³ In a randomized, three-group study of healthy adults, researchers assessed sleep, circadian timing, energy intake, weight gain, and insulin sensitivity during (1) sustained insufficient sleep (five-hour sleep opportunities over nine nights), (2) recurrent insufficient sleep following ad-libitum weekend recovery sleep (insufficient sleep over five days followed by two days of weekend recovery and then two nights of insufficient sleep), (3) in a control condition (nine hour sleep opportunity). A weekend recovery sleep of 1.1 h more than the estimated total sleep need from each participant (based on their baseline sleep duration) was not enough to repay their sleep lost during the workweek. As such, it was concluded that despite weekend recovery sleep, the healthy participants' energy intake and body weight increased, and insulin sensitivity reduced after a week of insufficient sleep duration. While the catch-up sleep of corporate executives is yet to be further explored, we provide preliminary evidence suggesting that the potential detrimental effects of insufficient weekday sleep and its associated increased risk for CMD and higher BMI may not be offset by an attempt at catch-up sleep during weekends.

Methodological considerations

A strength of our study is that in addition to measuring the subjective sleep characteristics of our participants, we were able to gain deeper insight into their sleep habits through objectively measured sleep using actigraphy over work and free days. While actigraphy is known to overestimate sleep and underestimate wake time during the sleep period,²⁵⁴ in part because quiet inactivity can be mistaken for sleep, our study minimised this concern by using sleep diaries and event marker buttons to establish the time that participants intended to start their sleep period. Moreover, actigraphy allows for the assessment of large numbers of participants in their everyday environments and over multiple nights, thus increasing its validity. Therefore, the use of actigraphy to quantify sleep measures provides a behavioural measure of sleep patterns that, unlike self-reported questionnaires, is not influenced by retrospective reporting bias.

Acknowledging that this study made use of a cross-sectional design, one cannot infer causal relationships between sleep characteristics and CMD risk or obesity. The sample size is relatively small and given the smaller proportion of women, future studies with a more evenly distributed number of men and women is recommended. Future studies with a larger sample size and a longitudinal design are needed to consolidate and confirm our observations.

Nevertheless, a strength of this study is the inclusion of the well-characterized sample of corporate executives who came from a similar working background and who participated in a standardized routine data collection procedure, which included a HRA comprising both questionnaires and a face-to-face clinical appointment. Moreover, body weight, height, waist circumference, and blood pressure were objectively measured in clinics by trained healthcare providers, and fasting glucose, triglyceride and HDL-cholesterol

levels were ascertained from blood specimens using gold standard in-laboratory techniques. In addition to this, we used data from a unique echelon of the workforce, investigated multiple occupational, psychological, lifestyle and sleep characteristics beyond self-reported sleep duration, included objective measures for sleep, obesity, CMD risk markers, and adjusted for potential confounders.

5.5 Conclusion

Corporate executives' sleep displaying shorter weekday TST and catch-up sleep on weekends appear to have poorer cardiometabolic health. These results support and strengthen the growing literature linking short sleep duration with CMD risk and obesity, by contributing data specific to non-shift working corporate employees. Given this, it is vital for business leaders to manage their sleep habits during the week to mitigate potential long term adverse health effects. Furthermore, these data provide evidence to support the call for organisations to advocate for the incorporation of sleep health management within existing workplace health programmes. Specifically, supporting employees to meet their sleep need during the working week may be protective against adverse CMD risk and obesity. Organisations are encouraged to examine policies and consider interventions that encourage adequate sleep among non-shift work employees.

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Chapter 6

Barriers and facilitators to participation, and key components of sleep health programmes: perspectives for the corporate work environment

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Objective

To explore the barriers and facilitators of participation and key components for sleep health programmes designed for the workplace.

Methods

Semi-structured interviews with corporate executives and occupational medicine specialists in the decision-making and management of workplace health promotion programmes (WHPP) within their companies were held before and during COVID-19. Interviews were transcribed verbatim and analysed using thematic content analysis to identify themes.

Results

Barrier and facilitator themes emerging from the data include sleep health awareness; work culture; work-family balance and confidentiality. Key components for sleep health programmes included: identifying the need for a programme; incorporating sleep health risk screening to WHPP and promoting sleep health by raising awareness thereof.

Conclusion

The identified barriers and facilitators to employee participation and key components of an ideal sleep health programme provide guidance for further WHPP.

Keywords: health promotion programmes, sleep health, workplace, employee perspectives, qualitative research

6.1 Introduction

While the negative consequences of insufficient sleep and poor sleep quality on physical and mental health have been well described,^{255,256} more recently the effects on workplace performance have come to light. In addition to the medical^{3,87} and psychological²⁵⁷ consequences of chronic poor sleep, the social and occupational functioning of working adults can also be impacted. Studies from many countries have found that employees often work after a night of short or poor quality sleep.²⁵⁸⁻²⁶⁰ Poor sleep health has thus been shown to be particularly problematic for employees within managerial positions where it has been reported that 40.5% get less than six hours sleep.⁶ Poor and inadequate sleep results in a variety of cognitive deficits, including an inability to maintain attention, decreased alertness, delayed reaction time, altered emotional and information processing, and a general inability to think clearly.¹⁵⁴ Moreover, it has been recognised that well-rested employees are absent from work less often, perform better when at work, make better decisions, and interact more positively interpersonally.^{137,139,261} From an economics perspective, there is evidence suggesting that healthy employee sleep and corporate success are directly correlated, and consequently, it has been proposed that business leaders seek ways to translate this research into work policies to not only improve employee health, but also to improve their own financial bottom lines.²⁶² Considering the negative impact of poor sleep on the health and work performance of employees, and that it evidently comes at an economic cost to organisations, it is vital to provide effective support with solutions to help prioritize good sleep habits.

Unfortunately, the positive impacts of healthy sleep on work performance may often be underappreciated by employees. Pressure to meet challenging performance objectives drives these employees to extend work hours or to create unrealistic work schedules, frequently favouring the perceived productivity gains of additional work time over the productivity losses that may arise due to suboptimal sleep.¹³⁹ As such, sleep health is not widely prioritized in organisational policies and practices. The role of workplace culture has also been found to impact on employee sleep. There is evidence to show that a workplace culture consisting of high work demands, especially for employees at management level, may indirectly impact sleep via extended work hours and elevated job stress.²⁰⁷ Many companies have adopted Workplace Health Promotion (WHP) Programmes targeting health-risk behaviours such as poor nutrition, physical inactivity, smoking or alcohol consumption as a means to reduce risk for non-communicable diseases or mental health disorders.^{28,263} Programmes that promote sleep health, however, are still lacking or limited, despite strong evidence for the relationship between insufficient sleep and adverse physical and mental health outcomes.^{264,265} Additionally, the success of programmes focussing on lifestyle behaviours are often limited by low participation rates and lack of adherence.^{266,267} In response to these challenges, and to enhance the sustainability of behaviour change, previous research on the success of WHP programmes indicated that employees should be involved in the planning and implementation processes.²⁶⁸ As such, the role of corporate executives is vital in contributing to the adoption and successful implementation of WHP programmes within their organisation,

as they hold the decision-making power in approving such health strategies throughout their business. Further, as business leaders, their involvement in the organisation's health promotion planning, policies and practises mean that their knowledge and past experience hold value in developing and enhancing a successful sleep health programme.

The importance of leadership to the success of employee wellness initiatives within organisations is widely acknowledged, yet the impact of this leadership in promoting sleep health is underrecognized. Given the evidence demonstrating that employees recognise leadership support based on the wellness programmes and policies their employers provide,²⁶⁹ there is opportunity for management to demonstrate their commitment to employee health through ensuring that WHP programmes are acted upon in the workplace. Interviews with managers who play an integral role of programme implementation, can provide valuable insights into the barriers and facilitators to employee participation in WHP programmes, along with what they would consider key components to include in such a programme. Such insights may therefore provide a foundation for developing an ideal framework of sleep health programmes, which ultimately can be adapted beyond the corporate setting into all segments of the business.

Despite the potential contribution toward sleep health management from organisational leaders, one cannot ignore the recent impact that the COVID-19 pandemic played for WHP programmes and sleep health in particular. Many studies have shown that sleep patterns and behaviours changed among the general populace worldwide during lockdown.²⁷⁰⁻²⁷² Earlier studies on the effect of COVID-19 restrictions such as home confinement, showed that these were linked to sleep problems in employees.²⁷³ For example, while some studies showed an increase in sleep duration that may have been due to work flexibility and less commuting time,^{274,275} others reported poor sleep outcomes mainly due to increased stress and anxiety.²⁷⁶ Owing to this, it is important to assess the same perspectives from organisational leaders pertaining to employee participation and the development of sleep health programmes before and during the pandemic.

This qualitative study aims (i) to explore perceived barriers and facilitators to participation in a sleep health programme for corporate executives and (ii) to gain insight from corporate executives and occupational medicine specialists around key components of an ideal sleep health programme that could be implemented as part of a WHP programme. Owing to the COVID-19 pandemic and associated lockdown that took place during data collection, the study was adapted to include a third aim: to re-interview participants after the first wave of the pandemic to determine whether insights and perspectives around a sleep health programme had changed.

6.2 Methods

Study design and setting

This study made use of a qualitative research design based on semi-structured interviews with senior business managers and occupational medicine specialists. This specific setting was chosen since senior business managers who, together with occupational medicine specialists, are typically involved in the decision-making and management of WHP programmes within their companies. Initial interviews were conducted in-person before the first wave of the COVID-19 pandemic in South Africa (February–June 2020) and follow-up interviews took place remotely between the first, second and third waves of the pandemic (December 2020–December 2021). The study was approved by the University of Cape Town’s Human Research Ethics Committee (HREC ref. no: 470/2017) and all participants provided written informed consent upon agreeing to take part in this study.

Participants and recruitment

A convenience sampling strategy was used to recruit participants for this study. Ultimately, seven participants (two men and five women) were interviewed. Of these, five were corporate executives (senior business managers) and two were occupational medicine specialists. The participants were employees of companies in the manufacturing and healthcare business sectors.

Data collection

Where possible, two members of the research team were present during the interviews, which were audio-recorded using a digital sound recording device. The primary researcher (PRP) facilitated the session by explaining the purpose of the study and by guiding the interview using pre-determined, open-ended questions designed to elicit conversation around potential barriers to and facilitators of participation in a sleep health programme, perceptions regarding the need for a sleep health programme among corporate employees, and views on key components in an ideal sleep health programme. The questions used for the follow-up interviews were adaptations of the initial interview questions to allow for consideration of the impact of the COVID-19 pandemic on the previous responses, in relation to sleep health and WHP programmes. The complete question schedule used in both interviews is shown in Supplementary Table S6.1 (Appendix 5). The follow-up interviews were conducted using the Microsoft Teams© application, and in all interviews, the researcher and participant had their laptop camera switched on for a virtual face-to-face session. Both initial and follow-up interviews lasted no longer than 60 minutes and were conducted in English.

Data analysis

Audio files were transcribed verbatim. Thematic analysis was used to analyse the collected data, starting with reading of the transcripts. Two researchers then independently coded the transcripts line-by-line in Microsoft Word (PRP coded all transcripts, AB coded 25% of the transcripts). The coding process was guided by interview topics, utilising a deductive approach. PRP and AB compared and discussed initial codes until consensus was reached, to ensure consistency of the analytical process. Next, codes were explored for similarities and discrepancies, ultimately grouping and combining codes into themes. After extensive discussions within the research team, themes were mapped onto a 'pre- 'and 'during' COVID thematic coding tree. Representative quotes from the interviews were added to illustrate the findings.

6.3 Results

Themes were identified within the two main topics of this study: (1) barriers to and facilitators of participating in a sleep health programme and (2) key components of an ideal sleep health programme designed to be implemented within the corporate work environment. An overview of the themes that emerged can be found in Table 6.1.

Table 6.3. Themes regarding the barriers to and facilitators of participation in a sleep health programme and the key components of an ideal sleep health programme.

Barriers to and facilitators of participating in a sleep health programme
1. Lack of sleep health awareness
2. Demanding workplace culture
3. Work–life balance
4. Confidentiality

Key components of an ideal sleep health programme
1. Identifying the need for a sleep health programme
2. Health risk screening
3. Education and raising awareness

Barriers to and facilitators of participating in a sleep health programme

- 1. Lack of sleep health awareness*

At the corporate level, the lack of awareness and guidance related to sleep health were considered barriers to participation in a sleep health programme. While the importance of sleep for general health was recognized, participants admitted that organisations did not prioritise the development and implementation of sleep health programmes, as they did not know how and where to begin. Further, it was evident from the interviews that despite the general knowledge around sleep health, appreciation of its significance for work performance and cognitive benefits was lacking. To resolve this, interviewees thought about how to overcome this barrier by indicating that it was vital to convey the link between adequate sleep and how it can optimize work performance, particularly to business leaders, as it would facilitate participation and make the business case for sleep health programmes.

Another barrier raised within the context of sleep health awareness was corporate executives denying having sleep problems or simply ignoring their sleep-related concerns, despite the recognition of experiencing exhaustion during the day at work. Participants felt that 'lack of sleep' was standard practice amongst corporate employees and that, although they could fall asleep at any opportunity, indicating their exhaustion, they denied needing any sleep support. It was further mentioned that participation in a sleep health programme would be low if employees did not recognize or admit their difficulties with sleep habits.

“So it would be really beneficial, and I think what happens and in my experience and talking with people in corporate environments is that they become so accustomed to these sleep habits that it's their new norm. And so, it becomes so much difficult to undo, they don't actually realise that the long-term effect of it is extremely bad for them.” (Pre-COVID-19, SI07A)

2. Demanding workplace culture

The workplace culture of organisations was perceived as a barrier to participation in a sleep health programme. Participants expressed that the work demands set at the managerial level may hamper participation in a sleep health programme. This barrier was relevant before and during the COVID-19 pandemic as it pertained to the stress-provoking and demanding management style adopted within the organisation. Workplace culture was governed by expectations of having to be available after hours and to complete work deadlines at short notice, resulting in high work pressure with challenging working days and a lack of time to prioritise sleep or adopt effective sleep management strategies.

“So, there may be expectations of ability, and reasonable availability, productivity driven you must be able to meet particular targets even if it's a weekend, or a Sunday, or at night.” (Pre-COVID-19, SI01A)

During COVID-19, these work expectations were further exacerbated due to the demanding expectations from clients which were experienced by managers across the corporate environment.

“And then also if you have very senior managers sending emails at all hours of the day, or night it kind of sets the tone for everyone else. So, yes there isn’t a verbal expectation, it’s not expressed that you need to be available all hours of the day, or night, or that’s how you should be working, but it’s kind of indirectly saying to everyone you know, that this is okay ...” (During COVID-19, SI05B)

In thinking about ways in which to overcome this barrier, some participants suggested that a change in work policies was needed to allow for more flexibility around work times. Given that they would be more likely to participate if a sleep health programme, or parts thereof, were made available during work hours, work flexibility was considered as a facilitator to participation and would make it easier for employees to prioritise sleep health. Likewise, employees felt that they would participate in a sleep health programme, provided that time spent in daily work meetings was reduced allowing for work to be completed during working hours, which in turn would allow more time for sleep support.

3. Work-life balance

According to participants before and during COVID-19, a proper balance between work hours and family time would be a prerequisite and facilitate participation in a sleep health programme. In contrast, the lack of balance, with work taking over family time, was raised as a barrier. This concern for work-life balance was expressed by emphasizing employees’ resistance to spending the limited available time after work hours on participation in a sleep health programme. Instead, they would rather spend it on family and home responsibilities. This was particularly emphasized in an interview before COVID-19:

“So, it’s difficult to get through the importance of it as a concept because you know, it’s just “Get on with it and sleep.” But it’s also about work-life balance, something we don’t have, and all that sort of thing.” (Pre-COVID-19, SI06A)

“... if it is more efficient, and they do all go on a program, and their sleep is improved perhaps more time at home, or family time...” (During COVID-19, SI07B)

Before the COVID-19 pandemic, most employees were expected to work from their office and as such, would have to wake up earlier to be at work on time, and leave the office later to avoid traffic and long commute times. Given this, many would restrict their sleep duration during their workdays. In contrast, during the pandemic when employees could work from home, this hindering factor was resolved. Yet, despite having the potential opportunity to lengthen their sleep duration, working from home too was accompanied by challenges. Participants felt that they experienced higher work demands and more stress having to combine work time and family care in a day, resulting in a lack of time and energy to invest in a sleep health programme.

As such, another work-related challenge during COVID-19 was related to the changes within the work environment during the pandemic (i.e., working from home). It was felt that the lack of in-person or face-to-face contact would make the implementation of a sleep health programme unfeasible or less likely to be effective and would complicate the monitoring process and inhibit employee participation.

“I think it's harder to implement it remotely, so I think that is a barrier too, because employers have probably got less control over their employees' routines when they are working remotely ...” (During COVID-19, SI03B)

Technology was also implicated in the interference of work-life balance and hinder participation in a workplace sleep health programme. Before COVID-19, the use of technology was associated with a generational gap in that older employees were not keen to participate in a programme if it were too digitalized.

“It depends on the person I think sort of younger people probably like, who are more sort of on their phones would probably take in up better, but then I mean, I've got some older patients who I think you'd end up causing more stress by giving them a sleep app. It's a generational thing I think.” (Pre-COVID-19, SI03B)

During COVID-19, participants reported that organisations struggled with the use of, and access to, technology and online systems, which were required at the time. Therefore, several respondents felt that this could further complicate the implementation of and participation in a sleep health programme that could be provided online. Additionally, it was pointed out that it would be ineffective to provide online support, as it would not reach all employees, or fail to entice those who do not have adequate technological skills.

“So yes, I was saying that I think for me what was the barrier was mainly, you know, the technology that we had to use at the time....” (During COVID-19, SI02B)

A different view regarding online support was given by a few participants, however, they expressed views that technology could support work-life balance and facilitate participation. Specifically, participants felt that easy-to-use mobile apps and virtual interventions would provide a novel and welcoming angle on which to introduce an online approach to sleep health programmes within the organisation. As such, this option would allow employees to access a sleep health programme at times most convenient to them, thus facilitating work-life balance.

“I would think that you could, every second day get together on Zoom, and you would chat about your week, or couple of days that...it would be interesting first of all, to get them to mark their pattern of their sleep, and then you could sort of link up with them to find out how things had changed, then you'd convince them that maybe they needed a sleep thing... ” (During-COVID-19, SI07A)

4. Confidentiality

The issues of privacy and confidentiality were raised before and during COVID-19, as participants felt participation in a sleep health programme would only be viable if full anonymity were guaranteed. Those who perceived lack of confidentiality as a barrier to participation were fearful that the organisation's wellness services may recognize and target them, and that their jobs may be jeopardized as a result. More specifically, they were concerned that their acknowledgment of poor sleep and its effect on their job performance may expose them as being vulnerable, weak and unable to cope with their work demands, ultimately threatening their credibility as business leaders. Another angle was given during COVID-19, where it was felt that online group meetings that discussed sleep health would deter employees from raising their sleep concerns due to the lack of anonymity.

"Very, very senior managers saying like "I will not call in, because who's going to see my file and who's going to know?" So, we're quite fine with calling in about a contract or some kind of legal issue, that's OK, "I don't mind giving you my ID number and you knowing who I am." But when it gets to the more personal things, definitely not OK with accessing what's available." (Pre-COVID-19, SI05A)

"There is such insecurity around will it show my manager that I'm weak, and they don't need me, and they can replace me, I've got to hold on to this job that you know, the job market is, so tough, and it just gets, so yeah, so confused, and they are not making good decisions, because they have not slept." (During COVID-19, SI04B)

Key components of an ideal sleep health programme

1. Identifying the need for a sleep health programme

Given the lack of awareness around the effects of sleep on health and workplace performance at the organisational level, the need for a sleep health programme is not self-evident. Two different approaches to identify the need for a sleep health program were raised. Firstly, the occupational medicine specialists in the group suggested doing a 'needs assessment', which involves the steps prior to WHP screening and implementation. Secondly, the corporate executives in the group proposed identifying the need for a sleep health programme within the organisation based on the lack of sleep health support for employees with sleep problems.

Occupational medicine specialists felt that through a needs assessment, the organisation would emphasise the necessity for such a sleep health programme, and by demonstrating the potential benefits, there would be a greater likelihood of influencing those in management to invest in sleep health.

“So, there is a whole range of stuff in that primary preventive space it's enabling, it's shifting, it's engaging, it's motivating, so that's essential that's a groundwork that's got to be there, and of course part of the needs in that, is to convey that there is a need, the degree of need, and set the scene for intervention.” (Pre-COVID-19, SI01A)

Further, corporate executives interviewed were adamant that employees needed a health programme specific to sleep, as there was inadequate focus on sleep health, nor any process or support they could follow for their sleep concerns. Therefore, they anticipated that employees would value such a programme if provided by their organisation.

“I think employees would just grab it [added by the authors: sleep health programme] and run with it I can tell you that.” (Pre-COVID—19, SI02A)

During COVID-19, occupational medicine specialists and corporate executives within the sample felt that a sleep health programme was needed as no emphasis was given to sleep health. Instead, it was only addressed indirectly through topics on mental health and general fatigue.

“So let me think about the corporate, where mental health and fatigue generally certainly have reached the general conversations at corporate, either by way of online talk sessions or by way of communications. Perhaps not as directly do they talk to sleep, let me say. Typically, it's more around fatigue itself and stress and the demands of working from home, work-life balance difficulties. So sleep perhaps is implied there, but not perhaps as visible as it could be, as a specific component of the fatigue equation.” (During COVID-19, SI01B)

“I think it's almost more important than it was before when we were having our normal way of life if you want to put it that way. I think we are desperate for better sleep, or good quality sleep now...” (During COVID-19, SI05B)

2. Sleep health risk screening

Both before and during COVID-19, participants felt that there was a requirement for identifying signs of daytime fatigue from lack of sleep, and symptoms of poor sleep, by a process of sleep health risk screening. Occupational medicine specialists gave their perspective from an organisational level, which involved large scale screening procedures allowing for groups of employees to be stratified based on data collected about their sleep health status (e.g., sleep duration, sleep quality). The remaining participants reflected on sleep health risk screening at employee level with reference to identifying individual employees and providing them with the sleep health support they would need. Yet in both instances, the shortage of sleep-specific screening to detect poor sleep health was made evident. During COVID-19, interviewees felt that sleep health risk screening and the appropriate management for those affected, be mandatory or at least prioritised. The

suggestion from some participants during follow-up interviews was that in response to sleep health risk screening, employees could have access to an online consultation regarding their sleep health risk.

“... any sort of occupational health program there would be sort of a screening aspect to it, kind of you know, screening out people who are at risk you know. Do a risk assessment of the employees, and look at who, which employees are at risk of sleep problems, and then kind of, implement any interventions which might mitigate those problems.” (Pre-COVID-19, SI03A)

“So, I would enforce it to start with, so that people also get to understand that they shouldn't be afraid of something like this, that it's actually in their best interest and also support them in whatever are the outcomes you know. If it is that they need to get booked off for a week because they are depleted whatever it is, and that it should be understood that it's part of remote working, it's part of working in a pandemic, and yeah, I think I would just do something, that's what I'm saying.” (During COVID, SI03B)

In contrast to the responses that online screening and virtual sessions would facilitate the sleep health risk screening process, some respondents felt that employees would benefit more from a sleep health risk screening process if it included a face-to-face consultation with a healthcare provider. Collectively, the general feeling was that sleep health risk screening be conducted in conjunction with a feedback session. Irrespective of when interviews took place, participants felt that a workplace sleep health programme would require an individualized approach to meet employees' needs based on their health screening.

3. Raising sleep health awareness and education

At both interviews, before and during COVID-19, respondents felt that raising sleep health awareness within the organisation, starting with the managers, was critical. At the managerial level, there was indeed acknowledgment that corporate executives understood the importance of adequate sleep for general well-being, but how it could improve their work performance as business leaders was unclear to them. Furthermore, this could be extended into education, as participants explained that corporate executives needed education around strategies that would enable them to identify when their sleep is problematic so that they would know when to seek support and how to restore their sleep health. During the follow-up interviews, participants also spoke of educating employees on ways to manage sleep patterns and create structure to their days.

“So, first of all we don't actually know how big the impact is, so I'm not actually clear unless someone admits to me that they can't sleep, or they don't, sleep, or they're tired, and other than you know observation ... so it's around an awareness program making people understand what it is, and then making people comfortable enough to say well yes

maybe this does affect them, and then interventions to help them with that balance, so I mean that will actually be multi-pronged, it will be on education, and awareness..“
(During COVID-19, SI06B)

“I think there's always a need for sleep education and I think especially now when people's routines are, so out of sync. They would definitely benefit from some education around sleep, and the importance of it.” (During COVID-19, SI03B)

6.4 Discussion

This study explored the barriers to and facilitators of participation in a sleep health programme and key components required for designing such a programme, both before and during the COVID-19 pandemic. At both timepoints, the overarching themes emerging with respect to barriers and facilitators included sleep health awareness, work culture, work-life balance and confidentiality. Specifically, the lack of sleep health awareness among employees was regarded a barrier to participation. Moreover, the demanding work culture, lack of work-life balance and insecurities regarding confidentiality were perceived barriers that would discourage employees from participating in a sleep health programme provided by their workplace.

When asked to conceptualise an ideal sleep health programme, the following themes emerged: identifying the need for a sleep health programme; incorporating sleep health screening into HRAs and promoting sleep health awareness through education. There were no striking differences in the perspective of participants before and during COVID-19 as at both timepoints they felt that sleep health was not prioritised and consequently no sleep support systems were available for employees in need thereof.

A barrier for participating in a proposed sleep health programme both before and during COVID-19, was the work pressure experienced in what was perceived to be a demanding workplace culture. The organisation's 'corporate culture', characterised by high work demands and time constraints, was considered as a factor that would hinder uptake and participation in a sleep health programme. Since our participants provided input from a corporate setting of a predominantly male demographic, the given corporate culture is in line with similar groups described in the literature. For example, there is evidence that in male-dominated industries, this demanding work culture perpetuates a masculinized culture by promoting conformity to longer work hours. As such, the time constraints found to hinder participation in our study suggests that, similar to previous studies, working overtime is normalized and an obligation to work additional hours "for the team", often comes at the expense of healthy sleep habits.²⁷⁷ Another study exploring barriers to participating in WHPs found that time constraints which would limit participation were evident among employees who showed a preference to keep work and private life separate. A possible solution proposed in the literature was to enable participation in the educational aspects of a sleep health program during work hours, thereupon keeping the private life separate from work-related activities.²⁷⁸

The lack of work-life balance was another barrier that could discourage participation in sleep health programmes. With new work structures adopted during COVID-19, additional strain was placed on work-life balance. This new way of working included working from home, which was mandatory during national lockdown, and which posed new challenges that impacted on employees' daily routines and priorities. As a result, working overtime left little time for any additional commitments, including participation in sleep health programmes. Another important finding was the ongoing availability and communication between employees due to the use of online and digital technology during COVID-19. The constant connectivity was found to interfere with the boundaries between work and home time, leading to the encroachment of work into family time. This finding is in line with another study showing that during COVID-19, work-related technology-use during home confinement and outside of working hours caused greater work-family conflict.²⁷⁹ Consequently, the new work environment with its altered daily routines and ongoing connectivity after work played vital roles in creating resistance to welcoming a sleep health programme. The greater emphasis on work-life balance during COVID may indicate a sense of shifting priorities to value family over work, especially in times of distress. Nevertheless, as employees have recently returned to the office, or adopted a hybrid model for work, the restored work structure and schedules may provide a revived interest in protecting their sleep, thus increasing the likelihood of participating in a workplace sleep health programme. To further explore this, additional studies would provide clearer insight.

The lack of awareness of how sleep is specifically linked to work performance was regarded as a barrier among corporate executives. This was because participants felt that if those at managerial level knew how optimising sleep could improve their ability to perform more efficiently and effectively as business leaders, they would be more likely to participate and invest in sleep health within their organisation. The ability to recognise and acknowledge one's own poor sleep patterns was considered as a start to raising sleep health awareness among corporate executives themselves. In contrast, self-denial of suboptimal sleep health would make it difficult for organisations to introduce a sleep health programme, as employees would not see the need to participate. This denial of having sleep health problems has been described previously to show that societal norms have encouraged the normalization of compromising sleep time for additional work time.²⁷⁷ Moreover, the barrier to participation due to lack of sleep health awareness may be different between corporate employees and employees in non-managerial roles. Managers may have easier access to knowledge on the importance of sleep on health outcomes, compared to employees without similar educational backgrounds or accessibility to health education. Nonetheless, it appears that managerial occupations are more likely to associate short sleep duration and irregular sleep patterns with work commitment and dedication. This too was reflected in a recent study exploring the perspectives of male employees on sleep, where working more and sleeping less was considered as a sign of macho-ism and stoicism for achieving greater work productivity.²⁷⁷ Despite the existing knowledge on general sleep health

that managers may have, our findings indicated that organisations fail to dedicate efforts toward sleep health programmes due to the lack of knowledge and experience of developing and implementing such projects.

The confidentiality barrier implicated among the present corporate executive participants was related to being fearful that they would feel exposed. Specifically, by choosing to participate in a sleep health programme, they may be considered to be less effective managers compared to their colleagues, thus risking their job. In a study of office employees, Klasen et al. (2021) found that 'privacy' was a perceived barrier to participating in a preventative strategy targeted at reducing long term sickness absence.²⁸⁰ The psychological stigma attached to sickness absence was implicated as the reason for non-participation. This too may provide a plausible explanation within the present group as it is well-established that a bi-directional relationship exists between psychological well-being and sleep.²⁸¹ The corporate executives for whom sleep may be problematic, could feel marginalised, as having poor sleep may lead to the perception that they also require mental health support. Given this, overcoming the confidentiality barrier is an important step in improving participation. Providing employees with clear information on data security and privacy policies may help to ease their fear of participating.

Our findings showed that to develop an ideal sleep health programme, organisations are required to identify why it would be needed. This is in line with a statement made by Dawes et al. (2001) emphasising that to improve workplace health, companies should realise what is needed in order to create a baseline from which new workplace health schemes can be adopted.²⁸² The National Institute of Clinical Excellence (NICE) provides guidance on steps to conduct a WHP programme needs assessment and emphasises the involvement of all stakeholders in identifying health priorities within the organisation, and planning the health action necessary.²⁸³ Moreover, it is vital to conduct a needs assessment prior to programme implementation as it would increase employee participation rates and avoid barriers that otherwise would be missed.²⁸⁴ Accordingly, following the needs assessment, organisations are guided to develop appropriate health risk assessments. Given this, it should be noted that for interviews with occupational medicine specialists, particularly before COVID-19, their perspectives on developing an ideal sleep health programme were in the context of a WHP needs assessment at organisational level. Yet, when it was discussed by corporate executive participants, reference was made to assess the needs for a sleep health programme based on the sleep health of employees at an individual level. Specifically, it was emphasised that currently those with sleep problems had no support or workplace sleep health management plan thus justifying the need thereof.

WHP programmes typically incorporate HRAs and biometric screening to stratify health risk based on the number of health risk factors present.²⁸⁵ As such, HRAs are tools that can be used for risk identification, risk assessment, and risk reduction.²⁸⁴ Sleep health risk screening could therefore be the foundation upon which the need for a sleep health programme would be identified in the workplace setting. Subsequently organisations can identify and tailor strategies to support employees requiring sleep health management.

Taken together, the implementation of such programmes begins with being aware of the importance of sleep health and identifying the need thereof which may be facilitated by sleep health risk assessments.

Methodological considerations

Having two successive interviews, one prior to COVID-19 and one during the pandemic may be considered a strength of this study. This allowed for possible changes in the perspectives on sleep related WHP programmes resulting from the COVID19 lockdown and altered work environment. Further, the interviews were done in real time, in other words, in-person during initial interviews or online, using the camera for face-to-face communication during follow-up interviews. This made it possible to collect richer, more comprehensive information and context to responses, compared to gathering information through surveys or questionnaires. In addition, despite the COVID-19 restrictions that occurred during the study, all follow-up interviews were conducted online which was advantageous as it allowed for easier accessibility and flexibility of the interviews and allowed participants to be interviewed in their own chosen space. In favour of this study was the focus on employees at management level and within the occupational health space. This provides a homogenous sample of corporate executives and occupational medicine specialists who came from a position of similar leadership responsibility, and that may have the decision-power to facilitate or support a sleep health management component to their existing WHP programmes. Given the lack of previous literature highlighting the insight of organisational leaders in this regard, the results of the present study may guide future situations so that a better management of the sleep health of corporate employees can be disseminated.

It is, however, important to acknowledge the small sample size within this study. The recruitment process was interrupted by the restrictions imposed during the COVID-19 pandemic. As such, we could only work with the sample size that we had when the pandemic started. Despite this, we were able to approach the same participants for a follow-up interview to which they all agreed, and by the end of the coding process, no new themes emerged. In fact, the need to adapt this study in the midst of the pandemic provided a unique opportunity to explore any potential changes in insights around barriers, facilitators and key components of providing a sleep health programme before and during a pandemic. Also, despite collecting data from a convenience sample, participants were identified because of their role and influence within the workplace wellness division of their organisations and as such would play a pivotal role in the future of sleep health within their business. Nonetheless, the current study is explorative and provides preliminary insight from the WHP decision-makers. Given this, our findings may provide the foundation from which future workplace sleep health programmes can develop taken from those who have the impetus to drive such projects. Lastly, the results describe the perspectives of corporate executives and occupational medicine specialists solely in South Africa. Although some findings might only apply within a South African context, themes pertaining to

the normalisation of a demanding workplace culture and work-life balance within the corporate setting, are relevant to similar settings worldwide.

Implications/recommendations

Valuable insights around provision of a sleep health component in WHP programmes have emerged from this qualitative study. The insights on the barriers and facilitators specifically have implications for practice and policy. Our findings showed the need to expand sleep health awareness of corporate executives by educating them on the importance of sleep, particularly with respect to their work performance. By achieving this, sleep health promotion in its entirety can be further expanded to the remaining workforce. As such, effective communication to underscore the vital impact of good sleep health in relation to optimising physical health, mental and work performance is recommended. Ensuring that business leaders and decision-makers within organisations understand the impact of sleep on health and work performance may lead to greater commitment in investing in sleep health management. For example, such communication to the decision-makers within organisations can emanate by presenting evidence to indicate that corporate environments which undermine sleep are harmful to the financial well-being of organisations. Further, educating the key players of WHP programme development on the importance of sleep for health and cognitive functioning is necessary by emphasising how poor sleep can have adverse short- and long-term effects.^{139,286} Therefore, the first step in contributing to better sleep outcomes for employees is for employers to recognise the importance of sleep and the adverse outcomes both for individuals and businesses stemming from insufficient sleep.

It is recommended that business leaders committed to supporting sleep health provide tailored worksite policies to maximize the potential for optimising and maintaining sleep health. In this way the business case for sleep health promotion can be emphasised within the corporate work environment and begin to shift the perception that lack of sleep equates to work dedication. Given this, a workplace culture that values sleep can be accomplished by implementing WHP programmes focussed on optimising employee sleep health, educating employees about sleep health, and raising awareness thereof. The evident workplace culture observed in the present study suggests that those in managerial roles may still feel that substituting sleep time for work translates to being a better leader. To shift this demanding culture towards valuing and supporting sleep health, it has been proposed that employers encourage sleep health through various practices including the expansion of employee WHP programme budgets to incorporate sleep health.²⁷⁷ Moreover, it is paramount for business leaders to be aware of the sleep needs of employees. For this, the business case for sleep health needs to be clear, and based on our findings, may be realised through the education of the decision makers within organisations.

WHP programmes which include HRAs and fatigue risk assessments should provide a more comprehensive assessment dedicated to sleep health where sleep duration, quality and sleep patterns are assessed. With

sleep data collected during a sleep health screening, risk criteria can be calculated as per evidence-based sleep health guidelines. Having company sleep health data allows for the identification of employees who need further sleep health management, provides sleep health metrics to report on the sleep health status of the workforce, and allows for sleep health trends and management to be assessed over time. Currently, where sleep is concerned, occupational health practices cover workplace fatigue and typically focus on industries characterised by shift work, such as the railway industry, flight crews, medical professionals, and truck drivers.⁸⁰ However, it is vital to recognise that daytime fatigue and sleep concerns in non-shift work settings may stem from different factors, and thus require a different strategic approach when compared to shift workers. Additionally, we propose that the occupational health sector be more active to promote sleep health especially since it has been acknowledged in a systematic review of the literature that the lack of knowledge related to sleep health represents a gap in the medical knowledge of occupational health doctors and nurses.²⁸⁷ Therefore, the comprehensive training and education of occupational health staff can enhance the sleep health risk screening process and sleep health management guidance accompanying it.

A sleep health programme should acknowledge the importance of a healthy work-life balance. Contrasting the compulsory home confinement which occurred during the pandemic, work flexibility which includes hybrid working and being given the choice from where to work, may allow some employees to manage their day more effectively. This arrangement may help employees balance their work and personal lives better, resulting in reduced stress levels, increased productivity and improved well-being.²⁸⁸ In the case of a pandemic, employers and supporting occupational healthcare professionals must be sensitive to individual employee needs or desires to alter work patterns to support work-life balance by considering family responsibilities and employee job demands. In this way, the barrier related to work-life imbalance may be overcome, encouraging employees to participate in a workplace sleep health programme.

Finally, one needs to consider that this study addresses the corporate work setting. As such, the involvement of corporate executives in the participation of a sleep health programme is highly encouraged and would demonstrate leadership support, which previously has been recognized as an important construct influencing employee participation in WHP programmes.²⁸⁹ Having corporate executives participate in the sleep health programme would send out a message that management understands the importance of sleep health and is prepared to devote considerable time and resources to identify and address sleep health issues.

6.5 Conclusion

The perspectives from organisational leaders in the decision-making and planning process of WHP programmes provided valuable insight into barriers, facilitators and key components relating to workplace sleep health programmes, before and during the COVID-19 pandemic. For corporate employees to participate in a sleep health programme, organisations should re-evaluate their demanding workplace culture to embrace the importance of sleep health, which in some instances, may require a cultural change

in organisational thinking. The significance of sleep health awareness is unrefuted and needs to incorporate the importance of sleep health for well-being and work performance. Equally important is the enhancement of HRAs to include sleep-specific sections which would identify those who need further sleep health management and provide baseline metrics for future programme evaluation. As such, we recommend that stakeholders, such as employers, business leaders and occupational health professionals who are involved in the development and implementation of WHP programmes, use this knowledge for the integration, development and implementation of sleep health into future WHP programmes.

Chapter 7

General Discussion

The main objectives of this thesis were to characterise the sleep characteristics of corporate executives, to describe the manner and extent to which these sleep characteristics were associated with CMD risk factors, what mediates these associations, and to explore the perspectives of organisational leaders who play a key role in workplace health promotion on developing an ideal sleep health programme.

The three overarching areas of occupational sleep, namely overall employees' sleep, corporate executives' sleep, and the opportunity for sleep health programmes as illustrated in Figure 7.1 are given context by presenting the main findings in the section below.

7.1 Main findings

7.1.1 Employees' sleep duration: association with ACM and CVDM mortality (Chapter 2)

Given that existing research on the relationship between sleep duration and mortality typically refers to the general population, **Chapter 2** presented findings exclusive to employed adults and answers the first question in Figure 7.1. The meta-analysis of data described the association between self-reported sleep duration and ACM and CVDM in cohorts of employed persons younger than 65 years of age living in urban settings indicating that employees sleeping ≤ 6 h compared to 6-8 h had a significantly increased risk for ACM (16%) and CVDM (26%) respectively. The review also indicated, albeit with high heterogeneity, that for most of the analysed cohorts, a longer sleep duration (≥ 8 h) was associated with an increased risk for ACM. This latter observation should be viewed as exploratory as presently there are insufficient eligible studies to investigate the sources of such wide variation through subgroup analyses. Overall, these findings motivate the call to support employees' sleep health and emphasises the inclusion of sleep as a health risk factor into existing workplace health promotion programmes as a means to mitigate the future risks of ACM and CVDM.

7.1.2 Corporate executives' sleep: associations with cardiometabolic disease risk (Chapters 3-5)

The observation that short sleep duration was associated with both ACM and CVDM provided the rationale to further investigate sleep in a specific subset of the employed population. Attention was drawn to corporate executives who represent a group of employed adults with unique work demands, such as rigorous deadlines, strategic business decisions and limitless accessibility to their workforce, often resulting in longer work hours, which consequently may impede their sleep. The cross-sectional study (**Chapter 3**) confirmed what has been observed in the general population notably that shorter sleep duration was associated with adiposity and higher overall CMD risk in a homogenous group of corporate employees, also highlighting some differences between men (blood pressure) and women (HDL cholesterol). This chapter showed that longer work hours and daily commute time to and from work and elevated stress were associated with shorter sleep

in corporate executives. Only among the men were less physical activity, longer working hours and elevated stress mediators of the sleep duration-cardiometabolic health risk relationship. This knowledge is useful since employees with management roles regularly face long working hours, coupled with high pressures experienced in their job responsibilities. As such, these findings should encourage the development of workplace health promotion programmes that promote a balance between work hours and sleep, combined with stress management to mitigate the development of CMD and its comorbidities in corporate executives. The gathered evidence in Chapter 2 prompted research to seek whether these associations remained over a longer period of time. Indeed, significant longitudinal associations were found between shorter sleep and poorer cardiometabolic health, along with gender-specific differences (**Chapter 4**). For example, the longitudinal association between shorter sleep duration and an increase in BMI was true for the entire group, but the association with larger waist circumference and a higher CMD risk score was only evident in the men. Thus, over time, corporate executives with shorter sleep may be more vulnerable to CMD, even when accounting for covariates such as age, physical activity, alcohol consumption, smoking, work - and commuting hours, depression, stress and anxiety. Considering that both cross-sectional and longitudinal self-reported sleep data yielded similar findings, the next progression was to refine our understanding of the sleep-cardiometabolic health relationship in corporate executives by assessing the sleep of this group using actigraphy, and in so doing, providing objective sleep measurements.

The use of actigraphy in combination with a sleep diary in **Chapter 5** gave insight into habitual sleep patterns and the sleep health profile of corporate executives through the use of multiple sleep characteristics beyond sleep duration. The outcome that the majority (70.5%) were sleeping less than the 7-9 h recommendation, more than half (51.7%) reported poor sleep quality and at least one in four were categorized as catch-up sleepers due to their sleep extension of 1.5 h or more on weekends confirmed the presence of poor sleep in these corporate executives. The fundamental findings of this chapter are that having an overall lower 7-day average sleep efficiency, shorter average TiB, shorter average TST and that specifically shorter weekday TST and being a catch-up sleeper significantly increased their risk for CMD, despite accounting for confounders. Given this, it is vital for corporate executives to manage their sleep behaviour during the work week to mitigate potential long term adverse health effects.

To our knowledge, these findings are the first to utilise objective sleep measures in conjunction with self-reported methods to describe the habitual sleep characteristics and the associations with CMD outcomes within a corporate executive group.

7.1.3 Considerations for sleep health programmes in the workplace (Chapter 6)

Having demonstrated that cross-sectional, longitudinal and objectively measured sleep yielded similar findings, Chapters 3 - 5 emphasises the need for policymakers and employers to take the sleep health of their

workforce into account by making efforts to develop and implement effective sleep health programmes in the workplace. Accordingly, the next step to consider would be to provide sleep health solutions to workplaces to mitigate these concerns. Using a qualitative approach, **Chapter 6** sought to gain a greater level of understanding from corporate executives and occupational medicine specialists who play significant roles in the development of workplace health promotion programmes within their companies. Considering from the outset that this could facilitate the translation from research into practice, Chapter 6 addressed the barriers and facilitators of participation in a sleep health programme that could be offered within the workplace, as well as key components thereof.

The key barriers to participating in a sleep health programme were identified in this chapter and may guide workplaces. Specifically, companies can encourage participation by improving sleep health awareness, addressing the demanding workplace culture to support work-life balance, and ensuring that confidentiality is prioritised. Likewise, valuable key components for consideration in developing a sleep health programme included: identifying the need for such a programme within the workforce, ensuring that sleep health risk screening become part of workplace health promotion programmes, and promoting sleep health by raising awareness thereof. The recommendations described in Chapter 6 of this thesis may guide organisations intending to offer sleep health programmes and serve as motivation for the inclusion of sleep in the development and implementation of existing workplace health promotion programmes.

7.2 A visual summary and brief overview

A visual summary of the main findings of this thesis are illustrated in Figure 7.1. In brief, it illustrates that Chapter 2 dealt with the overall observation of employees' sleep and Chapters 3-5 on the lesser known sleep health of corporate executives. The findings from Chapters 2-5 connected short sleep duration with increased mortality and CMD risk and provided the opportunity to explore avenues for workplace sleep health programmes. Given this, Chapter 6 provided a qualitative component to the thesis by discovering the perspectives of corporate executives and occupational medicine specialists on developing an 'ideal' sleep health programme (Chapter 6).

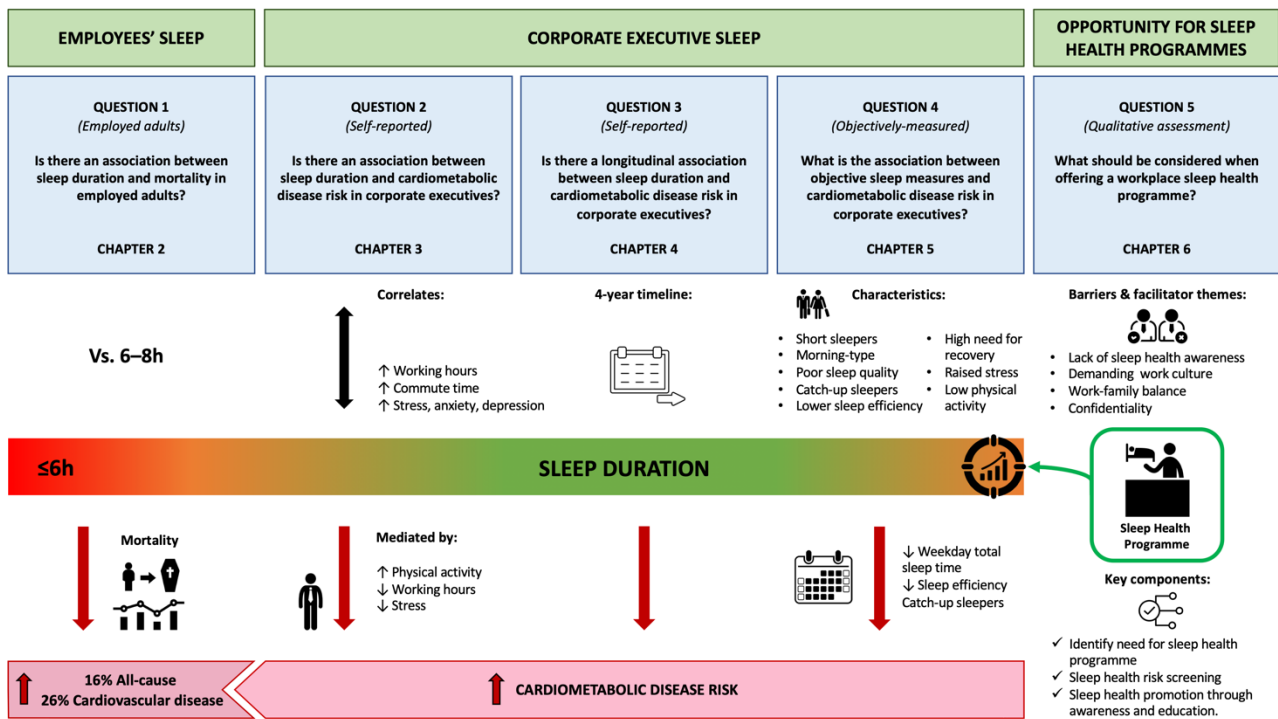


Figure 7.1 Visual summary of PhD thesis.

7.3 Methodological considerations

There are a few methodological considerations that warrant discussion. For example, Chapter 2 was limited by having included a small number of eligible studies, consequently limiting statistical power. A single measurement of exposure may not capture the sustained effects of sleep duration over time and therefore measuring sleep behaviour data over a longer time-period would strengthen the findings. Yet, a systematic review and meta-analysis has clear advantages over literature reviews allowing this thesis to commence with a research modality considered to be at the top of the evidence pyramid.²⁹⁰ Moreover, this chapter is one of the first to provide findings exclusive to the sleep duration and its mortality risk in employed cohorts.

The cross-sectional study design of Chapters 3 and 5, and that it was specific to a unique subset of the working population, namely corporate executives, mean that a causal relationship could not be determined and the findings cannot be generalized to the broader workforce. Despite this, to date there is very little research related to the sleep health of corporate executives, and even less when it relates to their sleep duration relative to CMD risk factors. These chapters therefore contribute to the current paucity in sleep health literature for this echelon of occupations.

In Chapters 3 and 4, self-reported data were used to describe sleep duration and consequently may have suffered from recall biases. The self-report of occupational, psychological and lifestyle factors and the perils of recall biases are also worth noting for Chapters 3 to 5. Another consideration specific to Chapters 3 to 5, was that blood pressure, diabetes and cholesterol treatments were only accounted for when analysing CMD risk, and therefore there is potential for confounding by medications that affect sleep, mental health or that

may interact to affect blood pressure or lipid profiles. Despite this, a major strength of Chapter 3 and 4 was that the analyses were based on a relatively large sample of well-characterized corporate executives. Further, for Chapters 3 to 5, all participants were from similar working backgrounds and participated in a standardised HRA with face-to-face clinical consultations. Together, these chapters provide subjective and objective measures, coupled with a standardized routine data collection procedure, which increases the internal validity of the presented results.

Chapters 3 and 4 were challenged by missing data for sleep quality, presenteeism and daytime fatigue questions. At the start of this thesis, only one question on sleep, namely sleep duration, was assessed, and no daytime fatigue nor presenteeism aspects were incorporated into the HRAs. Nonetheless, the sleep health and presenteeism sections within HRAs were updated shortly afterwards allowing for the compensation of missing data. Additionally, Chapter 3 dealt with the limitation of missing data by using linear mixed model analyses as it is highly suitable for the analysis of longitudinal data with missing values.^{199,200}

The relatively small sample sizes for Chapters 5 and 6 warrant future studies with a larger group of corporate executives and a more evenly distributed number of men and women. This limitation was unexpected for Chapter 6, as it was in part a consequence of the interruption in the recruitment process imposed by the COVID-19 pandemic. Given this challenge, only participants who had been recruited prior to the pandemic were eligible for the follow-up interview as it was a strategy chosen to provide some resolution amidst the pandemic. Nevertheless, all participants agreed and consented to follow-up interviews and by the end of the coding process, no new themes emerged. In fact, the need to adapt this study during the pandemic provided a unique opportunity to explore any potential changes in perspectives around barriers, facilitators and key components of providing a sleep health programme. Surprisingly, the views of these individuals were similar in both instances, with the overall opinion that sleep health was lacking within the workplace health promotion context.

Although the use of wrist-worn actigraphy (Chapter 5) has been indicated as an appropriate measure to objectively record habitual sleep patterns in a non-laboratory setting,²⁹¹ there is a risk that quiet inactivity can be confused with sleep. To mitigate this concern, all actigraphy-derived measures were interpreted alongside a sleep diary to establish the time that participants intended to start and end their sleep period. Having used objective sleep characteristics and incorporated other dimensions of sleep, such as sleep quality and timing, Chapter 5 also made it possible to overcome the limitation of solely using self-reported sleep duration data when investigating relationships with CMD risk. For example, Kanagasabai et al. (2017) whom used a similar CMD risk score as described in this thesis (Section 1.4.1), described the limitation of only utilising self-reported sleep duration and proposed the use of objective measures,¹⁸ which were fulfilled in Chapter 5.

On the one hand, collecting qualitative data from a convenience sample (Chapter 6) may be considered a limitation, but on the other hand, selecting participants with the power to influence workplace health

promotion within organisations provides unique information from corporate executives who play a pivotal role in the future of sleep health within their business. Nonetheless, Chapter 6 is explorative and provides preliminary insights from workplace health promotion decision-makers. Given this, our findings may provide the foundation from which future workplace sleep health programmes can develop, as it is derived from those who have the impetus to drive such projects. Lastly, the results obtained in Chapter 6 describe the perspectives of corporate executives and occupational medicine specialists solely in South Africa. Although some findings might only apply within a South African context, themes pertaining to the normalisation of a demanding workplace culture and work-family balance within the corporate setting, are relevant to comparable settings worldwide.

7.4 Recommendations for occupational health

In summary, this thesis has presented evidence to show that the sleep health of corporate executives is a critically important and underrecognized risk factor for adverse cardiometabolic health and its comorbidities; and that often it is left outside the scope of occupational health research. Nevertheless, the existing paucity provides opportunities for improving sleep health by embedding sleep into workplace health promotion programmes within the corporate work environment. Collectively, this thesis provides quantitative (cross sectional and longitudinal) and qualitative evidence to encourage employers, clinicians and the occupational health policymakers to approach the sleep health of corporate executives with more urgency.

Having found data to suggest that their sleep habits (sleep duration, sleep quality, catch-up sleep) may be predisposing them to an increased risk for CMD and obesity, and that corporate executives and occupational medicine specialists themselves show concern, there are clear indicators prompting for action. Although recommendations have been made to policymakers, these relate to the work-related regulations addressing shift work and mitigating the harmful health implications thereof.^{292,293} Whilst such recommendations are critical, it is apparent that other occupational levels, such as top level management (corporate executives), lack attention. Thus, taking into account the guidance provided by the participants in Chapter 6, the following section provides recommendations for consideration at the organisational and individual levels.

7.4.1 Organisational level

- Re-evaluate current workplace health promotion programmes to ensure that sleep health is included. This includes sleep health risk screening to identify employees who exhibit increased risk based on the recommended sleep health guidelines described in the introduction of this thesis. As such, sleep needs to be regarded as a modifiable health risk factor for adverse health outcomes measured in employee's HRA protocols.
- Ensure that sleep health be assessed beyond sleep duration so that multiple sleep health indicators such as sleep quality, regularity and timing be included.

- Include sleep health in the training and education of healthcare providers who provide occupational health services. Sleep needs to be prioritised and take a forefront in the skills training of such professionals, as this will enhance the screening, identification and referral of employees in the best manner.
- Initiate a change in workplace culture that ‘allows’ corporate executives to meet their sleep need and support work-life balance with a few suggestions such as:
 - Flexibility around work hours.
 - Introducing policies limiting after-hours and out-of-office communications.
 - Sleep health awareness campaigns to emphasise the importance of good quality and sufficient sleep.
- Embed a sleep health management programme into current workplace health promotion programmes with reputable sleep health specialists for those seeking sleep support.

7.4.2 Individual level

- Sleep health awareness targeted at the executive boards of companies (i.e. decision-makers) should be able to demonstrate
 - the link between sleep and work performance and productivity,
 - how sleep supports effective leadership by improving decision-making, problem solving, cognitive function, etc.,
 - the benefits of sleep health to the financial wellbeing of the company.
- Education around the importance of consistency in sleep duration to mitigate catch-up sleep on off days by providing achievable strategies through the help of qualified health professionals with knowledge of sleep.
- Coaching senior management about their own sleep health before rolling out a broader programme, as it can generate enthusiasm and support, and create a cascade effect throughout the organisation.

To add to this, given the clear mediating effect of elevated stress and low levels of physical activity on the sleep-CMD relationship, a multi-disciplinary approach encompassing psychology and physical activity is recommended when addressing sleep health.

7.4.3 Future research

The findings of this thesis sprout new opportunities and spur on further questions to highlight some priorities for future sleep health research within the corporate sector. Listed below are questions to consider in guiding the research of this nature:

- ***Are gender-specific differences in the sleep habits and associations with cardiometabolic disease risk maintained with a larger cohort in which the distribution of corporate men and women is equal?***

Having shown preliminary evidence that there are potential differences in the way sleep and cardiometabolic health relate between men and women, it is important to see whether this finding is replicated with a larger sample size of women. Additionally, combining subjective and objective measures of sleep would neatly characterise any differences in sleep profiles between men and women. In this way, there is potential to consider a more gender-specific tailored approach to sleep health programming for men and women.

- ***Does the sleep health of corporate executives affect their companies' financial bottom line?***

Those at executive level in companies may be more willing to invest in sleep health if there are data to indicate the direct and indirect financial implications of poor sleep in their workforce. For example, it has been suggested that the impact of insufficient sleep is not only felt at the individual level, where short sleepers are at higher risk for certain health conditions such as CMD, but also at the organisational level, as employees with poor sleep health are more likely to be off work (absenteeism) or at work but performing at lower efficiency (presenteeism), with costly consequences for employers.¹³⁷ As such, it would also be worth investigating the link between poor sleep health and workplace productivity as it impacts on economic outcomes.

- ***Does the association between actigraphy-derived sleep characteristics beyond duration (e.g., regularity, timing and quality) and cardiometabolic disease risk persist in larger corporate executive cohort studies?***

There is scope to study more than just sleep duration in the workplace health literature and extend the focus beyond shift work. For example, ensuring that aspects of sleep timing and quality is incorporated into data collection processes to enrich the literature in corporate sleep health, as this group of the working population remains understudied.

- ***How would an ideal sleep health programme perform in the corporate work environment?***

Translating research to practise by applying the findings and recommendations from Chapter 6 to develop a sleep health programme designed to be implemented in the corporate work environment would provide the opportunity to conduct a pilot study to evaluate the performance of such a programme. This subsequently could build sleep health awareness among stakeholders involved in workplace health promotion.

7.5 Conclusion

Overall, every chapter of this thesis provides answers to the research questions presented in Figure 1.3 which too is depicted in Figure 7.1. The thesis began without knowing whether the established associations

between short (<7 h) and/or long (>9) sleep duration and adverse health outcomes in the general population are comparable in employed adults (Chapter 2), nor whether a relationship between sleep duration and CMD is evident in corporate executives. From learning that occupational and psychological factors may limit their sleep duration and mediate the sleep–CMD risk relationship (Chapters 3 and 5), observing that associations between sleep and cardiometabolic health outcomes may differ between men and women (Chapters 3, 4 and 5), probing more extensively into their sleep profiles to find evidence of suboptimal variations in sleep duration (Chapter 5), to taking into account valued insight on avenues to consider in creating a workplace sleep health programme (Chapter 6), this thesis gives new light to sleep health in the workplace as it relates to the corporate executive employee.

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Appendices

Appendix 1

Supplementary tables for Chapter 2

Table S2.1 Search strategies by database

(A) PUBMED	
1	Sleep [MeSH] OR Sleep [tiab] OR insomnia [MeSH] OR insomnia [tiab]
2	((Mortality [MeSH] OR mortality [tiab] OR death [tiab])) OR (("Cardiovascular Diseases/mortality" [Mesh] OR cardiac [tiab] OR cardiovascular [tiab] OR "coronary heart disease" [tiab] OR "coronary artery disease" OR "ischemic heart disease" OR "ischaemic heart disease" OR stroke [tiab] AND (mortality [tiab] OR death [tiab]))
3	"cohort studies" [mesh:noexp] OR "longitudinal studies" [mesh:noexp] OR "follow-up studies" [mesh:noexp]
4	1990:2020 [dp]
5	Eng [la]
6	Humans [Mesh]
7	#1 AND #2 AND #3 AND #4 AND #5 AND #6
(B) SCOPUS	
Filters:	Year: 2020 Document type: Article Source type: Journal Language: English
1	sleep OR insomnia
2	((mortality OR death)) OR ((cardiac OR cardiovascular OR "coronary heart disease" OR "coronary artery disease" OR "ischemic heart disease" OR "ischaemic heart disease" OR stroke AND (mortality OR death))
3	"cohort stud*" OR "longitudinal stud*" OR "follow-up stud*"
4	#1 AND #2 AND #3
(C) ISI WEB OF SCIENCE	
Filters	Year: 2020 Document type: Article Keyword: Humans Language: English
1	sleep OR insomnia
2	mortality OR death
3	cardiac OR cardiovascular OR "coronary heart disease" OR "coronary artery disease" OR "ischemic heart disease" OR "ischaemic heart disease" OR stroke AND (mortality OR death)
4	#2 OR #3
5	cohort stud*" OR "longitudinal stud*" OR "follow-up stud*"
6	1 AND #4 AND #5

Table S2.2 Included studies and their sleep original duration categories, as well as the sleep duration categories used for the present meta-analysis.

Author, year	ACM	CVDM	Original sleep duration (h) categories for which mortality statistics were provided				Sleep duration categories used for the present meta-analysis			
							Shorter sleep (h)	Reference (h)	Longer sleep (h)	
Ferrie, 2007	✓	✓	≤5	6	7*	8	≥9	≤6	7	≥8
Garde, 2013	✓	✓	<6	6-7*		≥8		<6	6-7	≥8
Heslop, 2002	✓	✓		<7	7-8*	>8		<7	7-8	>8
Patel, 2004	✓		≤5	6	7*	8	≥9	≤6	7	≥8
Rhee, 2012	✓		≤5	6-7		≥8*		<6	6 - 7	≥8

ACM: all-cause mortality; CVDM: cardiovascular disease mortality, * indicates original reference category

Supplementary tables for Chapter 3

Table S3.1 Severity categories and scores for the Depression, Anxiety and Stress Scale (DASS) – 21 questionnaire.

Severity	Depression	Anxiety	Stress
Normal	0-9	0-7	0-14
Mild	10-13	8-9	15-18
Moderate	14-20	10-14	19-25
Severe+	21-27	15-19	26-33
Extremely severe+	≥28	≥20	≥34

+Extremely severe and Severe categories were combined in this study

Table S3.2 Mediation analyses of the association between sleep duration and body mass index, waist circumference, cardiometabolic risk score and HDL- cholesterol using the mediators working hours, stress and physical activity in women.

Independent variables		Mediators					
		Working hours ^a		Stress ^a		Physical activity ^b	
BMI (kg/m ²)	Effect	β (95%CI)	P value	β (95%CI)	P value	β (95%CI)	P value
			N=930		N=2227		N=2261
	Indirect	0.003 (-0.04; 0.05)	0.865	-0.04 (-0.12; 0.02)	0.245	-0.05 (-0.12; -0.007)	0.064
	Direct	-0.46 (-0.90; -0.03)	0.042	-0.44 (-0.86; -0.01)	0.046	-0.43 (-0.88; 0.04)	0.064
	Total	-0.45 (-0.90; -0.03)	0.044	-0.47 (-0.90; -0.05)	0.029	-0.48 (-0.94; -0.03)	0.036
Waist circumference (cm)		N=929		N=913		N=930	
	Indirect	0.02 (-0.08; 0.12)	0.743	-0.12 (0.08; -0.31)	0.130	-0.14 (-0.31; 0.02)	0.053
	Direct	-1.14; (-2.20; -0.14)	0.031	-0.97 (-1.96; 0.02)	0.053	-1.07 (-2.08; -0.05)	0.041
	Total	-1.13 (-2.19; -0.13)	0.033	-1.1 (-2.1; -0.11)	0.027	-1.21 (-2.21; -0.22)	0.019
CMD risk score		N=929		N=913		N=930	
	Indirect	-0.0009 (-0.007; 0.006)	0.797	-0.01 (-0.02; -0.002)	0.049	-0.009 (-0.02; -0.001)	0.061
	Direct	-0.07 (-0.14; -0.004)	0.040	-0.06 (-0.13; 0.009)	0.078	-0.07 (-0.14; -0.003)	0.047
	Total	-0.08; (-0.14; -0.007)	0.036	-0.07 (-0.14; -0.005)	0.036	-0.08 (-0.14; -0.01)	0.023

HDL cholesterol		N=929		N=913		N=930	
Indirect	0.002 (-0.0006; 0.007)	0.193	0.002 (-0.002; 0.006)	0.634	0.005 (0.0007; 0.01)	0.049	
Direct	0.03 (-0.0007; 0.06)	0.051	0.03 (-0.006; 0.05)	0.068	0.03 (-0.001; 0.06)	0.051	
Total	0.03 (0.002; 0.06)	0.034	0.03 (-0.003; 0.06)	0.50	0.04 (0.004; 0.07)	0.026	

^aThe effects were adjusted for age, smoking status, alcohol consumption and physical activity. ^bThe effects were adjusted for age, smoking status and alcohol consumption. BMI: body mass index; CMD: cardiometabolic disease; HDL: high density lipoprotein; β : beta co-efficient; CI: confidence interval

Supplementary table for Chapter 4.

Table S4.1 Cardiometabolic disease risk factors and defining levels¹

Risk factor	Defining level
Waist circumference	
Men	≥102 cm
Women	≥88 cm
HDL-cholesterol	
Men	<1.0 mmol/L
Women	<1.3 mmol/L
Triglycerides	≥1.7 mmol/L or on lipid medications
Blood pressure	≥130/≥85 mmHg or on antihypertensive medication
Fasting blood glucose	≥5.6 mmol/L or on diabetes medication

¹ defined by the Adult Treatment Panel III of the National Cholesterol Education Programme

HDL: high density lipoprotein

Supplementary table for Chapter 5.

Table S5.1 DASS-21 sub-scale severity ratings

Severity	Depression	Anxiety	Stress
Normal	0-9	0-7	0-14
Mild	10-13	8-9	15-18
Moderate	14-20	10-14	19-25
Severe ⁺	21-27	15-19	26-33
Extremely severe ⁺	28+	20+	34+

⁺Extremely Severe and Severe categories are combined in this study.

Table S5.2. Simple linear regressions exploring potential associations between candidate independent variables (age; gender; sleep factors; occupational, psychological, lifestyle covariates) and dependent variables (cardiometabolic disease risk score and body mass index) in corporate executives.

	CMD risk score		BMI	
	β (95% CI)	P value	β (95% CI)	P value
Age (y)	0.13 (0.02; 0.23)	0.017	0.08 (-0.07; 0.24)	0.294
No. men	2.81 (0.79; 4.83)	0.007	0.49 (-2.50; 3.48)	0.742
<u>Sleep factors</u>				
Average bedtime (hh:mm)	0.12 (-1.24; 1.48)	0.860	1.10 (-0.66; 2.86)	0.217
Average wake-up time (hh:mm)	-0.96 (-2.40; 0.48)	0.186	-1.00 (-3.08; 1.08)	0.340
Average TiB (h)	-1.19 (-2.56; 0.17)	0.086	-2.23(-4.04; -0.41)	0.017
Average TST (h)	-1.54; (-2.81; -0.36)	0.019	-2.11 (-3.88; -0.35)	0.020
Average SOL (min)	0.07 (-0.03; 0.16)	0.162	0.06(-0.07; 0.20)	0.362
Average SE (%)	-0.17 (-0.35; 0.01)	0.069	-0.09 (-0.37; 0.18)	0.496
Average WASO (min)	0.05 (-0.02; 1.12)	0.184	-0.01 (-0.11; 0.10)	0.859
Average arousal index (arousals/h)	0.34 (-0.03; 0.70)	0.072	0.09 (-0.45; 0.64)	0.732
Average midpoint of sleep (hh:mm)	-0.38 (-1.98; 1.22)	0.636	0.38 (-1.82; 2.58)	0.732
Average bedtime regularity (SD)	-0.50 (-3.45; 2.45)	0.735	1.15 (-3.09; 5.38)	0.590
Average wake-up time regularity (h)	0.92 (-2.18; 4.03)	0.554	3.05 (-1.36; 7.46)	0.172
Average TiB regularity (h)	0.02 (-2.70; 2.74)	0.989	1.01 (-2.92; 4.94)	0.608
Average TST regularity (h)	0.97 (-1.96; 3.90)	0.509	1.36 (-2.86; 5.59)	0.520
Weekday TST	-1.45 (-2.52; -0.38)	0.009	-2.22 (-3.70; -0.74)	0.004
Weekend TST	-0.37 (-1.26; 0.51)	0.399	-0.10 (-1.36; 1.16)	0.876
PSQI score	-0.09 (-0.45; 0.27)	0.609	0.00 (-0.51; 0.53)	0.957
Poor sleep quality ^a	-0.82 (-2.83; 1.19)	0.418	-0.34 (-3.21; 2.53)	0.814
ESS score	0.35 (-1.01; 1.70)	0.607	1.36 (-0.57; 3.30)	0.162

Excessive daytime sleepiness ^b	-1.61 (-4.17; 0.95)	0.214	0.34 (-3.41; 4.09)	0.856
ISI score	0.00 (-0.98; 0.98)	0.996	0.37 (-1.03; 1.77)	0.600
Clinically significant insomnia symptoms ^c	0.49 (-1.59; 2.56)	0.641	1.07 (-1.87; 4.01)	0.468
HO-MEQ score	0.07 (-0.04; 0.18)	0.193	-0.07 (-0.22; 0.09)	0.416
Evening- or Neither-type ^d	Ref		Ref	
Morning-type ^e	2.01 (0.02; 4.00)	0.047	0.60 (-2.35; 3.58)	0.679
Catch-up sleeper^f	1.90 (-0.26; 4.03)	0.083	3.50 (0.45; 6.53)	0.025
<u>Occupational factors</u>				
Work hours per week				
≤60 h	Ref		Ref	
>60 h	-0.83 (-4.17; 2.51)	0.620	-1.80 (-6.64; 3.05)	0.460
Daily work commute time (min/day)	0.24 (-0.17; 0.65)	0.249	0.20 (-0.35; 0.84)	0.411
Days absent in the past year				
None	Ref		Ref	
1 – 6	0.74 (-1.22; 2.71)	0.453	0.74 (-1.77; 3.25)	0.558
≥7	2.90 (-2.31; 8.11)	0.269	11.38 (5.80; 16.95)	0.00
Presenteeism in the past year				
Never	Ref		Ref	
1 -2 times	1.28 (-1.03; 3.60)	0.271	2.22 (-1.17; 5.60)	0.195
3 -4 times	0.09 (-2.89; 3.06)	0.954	1.92 (-2.43; 6.27)	0.380
≥5 times	-0.12 (-4.67; 4.43)	0.958	0.61 (-5.28; 6.50)	0.836
Reduced work ability^g	-0.88 (-3.12; 1.35)	0.431	-1.80 (-4.93; 1.32)	0.252
High NFR^h	-1.61 (-0.57; 2.03)	0.111	-1.50 (-4.39; 1.35)	0.293
Fatigue interfering with daytime function				
Rarely or never	Ref		Ref	
A few days a month	-0.84 (-3.08; 1.39)	0.451	0.18 (-3.08; 3.43)	0.914

A few days a week	-2.09 (-5.10; 0.92)	0.170	-1.72 (-6.12; 2.66)	0.434
Every or almost every day	-1.15 (-5.48; 3.17)	0.595	3.93 (-1.61; 9.48)	0.161
<u>Psychological factors</u>				
Raised depression score	-2.57 (-5.20; 0.07)	0.056	-0.20 (-4.17; 3.77)	0.919
Raised anxiety score	-1.98 (-4.65; 0.70)	0.144	-3.54 (-7.39; 0.31)	0.071
Raised stress score	-2.49 (-4.80; -0.19)	0.035	-3.81 (-7.15; -0.47)	0.026
<u>Lifestyle factors</u>				
Physical activity (min/week)	-0.00 (-0.01; 0.00)	0.709	-0.00 (-0.01; 0.00)	0.315
Low PA level ⁱ	1.40 (-0.56; 3.37)	0.157	2.70 (-0.04; 5.73)	0.089
Current smoker	-2.74 (-6.90; 1.43)	0.193	-2.70 (-8.81; 3.44)	0.384
Alcohol intake	-0.52 (-2.85; 1.81)	0.655		
None	Ref		Ref	
<14 units/week	-0.40 (-2.78; 2.00)	0.737	-3.62 (-6.83; -0.41)	0.028
≥14 units/week	-1.41 (-5.22; 2.40)	0.462	-4.74 (-9.65; 0.17)	0.058

Data are presented as unadjusted beta-coefficients (β) with 95% confidence intervals (CI). CMD: cardiometabolic disease, BMI: body mass index, Ref: Reference category, TiB: time-in-bed; TST: total sleep time; SOL: sleep onset latency; SE: sleep efficiency; WASO: wake after sleep onset. PSQI: Pittsburgh Sleep Quality Index, ESS: Epworth Sleepiness Scale; ISI: Insomnia Severity Index; HO-MEQ: Horne-Östberg Morningness-Eveningness Personality Questionnaire. ^aPSQI ≥ 6 . ^bESS >10 . ^cISI >14 . ^dEvening type: HO-MEQ score ≤ 41 ; ^dNeither-type: HO-MEQ score 42-58; ^eMorning-type: HO-MEQ score ≥ 59 . ^fCatch-up sleeper refers to catch-up sleep duration >1.5 h. ^gReduced work ability refers to scores >7 . ^hHigh need for recovery refers to scores ≥ 6 . ⁱPA < 150 minutes per week. **P values < 0.150 are shown in bold.**

Supplementary table for Chapter 6:

Table S6.1. Interview guide of semi-structured interviews: (a) before COVID and (b) during COVID.

1.	a. Are you satisfied with how sleep is promoted in the workplace?
	b. Are you satisfied with how sleep has been promoted in the workplace during this COVID-19 pandemic and lockdown?
2.	a. What is your opinion on the role of sleep on work performance?
	b. What is your opinion on the role of sleep on work performance during such a pandemic? Specifically, working from home/having a change in the work environment?
3.	a. In your experience, do you feel there is a need for sleep education or awareness amongst employees?
	b. In your experience, do you feel there is a need for sleep education or awareness amongst employees during circumstances such as we are experiencing during the pandemic?
4.	a. What do you think is interfering with (employee's) having an optimal sleep pattern?
	b. What do you think is interfering with (employee's) having an optimal sleep pattern during the COVID-19 pandemic?
5.	a. Have employees come to you with sleep issues.
	b. Have employees come to you with sleep issues during this time of the pandemic?
6.	a. What are your current strategies to managing employee fatigue?
7.	a. How does your workplace address sleep health?
	b. How has your workplace addressed sleep health during the COVID-19 pandemic?
8.	a. In your opinion, what would you consider to be barriers and facilitators of a sleep health programme in the workplace?
	b. In your opinion, what would you consider to be barriers and facilitators of a sleep health programme in the workplace during COVID-19?
9.	a. What do you think employees would like to know about sleep?
	b. What do you think employees would like to know about sleep during this time?
10.	a. What would you like to get out of a sleep health programme or sleep product? What do you think employees would like to get out of it?
	b. What would you like to get out of a sleep health programme or sleep product? What do you think employees would like to get out of it during this period of the pandemic?
11.	a. What would put you (them) off from participating in sleep health modules?
	b. What would put you (them) off from participating in sleep health modules during such a time?
12.	a. What aspect of your own (employee sleep behaviour) sleep behaviour would you most want to change?
	b. What aspect of your own (employee sleep behaviour) sleep behaviour would you most want to change during this time?
13.	a. Do you want to make a change that would help you get more sleep? Do you think employees would like to make changes that could help them improve their sleep.

14.
 - b. Do you want to make a change that would help you get more sleep, and do you think employees would like to make changes that could help them improve their sleep amid the COVID-19 pandemic?
 - a. How would you describe an ideal sleep health programme – what would work and how would you implement it if you could?
 - b. How would you describe an ideal sleep health programme that could be provided to a workplace during a pandemic (incl. lockdown) – what would work and how would you implement it if you could?