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

Paula R. Pienaar [a,b,*], Laura C. Roden [c,a], Cécile R.L. Boot [b], Willem van Mechelen [a,b,d,e,f], Jos W.R. Twisk [g], Estelle V. Lambert [a], Dale E. Rae [a]

[a] Health Through Physical Activity Lifestyle and Sport Research Centre & Division of Physiological Sciences, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa
[b] Amsterdam UMC, Vrije Universiteit Amsterdam, Department of Public and Occupational Health and Amsterdam Public Health Research Institute, Van der Boechorststraat 7, Amsterdam 1081 BT, the Netherlands
[c] Centre for Health and Life Sciences, Coventry University, Coventry CV1 2DS, United Kingdom
[d] Human Movement and Nutrition Sciences, Faculty of Health and Behavioural Sciences, University of Queensland, Brisbane, Australia
[e] School of Public Health, Physiotherapy and Population Sciences, University College Dublin, Dublin, Ireland
[f] Center of Human Movement Sciences, University Medical Center Groningen, University of Groningen, Groningen, the Netherlands
[g] Department of Epidemiology and Biostatistics, Amsterdam Public Health Research Institute, Amsterdam University Medical Centers, Vrije Universiteit Amsterdam, Amsterdam, the Netherlands

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ABSTRACT

Objective: This study aimed to determine the longitudinal associations between self-reported sleep duration and cardiometabolic disease (CMD) risk in corporate executives.
Methods: Self-reported sleep duration and lifestyle, occupational, psychological, and anthropometrical, blood pressure and blood marker variables were obtained from 1512 employees at annual health risk assessments in South Africa between 2016 and 2019. Gender-stratified linear mixed models, adjusting for age, lifestyle, occupational and psychological covariates were used to explore these longitudinal associations.
Results: Among women, shorter sleep duration was associated with higher body mass index (BMI) covarying for age only (ß 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 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]).
Conclusion: Corporate executives who report shorter sleep durations may 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.

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 (Knutson and Van Cauter, 2008; Tobaldini et al., 2019; Covassin and Singh, 2016). Existing evidence shows that sleep which is both longer (>9 h) and shorter (<7 h) than the 7-9 h recommended duration guidelines (Hirshkowitz et al., 2015) 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

* Corresponding author at: Health Through Physical Activity Lifestyle and Sport Research Centre & Division of Physiological Sciences, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa.
E-mail address: pnrpau001@myuct.ac.za (P.R. Pienaar).

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stress, anxiety and depression (Zhai et al., 2015; Bean and Ciesla, 2021).

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) (Burton et al., 2017). 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 (Hafner et al., 2016). 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 (Basner et al., 2007; Basner et al., 2014; Litwiller et al., 2017). Adequate sleep is critical for workplace performance (Barnes and Watson, 2019) 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 (Ganesh et al., 2018). 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 (Gish and Wagner, 2017).

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 (Mallampalli and Carter, 2014; Silva-Costa et al., 2020). Such evidence in corporate employees have been reported by a recent cross-sectional study investigating the association between sleep and CMD risk in corporate executives, which provides early evidence that there may indeed be gender differences in this group (Pienaar et al., 2021). 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 (Burton et al., 2017), 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 (Chen et al., 2018).

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 (Itani et al., 2017; Johannessen and Sterud, 2017). 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 employed population. 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.

2. Methods

2.1. Study design and population

This study is a secondary, longitudinal analysis of HRA data collected annually from 56 companies in South Africa between January 2016 to December 2019 ($n = 1840$). The companies were from the information technology, finance, telecommunication services, health, construction and engineering, consulting, manufacturing and production, retail and wholesale trade, mining, transportation and hospitality sectors. Employees completed a web-based HRA prior to a comprehensive clinical consultation and were full-time employees in senior or executive management positions. 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. Thus, of the 1840 employees who gave informed consent and for whom we had longitudinal data, the response rate was 82% (1160 men, 352 women). 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).

2.2. Measures

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

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 min.

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 (Lovibond and Lovibond, 1995a). 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 Cronbach's alpha for each scale for the DASS normative sample are depression: 0.91; anxiety: 0.84; and stress: 0.90 (Lovibond and Lovibond, 1995b). Outcome variables were represented as continuous variables for depression, anxiety and stress scores, where higher scores denoted greater severity.

Lifestyle factors: Participants reported the number and duration of physical activity sessions in a typical week, from which a weekly average

duration was calculated and 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) <14 alcoholic drinks per week, (3) 14–21 alcoholic drinks per week, and (4) >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); systolic (SBP) and diastolic blood pressure (DBP, 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, with each analysed as continuous variables and dichotomized according to the defined criteria by the Adult Treatment Panel III of the National Cholesterol Education Program (ATP-III) (Detection NCEPEPo, Adults ToHBCi, 2002) (Supplementary Table S1).

A continuous CMD risk score was calculated by summing the standardized z-scores for key variable as follows: -zHDL + zGlu + zTG + [(zBMI + zWC)/2] + [(zSBP + zDBP)/2]. Since HDL and WC 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 (Kanagasabai and Chaput, 2017; Chaput et al., 2013). A higher score indicates a less favourable CMD profile.

2.3. 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 (Twisk and de Vente, 2019). 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 (Twisk, 2013; Twisk et al., 2013). Given that findings from our cross-sectional study in the same cohort showed significantly different health risk profiles between men and women (Pienaar et al., 2021), all longitudinal analyses were performed separately for men and women. Data were analysed using Stata (v.15, StataCorp, Texas, USA) and significance accepted using an alpha value of $P < 0.05$.

3. Results

3.1. 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 1. The CMD risk factors of the cohort illustrated in Table 2 indicate that 25% had high WC 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 1 and 2 represent all data collected for the cohort's first HRA.

Table 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 (%).

3.2. Longitudinal analyses

Table 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 (ß = -0.19, 95%CI: −0.36, −0.03, $P = 0.019$); age and occupational factors (ß = -0.20, 95%CI: −0.36, −0.03, $P = 0.012$); and age and psychological factors (ß = -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

Table 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/m^2)	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.

four-year period (ß = 0.03, 95%CI: 0.00, 0.06, $P = 0.045$).

Among the men (Table 4), a shorter self-reported sleep duration was associated with a higher BMI when adjusting for age (β = -0.15, 95%CI: −0.22, −0.08, $P < 0.001$); age and lifestyle factors (β = -0.12, 95%CI: −0.21, −0.04, $P = 0.004$); age and occupational factors (β = -0.20, 95% CI: −0.22, −0.08, P < 0.001); and age and psychological factors (β = -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 (ß = -0.12, 95%CI: −0.20, −0.04, $P = 0.002$) and age and psychological factors (ß = -0.08, 95%CI: −0.15, −0.01, $P = 0.030$).

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 workers (Buchvold et al., 2018) or children and adolescents (Sun et al., 2020). 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 WC 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

Table 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		Model 2		Model 3		Model 4	
	$n = 361$	P value	$n = 298$	P value	n = 361	P value	$n = 356$	P value
BMI (kg/m^2)	−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 (ß) 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
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		Model 2		Model 3		Model 4	
	$n = 1168$	P value	$n = 1014$	P value	$n = 1169$	P value	$n = 1156$	P value
BMI (kg/m^2)	−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; −010)	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 (mmol/L)	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 (ß) 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 risk.

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 (Bacaro et al., 2020). For example, a study demonstrated that in the general population, on average, BMI was 0.35 kg/m^2 lower for every additional hour of sleep reported, and in a 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 (Knutson and Van Cauter, 2008; Pienaar et al., 2021).

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 such that more stress, less physical activity and poor dietary choices may contribute to shorter sleep and 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 (Zhou et al., 2019). 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 (Kaneita et al., 2008). 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 (Kanagasabai and Chaput, 2017; Chaput et al., 2013). 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 (Buxton et al., 2018). 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 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 >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 (Virtanen et al., 2009). Moreover, results from a meta-analysis on long work hours and occupational health showed the strongest association with short sleep duration (<6 h/day) (Wong et al., 2019). 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 (Pienaar et al., 2021). 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 (Ikesu et al., 2021). 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 (Soprovich et al., 2021; Seaton et al., 2019). Additionally, there is also evidence to suggest that many workplaces have adapted to working overtime (Bunjo et al., 2021), 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 towards 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 inspire comparable sleep behaviour change 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 accelerometery, and using questionnaires that are less restricted to categories and ranges. Sedentary behaviour and dietary intake measures could 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 (Liu et al., 2021). Nevertheless, the data obtained for this study were from standardized 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 an exclusive 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, only accounted for blood pressure, diabetes and cholesterol combined with treatment to analyse CMD risk, and therefore 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 major 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.

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.

Author contributions

PRP and DER contributed to the study conception and design; PRP collected the data; statistical analyses were performed by JST; PRP wrote the first draft; all authors reviewed, read and agreed to the final version of the manuscript.

Ethics statement, registration number and informed consent

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.

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This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. There are no potential competing interests to declare.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The data that has been used is confidential.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ypmed.2023.107724.

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