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Original citation & hyperlink:
Pienaar, PR, Roden, L, Boot, CRL, van Mechelen, W, Twisk, J, Lambert, EV & Rae, DE 2021, 'Association between self-reported sleep duration and cardiometabolic risk in corporate executives', International Archives of Occupational and Environmental Health, vol. 94, pp. 1809-1821. https://dx.doi.org/ 10.1007/s00420-021-01739-2

DOI 10.1007/s00420-021-01739-2
ISSN 0340-0131
ESSN 1432-1246

Publisher: Springer

The final publication is available at Springer via http://dx.doi.org/10.1007/s00420-021-01739-2

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

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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% (Roth et al. 2018). 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 (Ford and Kamerow 1989), sleep duration has emerged recently as a factor contributing to CMD.

More specifically, habitual short sleep duration (<7h) has been associated with increased risk for obesity, T2DM, hypertension, CVD, the metabolic syndrome and early mortality (Covassin and Singh 2016; Fernandez-Mendoza et al. 2017; Gallicchio and Kalesan 2009; Sullivan and Ordiah 2018; Tobaldini et al. 2019). While current recommendations are 7–9h sleep per night for optimal physical and mental health (Hirshkowitz et al. 2015; Watson et al. 2015), recent findings suggest that time allotted to sleep may have gradually declined over the past decades in the general population (Matricciani et al. 2017).

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 (Basner et al. 2007). 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 (Basner et al. 2007; Control and Prevention 2012; Kuhn and Lozano 2005). 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 (Åkerstedt et al. 2019). 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 (Budnick and Barber 2015; Lim and Dinges 2010; Miller et al. 2014; Wild et al. 2018).

Self-reported short sleep duration (≤6h 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 (Luckhaupt et al. 2010). 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 (Ganesh et al. 2018; Nishitani et al. 2013).

Both working hours and stress contribute to shorter sleep (Control and Prevention 2012; Kuhn and Lozano 2005; Nakashima et al. 2011), 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 (Wang et al. 2014), diabetes (Gan et al. 2014) and obesity (Sweeney et al. 2018), 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 (Metlaine et al. 2017); while in another study, shorter sleeping time was associated with depressive symptoms and anxiety (Nishitani et al. 2018).

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 (Choi et al. 2018), long working hours (Reynolds et al. 2018) and physical inactivity (Vincent et al. 2017) 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 sex differences with respect to cardiometabolic risk and sleep (Meers et al. 2019; Silva-Costa et al. 2020), particularly in occupational health studies, we attempted to address this gap and stratified our analyses by sex. 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.

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 sex (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 (sex, 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 <5h to ≥10h 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) <40h/week, (2) 40-60h/week, and (3) >60h/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 (Lovibond and Lovibond 1995b). 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 (Dreyer et al. 2019; Henry and Crawford 2005), 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 (Lovibond and Lovibond 1995a). 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 1. These cut-off scores were derived from a set of severity ratings, proposed by Lovibond and Lovibond (Lovibond and Lovibond 1996).

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 ≥150min/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 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 0.1kg. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared (kg/m^2). Waist circumference (WC) was measured around the midpoint of the lowest rib and iliac crest, to the nearest 0.1cm. 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 10h 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 risk score

A continuous cardiometabolic 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 cardiometabolic risk score was calculated for each participant as follows:

Cardiometabolic risk score = -zHDL + zGlu + zTG + [(zBMI + zWC)/2] + [(zSBP + zDBP)/2]

We used this score to estimate an individual's overall cardiometabolic 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 (Chaput et al. 2013; Kanagasabai and Chaput 2017). A higher score indicates a less favourable metabolic profile.

Data and statistical analyses

Data are presented as mean ± 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 (Baron and Kenny 1986). Figure 2 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 2) 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 (v.15, StataCorp, Texas, USA) and significance accepted using an alpha value of $p<0.05$.

Results

Participant characteristics

Descriptive characteristics of the sample are presented in Table 1. Whereas the sleep duration median was 7h for both men and women, 20.6% of men and 26.5% of women reported a sleep duration <7h. Sleep duration within the 7-9h range was reported in 79.5% of the men and 73.5% of the women. No participants reported a sleep duration >9h.

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 21-60min 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 ($p<0.001$).

Cardiometabolic risk factors of the men and women are presented in Table 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 ($p<0.001$).

Occupational and psychological correlates of self-reported sleep duration

The occupational and psychological correlates of self-reported sleep duration are presented in Table 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 <20min/day; $p=0.012$) had shorter sleep durations. Similarly, in women, working more than 60h/week (vs 40-60h/week, $p=0.001$), and travelling 21-60min/day ($p=0.041$) or >60min/day ($p=0.009$) vs <20min/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.

Associations between self-reported sleep duration and cardiometabolic risk factors

Table 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 cardiometabolic 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 cardiometabolic 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 1.

Mediation analyses

Table 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 S2)

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 cardiometabolic risk scores, while shorter sleeping women were more likely to have higher BMI, WC and cardiometabolic 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 cardiometabolic risk score, compared to their male counterparts (Kanagasabai and Chaput 2017), and when comparing the associations between sleep and individual cardiometabolic 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 (Pradhan 2014). Specifically, sex 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 (Dean et al. 2012). However, the average age of women in studies investigating sleep and hypertension was approximately 55 years (Cappuccio et al. 2007; Di Giosia et al. 2018), 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 <5h (Cappuccio et al. 2007; Di Giosia et al. 2018). In our sample of 1042 women, only 262 reported <6h 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 ≤6h/day was linked to low serum HDL cholesterol in women specifically (Kaneita et al. 2008). While the mechanisms behind these sex-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 (Aho et al. 2016).

Mediators of the association between sleep duration and cardiometabolic 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 cardiometabolic 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 (Nemoto 2013). Similarly, Cho et al. 2015 described a 'double burden' of work and family responsibilities in white collar women employees (Cho et al. 2015). 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 (Kervezee et al. 2020), 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 (≤7h/day) have been reported in employees with specialized work and in office workers, when compared to those in manual labour (Choi et al. 2018); and in another study, the primary predictor of short sleep in obese employees was stress (Vgontzas

et al. 2008). 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 active (Patel and Hu 2008) and increase daytime sleepiness, lower levels of physical activity have also been associated with increased daytime fatigue (McClain et al. 2014), 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 cardiometabolic 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 (12h/day), and short sleep (≤7h/day), were predictors of obesity (Di Milia and Mummery 2009). Moreover, older executives who worked ≥50h/week and slept ≤47h/week in their midlife reported poorer physical health and function (i.e. health-related quality of life) in old age (von Bonsdorff et al. 2017). Since our overall cohort had a mean age of 45 years of which many reported sleeping <7h and working ≥60h/week, our results are important in implementing a pro-active approach to workplace health, mitigating future adverse cardiometabolic 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 cardiometabolic diseases, 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 cardiometabolic 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 cardiometabolic 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 cardiometabolic 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.

Conclusion

These findings provide insight into the relationship between sleep duration and cardiometabolic risk in corporate executives, and help to fill the gap in the literature by describing sex 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 cardiometabolic 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.

Acknowledgements: The authors sincerely thank the healthcare professionals involved in the Life Healthcare Employee Health Services (corporate wellness programme) and the companies who agreed to participate in this study.

Funding: The authors received no funding for this work.

Compliance with ethical standards

Conflict of interest: This was not an industry supported study. However, for the avoidance of doubt we would like to report that WvM is director of Evalua Nederland B.V., advisor of Evalua international Oy. (Ltd). and non-executive board member of Arbo Unie B.V. All those companies are active in the European occupational health care market. And also; PRP is employed by Life Healthcare (SA), Employee Health Services, of which corporate HRA data was used for this study. There are no other potential competing interests to declare.

Ethical approval and consent to participate: 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.

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