Original Research

Multidimensional Sleep and Cardiometabolic Risk Factors for Type 2 Diabetes: Examining Self-Report and Objective Dimensions of Sleep

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Lisa Matricciani, PhD, Catherine Paquet, PhD, Dorothea Dumuid, PhD, Kurt Lushington, PhD, and Tim Olds, PhD

Abstract

Purpose: The purpose of the study was to determine the association between objective and self-report measures of sleep and cardiometabolic risk factors for type 2 diabetes.

Methods: This study examines data on Australian adults, collected as part of the Child Health CheckPoint study. Sleep was examined in terms of actigraphy-derived sleep duration, timing, efficiency and variability; and self-report trouble sleeping. Cardiometabolic risk factors for type 2 diabetes were examined in terms of body mass index and biomarkers of inflammation and dyslipidemia. Generalized estimating equations, adjusted for geographic clustering, were used to determine the association between measures of sleep and cardiometabolic risk factors.

Results: Complete case analysis was conducted for 1017 parents (87% mothers). Both objective and self-report measures of sleep were significantly but weakly associated with cardiometabolic risk factors.

Conclusion: Both objective and self-report measures of sleep are significantly associated with cardiometabolic risk factors for type 2 diabetes. Self-report troubled sleep is associated with poorer cardiometabolic health, independent of actigraphy-derived sleep parameters.

Sleep may be an important modifiable predictor of type 2 diabetes. Short sleep duration has been associated with an increased risk of adiposity, raised inflammatory markers, dyslipidaemia, and glucose intolerance—known predictors of type 2 diabetes.[1,2] More recently, other characteristics of sleep, including delayed sleep timing,[3-5] inconsistent sleep patterns,[6,7] and low sleep efficiency,[8,9] have also been identified as important.

Several mechanisms and pathways have been suggested to help explain the association between sleep and *cardiometabolic health*, a term used to describe anthropometric measures and biomarkers associated with cardiometabolic disease.[10-16] Sleep is thought to play an important role in maintaining metabolic homeostasis.[17] Poor sleep, characterized by short sleep duration, delayed sleep timing, variable sleep patterns, and poor sleep quality, are thought to result in hormone dysregulation (which increases appetite, calorie consumption, and unhealthy food preferences), lower physical activity levels, and adversely affect processes occurring in the hypothalamic-pituitary-adrenal axis, leading to systemic inflammation.[11,12,18] Collectively, these changes result in raised inflammatory markers, dyslipidemia, and obesity, which are all known to increase the risk of developing type 2 diabetes.[16,18-21]

Sleep is increasingly recognized as a multidimensional construct that encompasses a range of sleep characteristics across different domains. Buysse[22] proposed the term *sleep health* and suggested dimensions of sleep include sleep regularity, satisfaction, alertness during waking hours, timing of sleep, sleep efficiency, and sleep duration. Given the multidimensional nature of sleep, medical professionals, population health promotion strategies, interventions, and sleep surveillance programs increasingly recognize the need to consider multiple characteristics of "healthy" sleep.[22,23] While objective device-based measures of sleep (eg, duration, timing, variability, efficiency) and self-reported experiences of

From Clinical & Health Sciences, University of South Australia, Adelaide, Australia (Dr Matricciani); Alliance for Research in Exercise, Nutrition and Activity (ARENA), University of South Australia, Adelaide, Australia (Dr Matricciani, Dr Dumuid, Prof Olds); Allied Health and Human Performance (AHHP), University of South Australia, Adelaide, Australia (Dr Dumuid); Faculté des Sciences Administratives, Université Laval; Centre Nutrition, santé et société (NUTRISS), INAF, Université Laval; Centre de Recherche, Centre Hospitalier Universitaire de Québec - Université Laval (Dr Paquet); Murdoch Children's Research Institute, Parkville, Victoria, Australia (Dr Dumuid, Prof Olds); and Discipline of Psychology, Justice and Society Unit, University of South Australia (Prof Lushington).

Corresponding Author:
Lisa Matricciani, University of South Australia, North terrace, Adelaide, 5001, Australia.
Email: lisa.matricciani@unisa.edu.au

sleep are often recognized as important dimensions of healthy sleep,[22] the self-reported sleep experience is poorly understood, inconsistently defined, and relatively underexamined.[11] For example, the terms sleep *quality*, *sufficiency*, *satisfaction*, *trouble sleeping* and reports of difficulty initiating and maintaining sleep, early morning awakenings, and feeling refreshed upon awakening have all been used to describe the self-report sleep experience.[24-26]

Self-reported sleep experiences are often poorly correlated with objective sleep measures, particularly actigraphy-derived sleep parameters.[27-33] The self-reported sleep experience is thought to reflect a unique characteristic of sleep and/or a nonsleep phenomena, such as personality traits,[34] pain, or anxiety.[35,36] It is also possible that interindividual variability in expectations of what "good sleep" should be or the relative salience of sleep in day-to-day life is reflected in self-report measures of sleep.[37] While further studies are needed to better understand the self-reported sleep experience, it is important to note that this construct is what largely distinguishes individuals with insomnia from individuals without insomnia, whose objective sleep parameters often overlap.[38] The diagnostic criteria of insomnia is based on self-reported sleep and day-time parameters. Hence, the self-reported side of sleep is acknowledged as important and sufficiently so to be a diagnostic criterion.[39,40] The relative importance of objective and self-report measures of sleep for cardiometabolic health has not (to our knowledge) been considered.

Self-report measures of sleep have important clinical implications and often guide further screening and/or treatments.[41] Although not widely used in the literature, single-item questions that determine "troubled sleep" may reflect how individuals first identify and describe a problem with their sleep in "real-world" settings. What remains poorly understood is whether the self-reported sleep experience, like objective aspects of sleep (eg, duration, timing, and variability), is associated with cardiometabolic health.

Available (limited) studies suggest perceived "trouble sleeping" may be associated with the development of type 2 diabetes. Boyko et al,[42] in a study of 47 093 adults enrolled in the 6-year prospective Millennium Cohort Study, found self-reported trouble sleeping was significantly and independently associated with higher odds of developing type 2 diabetes, even after adjusting for sociodemographic covariates. Similarly, Meisinger et al,[43] in a multinational prospective study of 8269 adults, found participants who reported trouble sleeping were at an increased risk of developing type 2 diabetes. The risk of developing type 2 diabetes remained even after adjusting for obesity and hypertension. While it is possible that perceived trouble sleeping may simply reflect an underlying sleep disorder, Liu et al,[44] in a study of 3668 adults, distinguished participants with a sleep disorder from those who reported trouble sleeping. Their study found that participants who reported trouble sleeping (without a sleep disorder) had a similar, albeit not significant, elevated risk of having type 2 diabetes (OR, 1.38; 95% CI, 0.95-2.00) to those who reported being diagnosed with a sleep disorder (OR, 1.36; 95% CI, 1.06-1.73), which persisted after adjusting for sociodemographic characteristics, body mass index (BMI), and sleep duration, respectively.

Given that both objective and self-report measures of sleep are increasingly being recognized as important risk factors for type 2 diabetes, it is of interest to better understand how *both* aspects of sleep are associated with cardiometabolic health. Aims of the study were to (1) examine the correlation between self-reported trouble sleeping and actigraphy-derived measures of sleep (sleep duration, timing, variability, and efficiency) and (2) determine the association between self-reported trouble sleeping, actigraphy-derived measures of sleep, and cardiometabolic risk factors for type 2 diabetes (adiposity, inflammation, dyslipidaemia) in a sample of healthy Australian parents.

Methods

Data were collected as part of the cross-sectional Child Health CheckPoint study between February 2015 and March 2016. CheckPoint is a once-off study nested between Waves 6 and 7 of the Longitudinal Study of Australian Children (LSAC), which involved comprehensive, simultaneous physical health and biomarker assessments of children and one of their parents. LSAC is a population-based study that commenced in 2004 and used a 2-stage clustered design, involving the random sampling and selection of postcodes and participants (respectively) to recruit participants. Two cohorts of children and their families (B and K cohort) were recruited and have since been followed up biennially. During Wave 6 of the LSAC study, families in the B cohort were asked to consent to their contact details being shared with the CheckPoint team, who invited families to take part in the cross-sectional study. Further details of the CheckPoint study design and recruitment are outlined elsewhere.[45,46] Participants in this study were the parents of children in LSAC ages 11 to 12 years at the time of data collection.

Ethics and Consent

The CheckPoint study protocol was approved by the Royal Children's Hospital Melbourne Human Research Ethics Committee (33225D) and Australian Institute of Family Studies Ethics Committee (14-26). The attending parent/caregiver provided written informed consent for themselves and their child to participate in the study.

Sleep

Sleep was assessed in terms of self-report trouble sleeping and actigraphy-measured sleep characteristics.

Troubled sleep. Troubled sleep was assessed via self-report. Participants were asked to report how often they had trouble sleeping over the last month using a 5-point Likert scale (never, almost never, sometimes, often, almost always), which was collapsed for analysis into 3 categories: never (never, almost never), sometimes, and often (often, almost always). These categories were created for ease of interpretability and to ensure a more even spread of participants across the categories.

Actigraphy-derived sleep. GENEActiv monitors (Activinsights, Cambs, UK) were used to collect sleep data. Parents were fitted with a GENEActiv monitor to their nondominant wrist and were asked to wear the monitor continuously for 8 consecutive days. The device was configured to record at 50 Hz, starting at 2300 hour, through the manufacturer's software (GENEActiv PC Software, Activinsights, UK). Raw acceleration data, collapsed as 60-second epochs, were processed using Cobra software to derive 4 objective characteristics of sleep examined in this study:

1. sleep period (the difference between sleep onset and offset),
2. sleep midpoint (the midpoint between sleep onset and offset),
3. day-to-day sleep length variability (the coefficient of variation of sleep period),
4. sleep efficiency (the percentage of minutes scored as sleep between onset and offset).

These variables were selected to represent measures of sleep duration, timing, variability, and efficiency, respectively.

GENEActiv accelerometers have been used in previous studies that examine adults[47] and have been shown to agree with polysomnography measures of sleep duration (83% accuracy).[47] Participants were included for analysis if they had at least 4 nights of sleep data recorded, an average sleep time >200 minutes, and at least 1 weekend night (Friday or Saturday night) of sleep data. Further details of sleep data processing have been reported elsewhere.[48,49]

Covariates

Variables known to influence sleep include age,[50] sex,[50,51] season of data collection,[52,53] and socioeconomic position.[54-56] These measures were selected as covariates in statistical analyses. Sleep was weighted for day-type (weekday/weekend), which is also known to influence sleep.[50]

Socioeconomic position (SEP), a composite measure of self-reported household income, education, and occupation, was derived from the LSAC data set.[57,58] Using this standardized scale, higher scores represent higher SEP.

Age was calculated from date of birth and expressed as years.

Cardiometabolic Health

Cardiometabolic health was considered in terms of biomarkers and anthropometric measures. Adiposity, inflammation, and cholesterol balance were assessed in terms of BMI, glycoprotein acetyls (GlycA), and apolipoprotein B to apolipoprotein A1 ratio (ApoB/A1), respectively. These measures were selected as longitudinal studies have shown these cardiometabolic markers are predictive of future development of type 2 diabetes.[59-63]

Biomarkers. Semifasted venous blood samples were taken from consenting adults in the CheckPoint study. In some cases, participants declined to provide venous samples but provided capillary blood samples instead. Appropriately trained researchers or phlebotomists collected venous blood samples within assessment centers. Samples were then processed on site and stored at −80°C prior to shipping in dry ice as a single batch to the Melbourne Children's Bioresource Center (Murdoch Children's Research Institute) for processing. Further detail of blood collection, storage, and processing has been reported elsewhere.[64]

Biomarkers examined in this study included GlycA and ApoB/A1. GlycA and ApoB/A1 are relatively novel biomarkers, which reflect chronic inflammation and cholesterol balance, respectively.[65,66] Further detail of how these measures were derived have been reported elsewhere.[64]

Anthropometric measures. BMI was calculated from weight and height measures (kg/m^2), and waist circumference was used in the composite metabolic syndrome severity score. Weight was recorded via the InBody 230 Bioelectrical Impedance Analyser scales,[67] with participants barefoot and wearing light clothing. Height was assessed using a portable rigid stadiometer (Invicta IP0955, Leicester, UK). Waist circumference was measured by trained research assistants with a steel anthropometric measuring tape (Lufkin Executive Diameter W606PM, Maryland). Further detail of the methods used to collect anthropometric measures have been reported elsewhere.[64]

Statistical Analysis

All actigraphy variables were computed for each individual day and then averaged using a 5:2 weighting for weeknight (Sunday-Thursday) and weekend (Friday-Saturday). Data management and analyses were undertaken in R (version 4.1.0).

The distributions of cardiometabolic markers were examined for normality through visual inspection of histogram plots and assessment of kurtosis (>3) and skewness (>1). All continuous sleep measures and outcome variables were standardized as sample-specific z scores prior to analysis. To determine the correlation between perceived trouble sleeping and actigraphy-derived sleep characteristics, point biserial correlation coefficients were calculated.

The association between trouble sleeping and cardiometabolic markers was assessed in R version 4.1.0 using the geepack[68] package. Complete case analysis was undertaken. Generalized estimating equations (GEEs) were used in which standardized cardiometabolic health measures were considered as dependent variables. Robust standard errors (sandwich variance estimators) were used to account for the clustered sampling design of the study (geographic clustering of observations by postal code). All outcome measures were modeled with maximum likelihood (ML) for estimating variance and covariance parameters. Given that R^2 cannot be calculated in GEEs that account for clustering with ML estimates, pseudo-R^2 was calculated for each GEE model using the method suggested by Zheng[69] to determine the predictive power of troubled sleep and sleep characteristics. Consistent with any R^2 value, a pseudo-R^2 value is between 0 and 1.

Four models were examined to determine the predictive power of sleep characteristics. Model 1 only included the covariates (age, sex, and SEP). The second model additionally included the self-report sleep measure, and the model's pseudo-R^2 value was compared to that of Model 1 to determine how much additional variance in outcome was explained by self-report. The third model included covariates and objective measures of sleep, and the model's pseudo-R^2 value was compared to that of Model 1 to determine how much additional variance in outcome was explained by all objective measures. The fourth model included both self-report and objective sleep measures, and its pseudo-R^2 was compared to Model 1 to determine the predictive power of both measurement types together. Potential multicollinearity among actigraphy-derived sleep measures was assessed by quantifying variance inflation factor (VIF) and was low (VIF, 1.01-1.03). *P* values have been reported.

Results

Participant Characteristics

Of the 1874 parents who took part in the CheckPoint Study, 1045 had complete data on all measures considered for this study. As shown in Table 1, compared to participants excluded for analysis, participants included for analysis were slightly older, had a higher SEP and lower BMI, but no difference in self-report and objective sleep measures, except sleep efficiency. Valid sleep data were available for 1017 parents. As presented in Table 2, the mean age of adults was 44.8 years, with the majority being female (87%) and approximately half (48%) reporting that they never experienced troubled sleep. The SEP score was significantly lower in adults who reported more frequent experiences of troubled sleep. BMI, cholesterol balance (ApoB/A1), and inflammation (GlycA) was also higher with more frequent reports of troubled sleep.

Trouble Sleep and Objective Sleep Characteristics

As shown in Table 3, the correlations between actigraphy-derived sleep characteristics (sleep duration, timing, variability, and efficiency) and self-reported trouble sleeping were all less than .065 and only statistically significant for sleep duration and efficiency (r = .064, $P < .05$) and efficiency (r = −.072, $P < .05$).

Table 1. Comparison of Parents Included and Excluded for Analysis

	Excluded	Included	*P* Value
Demographics			
n	829	1045	
Sex (% females)	89%	87%	.190
Age (y)	44.0 (5.4)	44.6 (5.1)	**.004**
SEP	0.05 (1.02)	0.27 (0.96)	< **.000**
Trouble sleep			
Never	49%	48%	.721
Sometimes	35%	37%	
Often	15%	15%	
Sleep variables			
Sleep period (min)	499 (68)	499 (48)	.975
Sleep midpoint (24 h:min)	02:42 (74)	2:54 (50)	.100
Sleep efficiency (%)	84.9 (7.7)	86.2 (6.5)	**.001**
Sleep length variability (%)	10.1 (7.2)	10.2 (8.1)	.880
Cardiometabolic health			
BMI	28.2 (6.4)	27.5 (5.9)	**.014**
ApoB/A1	0.54 (0.14)	0.52 (0.14)	.097
GlycA	1.05 (0.16)	1.03 (0.17)	.100

The *P* value was calculated using analysis of variance for continuous measures and χ^2 for categorical measures (ie, sex and trouble sleeping). Unless otherwise reported, mean (SD) values are reported. ApoB/A1, apolipoprotein B/apolipoprotein A1; BMI, body mass index; GlycA, glycoprotein acetyls; SEP, socioeconomic position. Bold *P* values indicate significance <0.05.

Table 2. Demographic, Cardiometabolic, and Sleep Characteristics of Participants' Valid Sleep Data and Included for Analysis (n = 1017)

	Trouble Sleeping				
	All (n = 1017)	Never (n = 485)	Sometimes (n = 381)	Often (n = 151)	*P* Value
Sex (% female)	87	85	86	93	**.030**
Age (y)	44.8 (5.12)	44.6 (5.06)	45.1 (5.10)	44.5 (5.33)	.248
SEP	0.27 (0.96)	0.93 (0.93)	0.94 (0.93)	1.05 (1.07)	< **.000**
Sleep period (min)	496 (56)	496 (57)	496 (52)	510 (60)	**.020**
Sleep midpoint (24 h:min)	02:53 (49)	02:55 (48)	02:49 (48)	02:57 (52)	.107
Sleep efficiency (%)	86.1 (6.5)	86.5 (6.5)	86.2 (6.3)	84.9 (6.8)	**.028**
Sleep length variability (%)	10.2 (7.9)	9.9 (7.9)	10.4 (7.8)	10.8 (8.3)	.387
BMI	27.5 (5.9)	27.0 (5.6)	27.5 (5.9)	28.8 (6.5)	**.006**
ApoB/A1	0.52 (0.14)	0.51 (0.14)	0.53 (0.15)	0.54 (0.14)	.072
GlycA	1.03 (0.17)	1.02 (.17)	1.04 (0.17)	1.06 (0.16)	**.012**

The *P* value was calculated using analysis of variance for continuous measures and χ^2 for categorical measures (ie, sex). Unless otherwise reported, mean (SD) values are reported. ApoB/A1, apolipoprotein B/apolipoprotein A1; BMI, body mass index; GlycA, glycoprotein acetyls; SEP, socioeconomic position. Bold *P* values indicate significance <0.05.

Table 3. Point-Biserial Correlation Coefficients for Perceived Trouble Sleeping and Actigraphy-Derived Sleep Characteristics

	Sleep Period	Sleep Midpoint	Sleep Length Variability	Sleep Efficiency
Trouble sleeping	.064*	.002	.040	–.072*

Trouble sleeping = never, sometimes, or often.
*$P < .05$.

Sleep and Cardiometabolic Risk Factors for Type 2 Diabetes

Self-report and objective measures of sleep were significantly associated with cardiometabolic risk factors for type 2 diabetes. As shown in Tables 4 to 6, more frequent reports of troubled sleep were significantly associated with higher BMI, GlycA, and ApoB/A1 (Model 2), even after adjusting for objective dimensions of sleep (Model 4). Actigraphy-derived sleep characteristics were inconsistently associated with the cardiometabolic health outcome measures (Model 3). Specifically, later sleep timing was significantly associated with higher ApoB/A1 ($\beta = 0.11$, 95% CI, 0.05-0.17, $P \leq .001$) and GlycA ($\beta = 0.06$, 95% CI, 0.00-0.12, $P = .037$), longer sleep period was significantly associated with higher GlycA ($\beta = 0.06$, 95% CI, 0.01-0.11, $P = .026$), and greater sleep length variability was significantly associated with higher BMI ($\beta = 0.07$, 95% CI, 0.02-0.13, $P = .008$).

Trouble sleeping was a modest predictor of cardiometabolic health (Model 2: pseudo-R^2, .052-.121), similar to the predictive power of actigraphy-measured sleep characteristics (Model 3: pseudo-R^2, .055-.128), with negligible change when all dimensions were added to the same model (Model 4: pseudo-R^2, .060-.134). Covariates (age, sex, SEP) and clustering explained most of the overall predictive power (Model 1: pseudo-R^2, .046-.115).

Discussion

Both self-report and objective measures of sleep were significant independent predictors of cardiometabolic health. A relationship was observed for self-reported troubled sleep and measures of adiposity, inflammation, and cholesterol balance. This relationship remained even after adjusting for covariates and objective measures of sleep. Although weak and mostly explained by the covariates (age, sex, SEP), self-report and objective measures of sleep had similar predictive power. Consistent with previous studies, very weak, albeit significant, relationships between trouble sleeping and actigraphic measures of sleep duration and efficiency were identified.[27-32] Self-report trouble sleeping may be as strong as actigraphy-derived measures of sleep in predicting cardiometabolic risk factors associated with type 2 diabetes. Although the predictive power of both self-report and objective measures of sleep was small, it is important to note that such small values are typical for studies that examine human behavior in social science research.

Relatively few studies have examined the association between the self-reported sleep experience and cardiometabolic health. The self-report sleep experience was measured in terms of a global measure of self-report troubled sleep. Although direct comparisons cannot be made, findings align with previous studies that have examined the association between self-report trouble sleeping and the risk of type 2 diabetes.[42-44] Studies that use other self-report measures of sleep yield inconsistent findings.[70,71]

While there have been efforts to understand the interpretation and meaning of various single-item questions, limited research has examined the global measure of "trouble sleeping."[26,72,73] Very weak relationships ($r < .065$) were observed between self-reported trouble sleeping and actigraphy-derived measures of sleep. However, it is possible that trouble sleeping may reflect characteristics of sleep not assessed (eg, wake after sleep onset) or the integrated effects of multiple sleep characteristics.[35]

Alternatively, it is also plausible that trouble sleeping simply reflects psychological attributes, which may also influence health. For instance, self-reported sleep quality has been associated with levels of conscientiousness (which relates to impulse control and the ability to complete tasks), neuroticism (which relates to emotional reactivity), and optimism (which has been associated with engagement in health-promoting behaviours).[34,74,75] People who complain of more troubled sleep may therefore simply be less optimistic or have lower levels of engagement in health-promoting activities, poorer coping mechanisms, and greater emotional lability—attributes that may affect cardiometabolic health. This may help explain the general gradient toward poorer cardiometabolic health with more frequent reports of troubled sleep.

Weak and inconsistent associations were observed between actigraphy-derived sleep parameters and cardiometabolic health. Given that *all* sleep characteristics are considered important for cardiometabolic health, our finding are somewhat surprising. However, it is important to note that most participants in the current study slept within the recommended amount[76] and were relatively healthy, which may explain the weak associations.

Strength and Limitations

To our knowledge, this is the first study to examine the association between self-reported trouble sleeping, a range of actigraphy-measured sleep characteristics, and cardiometabolic risk factors known to predict type 2 diabetes in

Table 4. Generalized Estimating Equation Models of Associations Between Trouble Sleeping, Actigraphy-Derived Sleep Characteristics, and Body Mass Index

	Model 1 (Covariates Only)			Model 2 (Self-report Sleep)			Model 3 (Actigraphy-derived Sleep)			Model 4 (Self-report and actigraphy-derived sleep)		
	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value
Intercept	0.50	−0.10 to 1.11	.102	0.48	−0.12 to 1.09	.119	0.45	−0.16 to 1.05	.148	0.43	−0.18 to 1.04	.166
Females	−0.21	−0.37 to −0.05	**.010**	−0.23	−0.39 to −0.07	**.005**	−0.18	−0.34 to −0.02	**.024**	−0.20	−0.36 to −0.04	**.014**
Males (reference)												
SEP	−0.20	−0.28 to −0.13	< **.001**	−0.19	−0.26 to −0.12	< **.001**	−0.19	−0.27 to −0.12	< **.001**	−0.18	−0.26 to −0.11	< **.001**
Age	−0.00	−0.00 to 0.00	.372	−0.00	−0.00 to 0.00	.315	−0.00	−0.00 to 0.00	.425	−0.00	−0.00 to 0.00	.362
Trouble sleeping: often				0.22	0.03 to 0.41	**.023**				0.21	0.02 to 0.39	**.032**
Trouble sleeping: sometimes				0.09	−0.03 to 0.20	.154				0.08	−0.04 to 0.20	.182
Trouble sleeping: never (reference)												
Sleep length variability							0.07	0.02 to 0.13	**.008**	0.07	0.02 to 0.12	**.011**
Sleep timing							0.01	−0.05 to 0.07	.754	0.01	−0.05 to 0.07	.721
Sleep period							−0.02	−0.07 to 0.04	.561	−0.02	−0.07 to 0.03	.479
Sleep efficiency							−0.04	−0.10 to 0.01	.123	−0.04	−0.10 to 0.02	.160
Pseudo-R^2	.046			.052			.055			.060		

Abbreviations: CI, confidence interval; SEP, socioeconomic position. Bold *P* values indicate significance <0.05.

Table 5. Generalized Estimating Equation Models of Associations Between Trouble Sleeping, Actigraphy-Derived Sleep Characteristics, and Apolipoprotein B/Apolipoprotein A1

	Model 1 (Covariates Only)			Model 2 (Self-report sleep)			Model 3 (Actigraphy-derived Sleep)			Model 4 (Self-report and actigraphy-derived sleep)		
	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value
Intercept	0.70	0.14 to 1.26	**.014**	0.67	0.11 to 1.23	**.018**	0.71	0.16 to 1.27	**.012**	0.69	0.13 to 1.24	**.016**
Females	−0.91	−1.14 to −0.69	**< .001**	−0.93	−1.15 to −0.71	**< .001**	−0.92	−1.14 to −0.70	**< .001**	−0.93	−1.15 to −0.71	**< .001**
Males (reference)												
SEP	−0.15	−0.22 to −0.07	**< .001**	−0.14	−0.21 to −0.06	**< .001**	−0.13	−0.21 to −0.06	**.001**	−0.13	−0.20 to −0.05	**.001**
Age	0.00	−0.00 to 0.00	.578	0.00	−0.00 to 0.00	.697	0.00	−0.00 to 0.00	.610	0.00	−0.00 to 0.00	.746
Trouble sleeping: often				0.17	−0.01 to 0.34	.058				0.16	−0.02 to 0.33	.075
Trouble sleeping: sometimes				0.13	0.01 to 0.25	**.030**				0.15	0.03 to 0.26	**.016**
Trouble sleeping: never (reference)												
Sleep length variability							0.01	−0.05 to 0.06	.842	0.00	−0.05 to 0.06	.926
Sleep timing							0.11	0.05 to 0.17	**< .001**	0.11	0.05 to 0.17	**< .001**
Sleep period							0.04	−0.02 to 0.09	.168	0.04	−0.02 to 0.09	.183
Sleep efficiency							−0.02	−0.09 to 0.06	.630	−0.01	−0.09 to 0.06	.695
Pseudo-R^2	.115			.121			.128			.134		

Abbreviations: CI, confidence interval; SEP, socioeconomic position. Bold *P* values indicate significance <0.05.

Table 6. Generalized Estimating Equation Models of Associations Between Trouble Sleeping, Actigraphy-Derived Sleep Characteristics and Glycoprotein Acetyls

	Model 1 (Covariates Only)			Model 2 (Self-report Sleep)			Model 3 (Actigraphy-derived Sleep)			Model 4 (Self-report and actigraphy-derived sleep)		
	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value	β	95% CI	*P* Value
Intercept	0.72	0.13 to 1.31	**.016**	0.69	0.11 to 1.28	**.021**	0.72	0.12 to 1.31	**.018**	0.69	0.10 to 1.29	**.023**
Females	−0.53	−0.75 to −0.30	< .001	−0.54	−0.77 to −0.32	< **.001**	−0.53	−0.76 to −0.30	< **.001**	−0.54	−0.77 to −0.32	< **.001**
Males (reference)												
SEP	−0.19	−0.26 to −0.12	< **.001**	−0.18	−0.25 to −0.11	< **.001**	−0.17	−0.24 to −0.10	< **.001**	−0.17	−0.24 to −0.10	< **.001**
Age	−0.00	−0.00 to 0.00	.427	−0.00	−0.00 to 0.00	.337	−0.00	−0.00 to 0.00	.432	−0.00	−0.00 to 0.00	.342
Trouble sleeping: often				0.20	0.02 to 0.38	**.032**				0.17	−0.01 to 0.36	.070
Trouble sleeping: sometimes				0.14	0.02 to 0.25	**.026**				0.14	0.02 to 0.26	**.022**
Trouble sleeping: never (reference)												
Sleep length variability							0.01	−0.04 to 0.06	.777	0.00	−0.05 to 0.05	.864
Sleep timing							0.06	0.00 to 0.12	**.037**	0.07	0.01 to 0.13	**.028**
Sleep period							0.06	0.01 to 0.11	**.026**	0.06	0.01 to 0.11	**.030**
Sleep efficiency							−0.08	−0.16 to 0.00	.064	−0.08	−0.16 to 0.01	.077
Pseudo-R^2	.067			.074			.083			.089		

Abbreviations: CI, confidence interval; SEP, socioeconomic position. Bold *P* values indicate significance <0.05.

a large sample of Australian adults. There are a number of limitations that need to be acknowledged. First, most participants were mothers. Previous studies suggest females tend to report higher levels of sleep dissatisfaction.[77,78] The sample characteristics preclude generalization to fathers, nonparents, parents of younger or older children, and adults in general. Second, the global measure of perceived trouble sleeping may not adequately capture different aspects of troubled sleep (eg, trouble initiating sleep, maintaining sleep, and waking too early). Third, only 4 actigraphy-derived sleep characteristics were examined. It is possible that other characteristics might be more strongly correlated to self-reported trouble sleeping. It is also important to note that validity studies tend to suggest that while activity monitors generally have good sensitivity with polysomnography, specificity tends to be poor.[79] Validity of monitors therefore vary according to the sleep parameter assessed. Furthermore, while we controlled for age, sex, and SEP, it is unknown whether participants in the current study had an underlying sleep disorder or other comorbidities (eg, anxiety and depression). Lastly, this study is cross-sectional and cannot imply causality.

Future Directions

A significant independent association was found between self-reported trouble sleeping and cardiometabolic health in Australian parents, predominantly mothers. Self-reported trouble sleeping is just one way to describe and measure the subjective sleep experience. There is currently no consensus for how to best define the self-reported sleep experience, and it remains unclear as to whether trouble sleeping reflects a unique dimension of sleep or nonsleep phenomena. In line with growing interest to examine sleep as a multidimensional construct with objective and self-report sleep characteristics, future efforts are needed to better understand how to best measure and examine the self-reported sleep experience and distinguish how it may differ from actigraphy-derived measures of sleep, psychological attributes, and sociodemographic and lifestyle correlates.

Conclusions

Sleep is increasingly recognized as a multidimensional construct, important for cardiometabolic health and the risk of type 2 diabetes. Poorer cardiometabolic health was observed for more frequent reports of troubled sleep. The predictive power of objective and self-report characteristics of sleep was similar. While findings support suggestions[80] to consider self-report measures of sleep alongside objective measures of sleep, further research is needed to better understand self-report experiences of sleep.

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ORCID iD

Lisa Matricciani https://orcid.org/0000-0002-4480-4188

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