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Objectively-measured sleep patterns and cardiometabolic health in a rural South African setting: a cross sectional analysis

Ian Cook[1*] and Matlawa Mohlabe[2]

Abstract

Background: To investigate the relationship between objectively-measured, free-living sleep patterns, and cardiometabolic health, in a rural South African health and demographic surveillance site.

Methods: Wrist-mounted actigraphy data was collected over nine days from 167 adults (≥ 40 years). Sleep patterns were constructed from tertiles of sleep quantity and quality parameters (TST: total sleep time, AC: activity counts during sleep) from valid minute-by-minute data. The reference category was Moderate TST/Low-to-Moderate AC. Self-reported data included behavioural, health and socio-demographic variables. Biological data included anthropometry, resting blood pressure and fasting blood glucose, insulin and lipids. Binary and ordinal logistic regression models were constructed to determine the association between TST and AC, the factors associated with sleeping patterns, and the association between sleeping patterns and Insulin resistance (HOMA-IR) and Metabolic Syndrome (MetS). HOMA-IR and MetS were also examined across sleep patterns using analysis of variance models.

Results: A total of 139 adults (71.2% female) had a complete dataset. In unadjusted analyses, females had poorer sleep quality, were more physically active, and displayed poorer cardiometabolc health and greater adiposity than males ($p \leq 0.017$). There were no sex differences in TST or sleep pattern distribution ($p \leq 0.901$). Not being classified as Low TST/High AC or exposed to ≥ 1 bout of Low TST/High AC sleep was associated with lower physical activity, longer sleep duration, better sleep quality and lower IR ($p \leq 0.0452$). In multivariate analyses, there was no association between TST and AC ($p = 0.921$), while increasing age and people-to-bedroom density, and lower physical activity where significantly associated with increasing TST ($p \leq 0.027$). Participants classified as Low TST/High AC had significantly higher HOMA-IR, but not MetS, compared with Moderate TST/Low AC ($p = 0.021$). Being exposed to ≥ 1 bout of Low TST/High AC sleep was significantly associated with hypertension (OR = 2.31, 95%CI: 1.00, 5.34), but not for HOMA-IR or MetS ($p \geq 0.227$).

Conclusions: Long sleep was not associated with increased sleep fragmentation. Short, fragmented sleep was associated with insulin resistance. Exposure to at least one bout of short, fragmented sleep increased the likelihood of hypertension. Further studies are required to identify the factors associated with short, fragmented sleep in this setting.

Keywords: Insulin resistance, Body composition, Accelerometer, Actigraphy, Movement monitor, Measurement, Metabolic syndrome

*Correspondence: ian.cook@ul.ac.za
[1] Physical Activity Epidemiology Laboratory (EDST), University of Limpopo (Turfloop Campus), Sovenga, Mankweng, Limpopo Province, South Africa
Full list of author information is available at the end of the article

Background

To date, self-reported sleep duration data within the South African context, has revealed perplexing differences within different ethnic communities. The observation has been made that the phenomenon of long, self-reported sleep duration is consistently reported in some South African ethnic communities, irrespective of the level of urbanization and age (Rae et al. 2020, 2018). Indeed, within a rural African setting, self-reported sleep duration exceeded nine hours per night (Mashinya et al. 2018). Some speculate that this long self-reported sleep duration may indicate poor sleep quality, which in turn might be related to environmental and socio-demographic factors (Rae et al. 2020, 2018). In addition, the accuracy with which sleep is reported, and how sleep questions are interpreted across different populations is unknown (Rae et al. 2020, 2018), which highlights the need for objectively-measured sleep data. We have recently reported some of the first free-living, objectively-measured sleep data in a rural South African setting (Cook et al. 2020, 2021). Compared with current self-report data, average sleep time is 2 h or less, and there is a lower prevalence of long sleep (4.1%) (Cook et al. 2020). In addition, sleep quality measures were more consistently and independently associated with increasing levels of insulin resistance than sleep quantity (Cook et al. 2021). Few studies have explored the interaction between sleep quantity and quality, and cardiometabolic health (Lu et al. 2020, 2015; Ji et al. 2020; Lou et al. 2014), especially using objective measures of sleep (Domínguez et al. 2019).

Further investigation is suggested, given the importance of sleep behaviours in relation to cardiometabolic health (Smiley et al. 2019), and the current uncertainty around whether poor sleep quality undergirds long sleep durations within specific South African contexts. The objectives of this analysis were first to establish whether increasing levels of objectively-measured poor sleep quality are associated with longer sleep durations, using an unbiased measure of sleep quality. Second, to investigate whether constructed sleeping patterns (comprised of sleep quantity and quality measures) are associated with poor cardiometabolic health. In addition, if exposure to a poor sleeping pattern is associated with poor cardiometabolic health. Third, to determine what biological, behavioural and social factors are associated with levels of exposure to the constructed sleeping pattern.

Methods

Dikgale Health and Demographic Surveillance System site sample

We recruited a convenience sample of 167 adults ($\geq$ 40 years), permanently resident in the Dikgale site for at least 6 months of the year (Alberts et al. 2015) and part of the larger AWI-Gen Phase 1 Study cohort. Pregnant women were excluded from the study (Mashinya et al. 2018; Ali et al. 2018). Self-reported, measured and biological data were collected from participants by trained field workers using questionnaires, anthropometry, oscillometric blood pressure measurement, ultrasound scans (not considered for this analysis), and venipuncture (Mashinya et al. 2018; Ali et al. 2018). Anthropometric indices were calculated (Body Mass Index, kg/m^2; Conicity Index) (Valdez et al. 1993)). Questionnaire data included behavioural, health and socio-demographic variables (Mashinya et al. 2018; Ali et al. 2018). Poor cardiometabolic health was defined as an increase in risk factors for cardiometabolic diseases (cardiovascular disease and type 2 diabetes mellitus). The risk factors include obesity, hypertension, dyslipidemia, insulin resistance and hyperglycaemia (Rangaraj and Knutson 2016). Free-living, wrist-mounted actigraphy data was collected over nine days (Cook et al. 2020).

Blood sample collection and analysis

Fasting blood samples were collected by a registered nurse and the samples were analysed centrally (Ali et al. 2018). The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) was calculated from fasting blood glucose and insulin (Matthews et al. 1985).

Criteria for Metabolic Syndrome

As described in detail elsewhere (Cook et al. 2021), we used the harmonized Joint Interim Statement (JIS) definition (Alberti et al. 2009) to determine the presence of the Metabolic Syndrome (MetS). From the five criteria of the JIS definition for MetS, we calculated sex-specific z-scores for each criterion, which were summed to create a MetS z-Score (Lee et al. 2019).

Accelerometer data collection and data reduction

Participants wore a wrist-mounted tri-axial accelerometer for nine days (ActiGraph wGT3X-BT, Actigraph, LLC, Pensacola, FL, 2013) (Whitaker et al. 2018; Migueles et al. 2017; Slater et al. 2015; Full et al. 2018). The deployment, data processing and extraction of accelerometry-derived physical activity (PA) and sleep parameters are described in detail elsewhere (Cook et al. 2020). Variables were expressed as daily averages. In addition, we also extracted day-by-day data for each sleep period.

Vector Magnitude (VM)- and Ambulation-defined PA variables were reported as counts/day and steps/day, respectively (Wennman et al. 2019). The sleep quantity measure for this analysis was Total Sleep Time (TST). Sleep quality indices were Sleep Fragmentation Index, Sleep Efficiency, Wake after Sleep Onset and Activity

Counts during the sleep period (AC) (Actigraph 2012; Ancoli-Israel et al. 2015).

Sleep Fragmentation Index, Sleep Efficiency and Wake After Sleep Onset were significantly correlated with TST ($p \leq 0.0143$, $r = -0.61$, $r = 0.59$, $r = -0.21$, respectively). Because AC was not correlated with TST ($r = -0.02$, $p = 0.8447$), but was significantly associated with Sleep Fragmentation Index, Sleep Efficiency and Wake After Sleep Onset ($p \leq 0.0129$, $r = 0.21$, $r = -0.69$, $r = 0.83$, respectively), AC was used as the sleep quality variable (Hung et al. 2013). We have found increasing levels of AC to be significantly and independently associated with increasing HOMA-IR levels (linear trend, $p = 0.021$) and being in the third, compared to the first and second tertiles of AC, increased the odds of being in the highest HOMA-IR tertile three-fold ($p = 0.020$) (Cook et al. 2021).

For the average daily and day-by-day sleep data (TST, AC), tertiles were created for each variable (Q_1 = Low/Short, Q_2 = Moderate, Q_3 = High/Long). Through cross tabulation of TST and AC tertiles, six sleep patterns were obtained:

- Short Sleep – Low to Moderate AC
- Short Sleep – High AC
- Moderate Sleep – Low to Moderate AC
- Moderate Sleep – High AC
- Long Sleep – Low to Moderate AC
- Long Sleep – High AC

For subsequent analysis the Moderate Sleep – Low to Moderate AC category was chosen as the reference group.

Statistical analyses

Descriptive statistics comprised means (one standard deviation), medians (inter-quartile range), variances (minimum, maximum) and frequencies. Bivariate relationships were examined using logistic regression. Relationships between categorical variables were examined through Fisher's Exact Test. For continuous data, independent *t* tests and Levene's test examined differences between sexes. Non-normally distributed variables were log transformed or a non-parametric test was employed. Trends across groups were evaluated using contrasts from bivariate linear regression, and where required, non-parametric methods were employed.

We constructed two ordinal logistic regression models to examine first, the relationship between sleep quantity (TST) and sleep quality (AC), and second to explore the factors associated with the levels of exposure to a poor sleep pattern. An Analysis of Variance model was constructed to examine measures of cardiometabolic health across sleep patterns. Finally, we constructed binary and ordinal logistic regression models to explore the relationship between MetS and HOMA-IR, respectively, and levels of exposure to a poor sleep pattern.

The purpose of multivariate binary and ordinal logistic regression in this analysis was to evaluate the robustness of bivariate associations, given important co-variates.

A forced-entry ordinal logistic regression model, with post hoc contrasts, was constructed to examine the relationship between quantiles of sleep time (TST) and sleep quality (AC), The model was adjusted for socio-demographic (age, socio-economic status, partnership status, people-to-bedroom density), behavioural (tobacco and alcohol usage, Vector Magnitude) and biological (sex, Conicity Index, HIV status, presence of hypertension and diabetes mellitus) variables.

Unadjusted and adjusted (age, sex, socio-economic status, fruit and vegetable intake, sugar-sweetened beverages, alcohol and tobacco usage, Vector Magnitude, Conicity Index, HIV status) Analysis of Variance models were run to examine the effect of sleep pattern on HOMA-IR and MetS z-score. Conicity Index was included in the HOMA-IR model but not the MetS z-score model, because waist circumference formed part of the dependent variable. *Post-hoc* comparisons were run across sleep patterns, with an a priori defined reference group.

Day-by-day data were analyzed to identify individuals exposed to ≥ 1 episode of a high-risk sleep pattern (based on the Analysis of Variance models). Forced-entry binary logistic regression models were constructed to examine the relationship between MetS risk (low/high) according to the JIS definition (Alberts et al. 2015), and exposure (Yes/No) to ≥ 1 episode of a high-risk sleep pattern. Models were adjusted for age, sex, socio-economic status, fruit and vegetable intake, sugar-sweetened beverages, alcohol and tobacco usage, Vector Magnitude and HIV status. Conicity Index was not included as an independent variable because waist circumference formed part of the JIS risk definition. Likewise, forced-entry, adjusted ordinal logistic regression models were constructed to examine the relationship between HOMA-IR levels (tertiles), and exposure (Yes/No) to ≥ 1 episode of a high-risk sleep pattern, using the same co-variates as for the binary models but with the additional inclusion of Conicity Index.

Finally, a forced-entry ordinal logistic regression model, with post hoc contrasts, was obtained to identify the factors associated with levels of exposure to a high-risk sleeping pattern (based on the Analysis of Variance analysis). The model was adjusted for socio-demographic, behavioural and biological variables.

Regression coefficients for binary and ordinal logistic regression models were expressed as odds ratios (OR ± 95% confidence intervals). Goodness of fit criteria included Akaike's Information Criteria and pseudo R^2. Data were analysed using appropriate statistical software (Stata/SE for Windows: Release 15.1. College Station, TX: StataCorp LP, 2020). Significance for all inferential statistics was set at $p \leq 0.05$.

Results

There were 157ex167 raw accelerometry data files with valid data for at least one weekday and one weekend day, and once combined with the questionnaire and biological data, 139 had complete data. A total of 1101 sleep periods were recorded.

Females consumed more sugar-sweetened beverages, used less tobacco and alcohol, reported a higher people-to-bedroom density, self-reported less sleep, were more physically active, had poorer sleep quality, and displayed greater adiposity and poorer cardiometabolic health, compared with males ($p \leq 0.040$, Table 1). The distribution of sleep pattern categories did not differ between the sexes ($p \geq 0.709$).

There was no bivariate or multivariate association between TST and AC ($p \geq 0.440$, see Additional File 1). Increasing TST was significantly and independently associated with increasing age (OR = 1.16, 95%CI: 1.02 – 1.31), increasing people-to-bedroom density (OR = 1.32, 95%CI: 1.03 – 1.70) and lower Vector Magnitude (OR = 0.56, 95%CI: 0.37 – 0.85) (see Additional File 1).

There were significant, non-adjusted trends in biological, socio-demographic and health behaviour variables, across exposure levels to short, fragmented sleep (Table 2).

The proportion of females increased with increasing level of exposure ($p = 0.0263$). Higher levels of tobacco and alcohol usage were associated lower levels of exposure to short, fragmented sleep ($p = 0.0360$). Both sleep quantity and quality indices worsened with increasing exposure to short, fragmented sleep ($p < 0.001$). PA and HOMA-IR were directly associated and non-linearly related with increased exposure to short, fragmented sleep ($p \leq 0.0452$), respectively (Table 2).

When adjusted for socio-demographic, behavioural and biological variables, there was a significant difference between sleep patterns. HOMA-IR for short, high disturbance sleep was significantly higher than HOMA-IR for moderate, low disturbance sleep ($p = 0.021$) (Fig. 1). The MetS z-score adjusted model was not significant ($p = 0.4538$). There was no significant interaction between the effects of gender and sleep pattern in either the HOMA-IR or MetS z-score models ($p \geq 5702$, Fig. 1).

In contrast to highly fragmented AC sleep patterns, low-to-moderately fragmented AC sleep patterns constituted the majority of the average daily sleep pattern (66.8%, see Additional File 2: Figure S1) and tended to be more variable across days (variance: 7.1%—14.0%). The short sleep—high AC sleep pattern was the most invariant sleep pattern across days (variance: 2.2%) and contributed the least toward the daily sleep pattern (9.1%, see Additional File 2: Figure S1).

There was no significant bivariate association between level of exposure to short, fragmented sleep and any cardiometabolic health parameter ($p \geq 0.174$, see Additional File 1).

In multivariate analysis, being exposed to ≥ 1 bout of short, fragmented sleep, was not associated with a greater likelihood of being in HOMA-IR Q3, compared with HOMA-IR Q1 and Q2 (adjusted OR: 1.11, 95%CI: 0.56 – 2.19) (see Additional File 1). Excluding participants classified as having short, fragmented sleep on the basis of average values over the monitoring period ($n = 16$), did not alter the point statistic (OR: 1.01, 95%CI: 0.48 – 2.13).

Classification as having MetS JIS or individual constituent components, except for blood pressure, were not associated with exposure to short, fragmented sleep ($p \geq 0.227$) (see Additional File 1). There was a significant 2.31 greater adjusted-odds (95%CI: 1.00 – 5.34) of being in the JIS-defined blood pressure risk group (≥ 130/85 mmHg) with exposure to ≥ 1 bout of short, fragmented sleep (see Additional File 1). Excluding participants classified as having short, fragmented sleep on the basis of average values over the monitoring period ($n = 16$), resulted in a similar odds ratio for JIS-defined blood pressure (OR: 2.58, 95%CI: 1.01 – 6.33).

Multivariate-adjusted socio-demographic and behavioural factors were associated with increasing levels of exposure to short, fragmented sleep (see Additional File 1, Collinearity: VIF ≤ 1.22, Tolerance ≤ 0.9589). People-to-bedroom density, SES, tobacco and alcohol use and PA were significantly and independently associated with exposure to short, fragmented sleep ($p \leq 0.040$). A change in people-to- bedroom density status and SES (increasing group quintile status, $Q_i \rightarrow Q_{i+1}$) decreased the odds of chronic exposure versus the combined acute-no exposure, or no exposure versus the combined acute-chronic exposure by 24% (OR = 0.76, 95%CI: 0.58 – 0.99) and 27% (OR = 0.73, 95%CI: 0.37 – 0.95), respectively. Similarly, increasing exposure to tobacco and alcohol (tertiles), decreased the odds to increased short, fragmented sleep exposure levels by 45% (OR = 0.55, 95%CI: 0.37 – 0.95). An increase in PA level (VM tertile status) increased the odds of chronic exposure versus the combined acute-no exposure, or no exposure versus the combined acute-chronic exposure by 2.32-fold (95%CI: 1.46 – 3.67).

Table 1 Descriptive statistics by sex

	All ($n=139$)	Female ($n=99$)	Male ($n=40$)	*p*-value
Age (years)	52.6 (7.1)	52.0 (6.9)	53.9 (7.5)	0.165
SES Quintile	3.6 (1.3)	3.6 (1.3)	3.5 (1.3)	0.652
Sugar sweetened beverages (servings/day) [a]	0.29 (0.00)	0.29 (0.10)	0.29 (0.00)	**0.010**
Fruit and vegetable intake (servings/day) [a]	1.29 (0.71)	1.29 (1.00)	1.14 (0.36)	0.070
Marital status (Married/Co-habiting) [b]	54.7 (76)	54.5 (54)	55.0 (22)	0.648
Housing density (people/bedroom) [a]	1.50 (1.25)	1.67 (1.25)	1.37 (1.00)	**0.040**
Alcohol & Tobacco use (both current; Yes) [b]	18.7 (26)	8.1 (8)	45.0 (18)	**< 0.001**
Hypertension and diabetes present (both concurrent; Yes) [b]	11.5 (16)	12.1 (12)	10.0 (4)	0.939
HIV status (Yes) [b]	23.0 (32)	22.2 (22)	25.0 (10)	0.824
Self-reported sleep (hours/night)	9.2 (1.5)	9.0 (1.5)	9.7 (1.4)	**0.0204**
Objectively-measured Sleep Indices				
Total Sleep Time (minutes/day)	457 (80)	458 (67)	456 (106)	0.901
Wake after Sleep Onset (minutes)	51 (17)	54 (16)	44 (17)	**0.001**
Sleep Fragmentation Index (%)	27.1 (6.9)	26.7 (7.0)	28.2 (6.6)	0.243
Sleep Efficiency (%)	88.2 (4.4)	87.6 (4.2)	89.5 (4.6)	**0.017**
Activity Counts (counts)	32 868 (12 231)	35 058 (12 135)	27 448 (10 818)	**0.001**
Categories of sleep patterns [b]				
Total Sleep Time vs. Activity Counts:				
Short sleep – low to moderate AC	21.6 (30)	21.2 (21)	22.5 (9)	0.997
Short sleep – high AC	11.5 (16)	12.1 (12)	10.0 (4)	
Moderate sleep – low to moderate AC	23.0 (32)	23.2 (23)	22.5 (9)	
Moderate sleep – high AC	10.8 (15)	10.1 (10)	12.5 (5)	
Long sleep – low to moderate AC	22.3 (31)	22.2 (22)	22.5 (9)	
Long sleep – high AC	10.8 (15)	11.1 (11)	10.0 (4)	
Exposure to Short sleep – high AC [b]				
No exposure	57.6 (80)	55.6 (55)	62.5 (25)	0.709
1 bout	26.7 (37)	27.3 (27)	25.0 (10)	
$\geq$ 2 bouts	15.8 (22)	17.2 (17)	12.5 (5)	
Objectively-measured Physical Activity				
VM counts/day ($\times 10^6$)	2.35 (0.70)	2.49 (0.63)	2.00 (0.77)	**< 0.001**
Steps per day	14 531 (4662)	14 288 (3691)	15 132 (6492)	0.443
Conicity Index	1.23 (0.10)	1.23 (0.10)	1.23 (0.08)	0.940
Waist circumference (cm)	89.5 (15.7)	93.0 (16.3)	80.8 (9.7)	**< 0.001**
Body mass index (kg/m^2)	27.9 (7.8)	30.4 (7.6)	21.7 (3.8)	**< 0.001**
Body mass index categories [b]				
Under- weight (< 18.5 kg/m^2)	7.2 (10)	3.0 (3)	17.5 (7)	**< 0.001**
Normal weight (18.5–24.9 kg/m^2)	30.9 (43)	21.2 (21)	55.0 (22)	
Overweight (25 – 29.99 kg/m^2)	24.5 (34)	23.2 (23)	27.5 (11)	
Obese ($\geq$ 30 kg/m^2)	37.4 (52)	52.5 (52)	0.0 (0.0)	
MetS z-Score [c]	7.2 (-7.3; 9.4)	7.7 (-7.3; 9.4)	6.1 (-4.5; 6.6)	**< 0.001**
HOMA-IR [d]	1.32 (2.47)	1.51 (2.37)	0.96 (2.54)	**0.003**

Data reported as mean (SD), [a] median (IQR), [b] % (n), [c] variance (minimum; maximum) or [d] geometric mean (SD), *AC* Activity Counts, *HIV* Human Immunodeficiency Virus, *HOMA-IR* Homeostasis Model Assessment of Insulin Resistance, *MetS* Metabolic Syndrome, *SES* Socio-Economic Status, *VM* Vector Magnitude

Of note that VM was significantly associated with TST and the level of exposure to short, fragmented sleep, in both bivariate and multivariate analyses ($p \leq 0.04$, see Additional File 1).

Discussion

This analysis is novel in that, as far as the authors are aware, this is the first free-living, actigraphy-measured sleep study which has considered the sleeping pattern and cardiometabolic health from a South African

Table 2 Descriptive statistics by level of exposure to short, fragmented sleep

	Chronic exposure (monitoring period average) ($n=16$)	Acute exposure (≥ 1 bout during the monitoring period) ($n=46$)	No exposure during the monitoring period ($n=77$)	Trend *p*-value
Sex [a]				
Female	75.0 (12)	71.7 (33)	70.1 (54)	**0.0263**
Male	25.0 (4)	28.3 (13)	29.9 (23)	0.6947
Age (years)	51.1 (5.4)	53.0 (7.0)	52.6 (7.5)	0.4485
SES Quintile	3.4 (1.2)	3.5 (1.4)	3.7 (1.3)	0.3126
Sugar sweetened beverages (servings/day) [b]	0.29 (0.0)	0.29 (0.00)	0.29 (0.14)	0.0600
Fruit and vegetable intake (servings/day) [b]	1.14 (0.29)	1.28 (0.57)	1.28 (1.28)	0.2240
Marital status (Married/Co-habiting) [a]	56.3 (9)	52.2 (24)	55.8 (43)	0.4820
Housing density (people/bedroom) [b]	1.0 (1.2)	1.5 (1.3)	1.7 (1.7)	0.0850
Alcohol & Tobacco use (both current; Yes) [a]	12.5 (2)	13.0 (6)	23.4 (18)	**0.0360**
Hypertension and diabetes present (both concurrent; Yes) [a]	6.3 (1)	15.2 (7)	10.4 (8)	07,760
HIV status (Positive) [a]	12.5 (2)	21.7 (10)	26.0 (20)	0.2481
Self-reported sleep (hours/night)	9.5 (2.0)	9.2 (1.3)	9.2 (1.5)	0.4576
Objectively-measured Sleep Indices				
Total Sleep Time (minutes/day)	318 (34)	373 (56)	444 (79)	**<0.0001**
Wake after Sleep Onset (minutes)	74 (12)	53 (14)	45 (15)	**<0.0001**
Sleep Fragmentation Index (%)	33.7 (5.5)	29.0 (6.1)	24.6 (6.4)	**<0.0001**
Sleep Efficiency (%)	80.8 (2.6)	87.0 (3.1)	90.3 (3.3)	**<0.0001**
Activity Counts (counts)	45 373 (2 421)	34 431 (9 406)	29 336 (12 398)	**<0.0001**
Objectively-measured Physical Activity				
VM counts/day ($\times 10^6$)	2.91 (0.45)	2.42 (0.75)	2.19 (0.65)	**0.0001**
Steps per day	16 388 (3 172)	15 387 (4 728)	13 633 (4 717)	**0.0301**
Conicity Index	1.21 (0.11)	1.24 (0.09)	1.23 (0.10)	0.4623
Waist circumference (cm)	88.4 (13.3)	89.2 (14.5)	89.9 (17.0)	0.7247
Body mass index (kg/m^2)	28.7 (6.7)	27.0 (7.1)	28.3 (8.4)	0.8520
MetS z-Score	-0.12 (2.28)	0.02 (2.75)	0.013 (2.76)	0.8569
HOMA-IR [c]	1.79 (2.55)	1.10 (2.18)	1.40 (2.60)	**0.0452**

Data reported as mean (SD), [a] % (n), [b] median (IQR), [c] geometric mean (SD), *HIV* Human Immunodeficiency Virus, *HOMA-IR* Homeostasis Model Assessment of Insulin Resistance, *MetS* Metabolic Syndrome, *SES* Socio-Economic Status, *VM* Vector Magnitude

setting. The main findings of this analysis were first that increasing sleep duration was not characterised by fragmented, lower quality sleep, when assessed using actigraphy-measured sleep quality and duration indices. Second, one specific sleep category (short, fragmented sleep) appears to confer poor cardiometabolic health in this rural, African setting.

Short, fragmented sleep has been described in a rural Malagasy community (Samson et al. 2017), while lower quality sleep has been reported in a rural Mozambican community (Beale et al. 2017). We found a similar total sleep time in the non-exposed group, but higher sleep efficiency and lower sleep disturbances compared to Mozambican groups (Beale et al. 2017). In contrast, the acutely- and chronically-exposed groups had total sleep times comparable to a rural Malagasy community (Samson et al. 2017), but similar sleep efficiency to Mozambican data (Beale et al. 2017).

Generally, our findings are in agreement that unhealthy sleep, whether sub-optimal sleep duration or fragmented sleep, is related to poor cardiometabolic health (Rangaraj and Knutson 2016; Reutrakul and Cauter 2018). Previously, we have reported significant, independent associations between objectively-measured sleep quantity and quality measures and insulin resistance (Cook et al. 2021), and significant, independent, inverse relationships between adiposity and sleep duration in this community (Mashinya et al. 2018; Cook et al. 2020). Actigraphy-measured free-living sleep patterns (short, fragmented sleep) are related to subclinical atherosclerosis in an urban European sample (Domínguez et al. 2019). In contrast, short sleep is negatively associated with 10-year cardiovascular disease risk in a predominantly rural

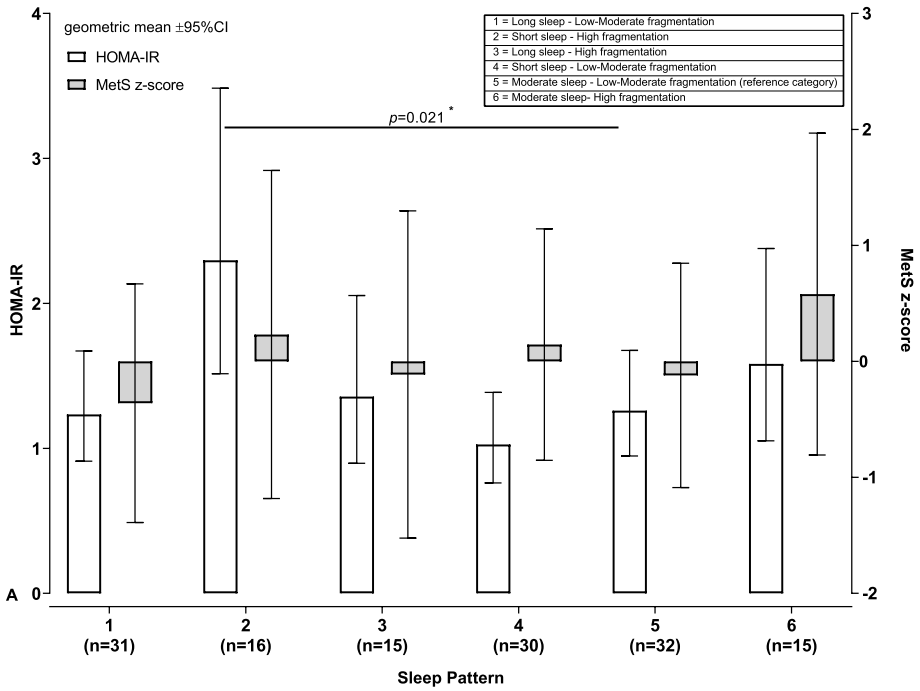

Fig. 1 Cardiometabolic profiles across categories of accelerometry-derived sleep patterns. Cardiometabolic profile across sleep time and sleep fragmentation patterns (HOMA-IR [$p < 0.0001$, adjusted $R^2 = 0.2393$]: age, sex, Conicity Index, socio-economic status, fruit and vegetable intake, sugar-sweetened beverages, alcohol and tobacco usage, Vector Magnitude, HIV status; MetS z-score [$p = 0.4538$, adjusted $R^2 = 0.0009$]: age, sex, socio-economic status, fruit and vegetable intake, sugar-sweetened beverages, alcohol and tobacco usage, Vector Magnitude, HIV status). Sleep Pattern versus Sex interaction: $p \geq 0.5702$. The horizontal lines between the sleep pattern categories indicate significant post hoc differences between categories at either end of the line. * Comparison with reference category (Moderate Sleep – Low-moderate AC)

African setting (Cole et al. 2017). In agreement with our findings, sleep duration is not related to MetS in African participants using self-report (Rae et al. 2020) or objective measures of sleep (Cole et al. 2017). In keeping with our finding of increased HOMA-IR in short- fragmented sleep, glycosylated haemoglobin is higher in a multiethnic sample of actigraphy-measured short-sleepers (Whitaker et al. 2018). HOMA-IR is lower in self-reported short-sleepers and higher in long-sleepers ($p < 0.05$), in urban South African women – this relationship held for black women only (Rae et al. 2018).

In agreement with our finding of a significant association between the increasing level of exposure to short, fragmented sleep and elevated blood pressure, shorter (4–7 h), poor quality self-reported sleep is associated with hypertension ($p < 0.05$) in a rural South African cohort (Gomez-Olive et al. 2018). Similarly, actigraphy-measured short sleep duration is associated with a higher nocturnal blood pressure, while lower sleep efficiency is associated with higher average and temporary diurnal systolic blood pressure, in an urban, industrialised setting (Doyle et al. 2019). In contrast, higher resting blood pressure is significantly related to longer sleep time in urban African females ($p < 0.05$) (Pretorius et al. 2015).

It is likely that measurement modalities (self-report vs. actigraphy) and variations in socio-ecological factors (Grandner et al. 2010) are mediating the relationship between sleep and health, such that discrepant findings between sleep and health, specifically within the South African setting, are characterising the current state of knowledge.

We found that usual short, fragmented sleep was associated with higher HOMA-IR, while exposure to one bout or more of short, fragmented sleep increased the likelihood of elevated resting blood pressure, but not the likelihood of increased insulin resistance or MetS. The significantly higher insulin resistance in the average short, fragmented sleep group, and the non-significant relationship between HOMA-IR level, and those participants experiencing at least one or more bouts of short, fragmented sleep, might reflect long-term sleep behaviours and resultant metabolic adaptations in the average sleep profile group (Nedeltcheva et al. 2009). However, acute exposure to fragmented sleep can result in changes in glucose metabolism (Stamatakis and Punjabi 2010).

Contrary to suggestions that the self-reported long sleep is the result of fragmented sleep (Rae et al. 2020, 2018), we found that increasing age, an increased

people-to-bedroom density, a higher SES and reduced total PA were significant, independent predictors of total sleep time and exposure to short, fragmented sleep. Surprisingly alcohol and tobacco use was predictive of less exposure to short, fragmented sleep. Heavy drinking has been shown to be associated with better perceived sleep in an industrialised setting (Grandner et al. 2015). In keeping with our results of poor sleep quantity and quality associated with increasing PA, poor sleep quality (nocturnal activity) is associated with greater levels of PA during the day in a rural Mozambican setting (Beale et al. 2017). High levels of PA might result in musculoskeletal pain (Beale et al. 2017) or excessive fatigue that disrupts sleep.

Our bedroom density variable ranged from 0.29 to 5.00 people-to-bedroom, and was directly related to sleep time. In particular, quintile 3 and 4 (values ranging from 1.34 to 2.33) were highly related to total sleep time (OR = 15.12 to 16.92, $p \leq 0.001$). Household size was the same as other rural South African settings (Gomez-Olive et al. 2018) (Dikgale: 5.1 ± 26 people; Agincourt: 5.3 ± 3.3 people), but higher than urban, industrialized settings (3.2 ± 1.6 people) (Grandner et al. xxxx) and lower than Mozambican communities (5.8 to 6.0 people) (Beale et al. 2017). People-to-room density for females was nearly identical to obese females from an urban South African setting (≈1 person) (Rae et al. 2018). However, compared to urban and rural Mozambican communities, the people-to-bedroom density is substantially lower (Dikgale: 0.98 people; Mozambique: 2.2 to 3.0 people) (Beale et al. 2017). Increasing people per dwelling or room is associated with fragmented sleep in both North American (Grandner et al. 2015) and sub-Saharan (Beale et al. 2017) settings, but not in some rural communities (Smit et al. 2019). These divergent findings suggest that "crowding" may have different contexts and relative optimum levels, such that it can be conducive to sleep in certain communities while be deleterious to sleep in other communities.

In agreement with our finding of a reduced likelihood of exposure to short, fragmented sleep with increasing socio-economic status, adult urban African Americans (21–95 years) with a low socio-economic position are more likely to experience poor sleep quality, but long sleep duration ($p < 0.01$) (Johnson et al. 2016). In contrast, rural Africans (> 50 years) from higher income quintiles tend to have shorter sleep (Gomez-Olive et al. 2018), and multi-ethnic urban adults (≥ 18 years) with an increased income tend to have an increase in perceived insufficient sleep (Grandner et al. 2015). The more disturbed sleep in adult, rural Mozambicans is associated with aspects such as the type of bed used for sleeping (woven mats on the floor) ($p = 0.042$) (Beale et al. 2017). Greater nocturnal noise due to farming animals and livestock being kept in the house or enclosed in the backyard is also a factor (Beale et al. 2017). It is likely that a higher socio-economic status in our rural sample reflect environmental, neighbourhood and household conditions which are conducive to better quality sleep. Further investigation in this area is required.

To date, studies investigating the interaction of sleep quality/duration and cardiometabolic health have used questionnaire-based sleep assessments which do not rely on correlated algorithmic rules to determine sleep quality and quantity (Lu et al. 2020, 2015; Ji et al. 2020; Lou et al. 2014), or have conducted separate analyses of actigraphy-measured sleep quantity and quality measures (Domínguez et al. 2019). Poor sleep quality in combination with short or long sleep has an additive effect of increasing the risk for stroke (Ji et al. 2020), impaired fasting glucose (Lou et al. 2014), and the prevalence of MetS (Lu et al. 2020). Similarly, the combination of short sleep and poor sleep quality additively increases the prevalence of hypertension (Lu et al. 2015), and the prevalence of impaired fasting glucose (Lou et al. 2014). Our results are in general agreement in that one sleep pattern (short, fragmented sleep) is associated with an increased disturbance in glucose metabolism and hypertension. However, our analysis did not consider whether the effects of poor sleep quality fragmented sleep duration are additive.

Conclusions

In conclusion, in this community setting long sleep duration was not associated with increased sleep fragmentation, but was associated with age, the amount of daytime physical activity and people-to-bedroom density. In contrast to hypothesized long, fragmented sleep, short and fragmented sleep was associated with insulin resistance, and exposure to at least one bout of short, fragmented sleep significantly increased the likelihood of hypertension. Further studies are required to test our findings and to identify and explore the factors underlying short, fragmented sleep in this setting. In keeping with the community outreach imperatives in the Dikgale Health and Demographic Surveillance System site, interventions targeting these factors can be incorporated into community feedback programmes and research initiatives.

Limitations

Due to the small sample size and cross-sectional, convenience sampling in this study, the results cannot be readily generalized, nor can causality be shown.

Abbreviations

AC: Activity Counts; HIV: Human Immunodeficiency Virus; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; JIS: Joint Interim Statement;

MetS: Metabolic Syndrome; PA: Physical Activity; SES: Socio-Economic Status; TST: Total Sleep Time; VM: Vector Magnitude.

Supplementary Information

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Additional file 1. Logistic Models

Additional file 2. Distribution of quantile-defined sleep patterns across days of the week

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Authors' contributions
IC was the principal investigator of the accelerometer data on which this manuscript is based, who initiated the research and obtained funding, co-wrote and edited the research proposal, supervised the data entry, analyzed the data and wrote the first draft manuscript. MM co-wrote and edited the research proposal, collected field data, supervised the field work, performed the data entry, commented on- and contributed to the manuscript. All authors read and approved the final manuscript.

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Availability of data and material
The dataset analysed during the current study is available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Ethics approval for the AWI-Gen Phase 1 survey data and the accelerometer data collection was obtained from the Medunsa Research and Ethics Committee of the University of Limpopo (MREC/HS/195/2014:CR). The participants recruited into the original study were informed about the study objectives, expected outcomes, benefits and the risks associated with it. Written informed consent was obtained from the participants prior to interviews and measurements.

Consent for publication
Not applicable

Competing interests
The authors declare no competing interests.

Author details
[1]Physical Activity Epidemiology Laboratory (EDST), University of Limpopo (Turfloop Campus), Sovenga, Mankweng, Limpopo Province, South Africa. [2]Department of Pathology and Medical Sciences, University of Limpopo (Turfloop Campus), Sovenga, Mankweng, Limpopo Province, South Africa.

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