*Scandinavian Journal of Medicine & Science in Sports*

WILEY

ORIGINAL ARTICLE OPEN ACCESS

# Associations of Wearable Ring Measured Sleep, Sedentary Time, and Physical Activity With Cardiometabolic Health: A Compositional Data Analysis Approach

Maisa Niemelä[1,2] | Anna Maijala[1] | Laura Nauha[1,3,4] | Timo Jämsä[1,5] | Raija Korpelainen[3,4,5] | Vahid Farrahi[1,6]

[1]Research Unit of Health Sciences and Technology, University of Oulu, Oulu, Finland | [2]Centre for Wireless Communications, University of Oulu, Oulu, Finland | [3]Research Unit of Population Health, University of Oulu, Oulu, Finland | [4]Department of Sports and Exercise Medicine, Oulu Deaconess Institute Foundation sr., Oulu, Finland | [5]Medical Research Center Oulu, Oulu University Hospital and University of Oulu, Finland | [6]Institute for Sport and Sport Science, TU Dortmund University, Dortmund, Germany

**Correspondence:** Maisa Niemelä (maisa.niemela@oulu.fi)

**Received:** 28 August 2023 | **Revised:** 15 May 2024 | **Accepted:** 22 July 2024

**Funding:** NFBC1986 33-35y follow-up study received financial support from University of Oulu and Oulu University Hospital (K65760). The study was supported by the Ministry of Education and Culture (Grants OKM/20/626/2022, OKM/76/626/2022, OKM/68/626/2023). The study is also connected to the DigiHealth-project and 6GESS-project, strategic profiling projects at the University of Oulu, which are supported by the Academy of Finland (project numbers DigiHealth 326291, 6GESS 336449) and the University of Oulu.

**Keywords:** activity composition | adulthood | cardiovascular health | movement behavior

## ABSTRACT

Movement behaviors within the 24-h day, including physical activity (PA), sedentary time, and sleep, are associated with cardiometabolic health. We aimed to determine the association between 24-h movement composition and cardiometabolic health while accounting for sleep efficiency. Altogether, 1134 participants from the Northern Finland Birth Cohort 1986 study, free from prior cardiovascular disease, provided at least 4 days of 24-h activity and sleep efficiency measured with a wearable ring. Participants' body composition was assessed with bioimpedance, blood pressure, and waist circumference were measured, and lipids and glucose were analyzed from a fasting blood sample. Linear regression models for cardiometabolic outcomes were created with 24-h movement composition and covariates, including sleep efficiency and behavioral and socioeconomic factors. Isotemporal time reallocations were used to demonstrate the dose-dependent associations between time use and outcomes. Beneficial associations with the outcomes were detected when sedentary time was reallocated to light PA, moderate-to-vigorous PA (MVPA), or sleep. For example, substituting 30 min of sedentary time with MVPA was associated with 7.2% (95% CI from −9.8% to −4.5%) lower visceral fat area, 4.9% (95% CI from −6.5% to −3.3%) lower body fat percentage, 1.6% (95% CI from −2.3% to −0.9%) smaller waist circumference, and 2.4% (95% CI from 1.2% to 3.5%) higher high-density lipoprotein (HDL) cholesterol after accounting for gender, marital status, education level, employment, smoking, alcohol consumption, and sleep efficiency. Substituting sedentary time with sleep or light PA showed beneficial but smaller differences in adiposity measures and HDL cholesterol. Limiting sedentary time should be encouraged in adulthood.

## 1 | Introduction

Poor cardiometabolic health predisposes individuals to cardiovascular disease (CVD), which is the leading cause of death, contributing to around 30% of all deaths worldwide [1]. While the risk of CVD increases with age, increased CVD risk also has been reported in younger populations due to prevalent modifiable risk factors, including obesity, smoking, and physical

inactivity [2]. With adequate lifestyle changes, disease burden could be significantly reduced, and more than three quarters of CVD deaths could be prevented [3].

Movement behaviors, including physical activity, sedentary time, and sleep, are all associated with cardiometabolic health. Physical activity (PA) has beneficial effects on blood pressure, lipid profile, and body composition, all of which are associated with CVD risk [4]. From all the movement behaviors, meeting the minimum recommended level of 150 min/week of moderate-to-vigorous physical activity (MVPA) has been identified as the most significant determinant of CVD risk reduction [4, 5]. Nevertheless, new research suggests that any physical activity, including light PA (LPA) and lower than recommended levels of MVPA, can lead to lower disease risk and better cardiometabolic health indicators, such as lower adiposity and more favorable insulin and triglyceride levels [4, 6–8]. Excessive sedentary time has been linked to poorer cardiometabolic health [9]. In contrast, PA seems to have a protective, modifying role in the association between sedentary time and CVD mortality [10]. In addition, sleep duration and quality have been associated with CVD risk factors in young adults [2, 11]. A too short sleep duration has been linked to detrimental metabolic responses, such as increased insulin resistance, elevated glycated hemoglobin, and increased glucose levels [11]. Both too long (>9 h) and too short sleep (<7 h) have been linked to higher CVD mortality risk in adults [12].

Previous studies have mainly investigated separate associations between one or few movement behaviors, cardiometabolic health, and disease risk and failed to fully account for time spent in all other behaviors [2, 4, 6, 7, 9]. Furthermore, the multicollinearity among the components of 24-h movement behavior necessitates the utilization of appropriate statistical methodologies capable of addressing this inherent challenge [13]. Among the statistical approaches proposed for the study of 24-h time-use, compositional data analysis (CoDa) provides a methodology through which the perfectly multicollinear composition of 24-h time use in movement behaviors can be transformed from the constrained simplex to unconstrained real space when expressed as log ratio coordinates [13–15]. CoDa considers the codependence of movement behaviors and reflects the results as a time exchange between them [16]. It also enables the evaluation of the combined effects of all 24-h movement behaviors in traditional multivariable analyses [14].

Previous studies using CoDa methodology have indicated that replacing sedentary time with MVPA or LPA is related to a more favorable cardiometabolic profile [8, 16–18]. In those studies, the role of sleep duration has been more inconclusive. This may be partly because existing studies have primarily used self-reports to assess sleep duration, and combined it with accelerometer-estimated estimation of waking activities [8, 16–18]. Reallocating time from sleep to MVPA has been beneficially associated with cardiometabolic biomarkers and adiposity [8, 16, 18]. The association between allocating time to other awake behaviors and sleep and health seems to depend on sleep duration; if sleep duration is insufficient, increasing it at the expense of sedentary time (but not PA) is associated with better health outcomes [17].

In addition to sleep duration, poor sleep quality has been associated with increased CVD risk, and research suggests sleep quality should be considered when investigating the association between sleep and cardiometabolic health [19, 20]. For example, Hoevenaar-Blom and colleagues [19] reported a 63% higher risk for incident CVD in adults who had self-reported both poor sleep quality (defined as usually not rising rested) and short sleep duration compared to adults with normal sleep duration and good sleep quality, whereas either condition (short sleep duration or poor sleep quality) alone did not increase the disease risk. Among short sleepers, poor compared to good sleep quality increased the disease risk by 32% [19]. There is no standard single measure to define sleep quality but often information from one or several perceived features such as daytime sleepiness, problems with falling or staying asleep, or perceived sleep quality are inquired [21]. Objective sleep indices measured with polysomnography describing sleep continuity (sleep efficiency, wake after sleep onset, and number of awakenings) have also been used to describe sleep quality [22]. Sleep efficiency describes the percentage of total sleep period spent asleep. Higher sleep efficiency has been shown to decrease systolic blood pressure (SBP) and increase high-density lipoprotein (HDL) cholesterol [23]. Poor sleep efficiency (<80%) has been also linked to higher CVD incidence [24].

This study aims to evaluate the association between 24-h movement behavior composition, including device-based estimates of PA, sedentary time, and sleep in adults, and cardiometabolic health, accounting for device-estimated sleep efficiency.

## 2 | Materials and Methods

### 2.1 | Study Population

Data used in the current study comes from the population-based Northern Finland Birth Cohort 1986 (NFBC1986) study [25, 26]. The NFBC1986 included all newborns from Northern Finland whose birth was expected between July 1, 1985, and June 30, 1986 ($N = 9432$ liveborn). The birth cohort participants have been prospectively followed regularly. The Ethical Committee of the Northern Ostrobothnia Hospital District (108/2017) in Oulu, Finland approved the study. The participants and their parents signed a written informed consent form to participate in the study. Personal identity information was encrypted and replaced with identification codes to provide full anonymity.

At 33 years of age, cohort participants living in the city of Oulu and surrounding areas (250 km from Oulu) were invited to undergo a health examination, complete extensive questionnaires about health and behavior, and participate sleep and PA monitoring ($N = 5717$). Participants in the present study consisted of NFBC1986 members who participated in a health examination ($N = 1807$), provided valid PA and sleep data ($N = 1337$), had all covariate information available, were not pregnant, and were free of prior doctor-diagnosed CVD or diabetes (final sample $N = 1134$).

### 2.2 | Health Examination and Questionnaires

During the clinical measurements, participants' weight, body fat percentage, and visceral fat area (VFA) were measured in

light clothing after overnight fasting with the bioimpedance method (InBody 770, InBody Co., Ltd., Seoul, South Korea). Participants' height was measured without shoes, and waist circumference was measured between the inferior margin of the ribs and the superior border of the iliac crest and rounded to the nearest 0.5 cm. In the laboratory, venous blood samples were drawn after overnight fasting for the analysis of fasting glucose, triglycerides, serum total cholesterol, low-density lipoprotein (LDL), and HDL cholesterol levels. Serum glucose was analyzed using an enzymatic hexokinase/glucose-6-phosphate dehydrogenase method. Triglycerides, serum total cholesterol, and LDL and HDL cholesterol levels were determined using an enzymatic assay method. SBP and diastolic blood pressure (DBP) were measured three times while in a seated position after 15 min of rest (three measurements were averaged; Omron M10, Omron 124 Healthcare, Kyoto, Japan).

The participants completed electronic questionnaires about their background, health, behavior, and work life. From these questionnaires, information was obtained about smoking, alcohol consumption, marital status, employment, and education.

### 2.3 | Measurement of 24-h Movement Behaviors

In the study, the Oura ring generation 2 (Ōura Health Ltd, Oulu, Finland) was used to measure 24-h movement behaviors. The Oura ring is a lightweight and waterproof multisensory tracker that includes photoplethysmography sensors for heart rate and respiration, a temperature coefficient sensor, and a 3D accelerometer. The collected data from the ring was automatically transferred to the cloud server. During the health examination, participants were instructed to wear the ring on any finger of the non-dominant hand (US standard ring sizes from 6 to 13 were available) continuously for 14 days, excluding the sauna. The ring was requested to be worn for two full weeks along with two other wearable monitors. Participants did not receive a ring charger to prevent the possibility of a factory reset, which would have terminated the data collection. According to the manufacturer, the battery life of the fully charged ring is 6 consecutive 24-h periods. In our data collection, a fully charged ring was able to collect data up to 14 consecutive 24-h periods, and the median number of recorded 24-h periods was 6. Participants did not receive any feedback from the ring about their sleep or physical activity.

In a laboratory setting, the Oura ring demonstrated 96% sensitivity (ability to detect sleep) and 48% specificity (ability to detect wakefulness) in evaluation against polysomnography, which is considered the golden standard measurement of sleep [27]. In free living, a strong correlation ($r = 0.86$) in total sleep time between the Oura ring and actigraphy has been reported [28]. In the present study, the sleep period for each night was assessed as the duration of the longest sleep period from the detected start of bedtime to the end of bedtime in minutes. Start and end of bedtimes were recognized with Oura's own algorithms. The multisensory Oura ring combines information from accelerometer and temperature sensor to recognize falling asleep and waking up. During sleep, Oura ring measures heart rate (inter-beat intervals) with photoplethysmography sensors at 250 Hz frequency and combines heart rate data to respiration, temperature, and movement data to compose other sleep variables [29]. A sleep period can include bouts of wakefulness in between the detected start and end of bedtime. We assessed the sleep efficiency as the proportion of the sleep period spent asleep (ratio of total sleep time to sleep period multiplied by 100 to yield a percentage). The average nightly sleep period was calculated for all participants providing at least 4 recorded nights. The average sleep efficiency was calculated to describe the sleep quality.

The Oura ring measures PA using a 3D accelerometer with a sampling frequency of 50 Hz [30]. The ring tracks the number of movements, their type, and intensity in 60-s bouts. These movements are used to estimate daily PA in METs (metabolic equivalent of a task). Sedentary time measured by the Oura ring has been shown to correlate ($r = 0.61$) with sedentary time measured with a wrist-worn ActiGraph monitor (Pensacola, FL, USA) and defined with a cutoff-value of <1853 vector magnitude counts per minute [31]. In a laboratory setting, comparison of measured energy expenditure between Oura ring and indirect calorimetry including wake activities of different intensities from sitting to fast running yielded overall high correlation ($r = 0.93$). In free-living, energy expenditure as METs from Oura ring compared against energy expenditure from accelerometers in different wear-locations (wrist, thigh, and hip) showed strong correlation ($r \geq 0.76$) and significant yet small mean difference in METs (−0.34 to 0.26) [32]. A high correlation has been reported when comparing the Oura ring to the ActiGraph for the measurement of total energy expenditure ($r = 0.85$) and MVPA time ($r = 0.82$) [30]. In the present study, daily durations (min/day) of sedentary time, LPA, and MVPA were determined by Oura's own algorithms for each measured day excluding the first measurement day (wake period until falling asleep) when participants received the ring. The MET thresholds used for differentiating between sedentary time, LPA, MVPA have not been published by the device manufacturer. Daily average durations were calculated for all participants providing at least 4 valid days, defined as at least 600 min/day of wear time of the ring during awake hours. Daily average sedentary time, LPA, and MVPA were combined with average nightly sleep period duration to obtain the 24-h time-use composition.

### 2.4 | Statistical Analysis

The descriptive data are presented in counts, proportions, means, and standard deviations (SDs). Statistical significance was set at 0.05. Compositional data analysis was performed using R version 4.1.2 and "compositions" and "deltacomp" packages. To account for the perfect multicollinearity between 24-h movement behavior variables in the linear regression model, compositional data analysis with isometric log-ratio (ILR) transformations was conducted in accordance with previous literature using the statistical approach for movement behavior data [15]. The compositional means were determined by rescaling the geometric mean of each behavior to total 24 h. For each participant, time spent in sleep, sedentariness, LPA, and MVPA were transformed into ILR coordinates. Using a four-part composition, each behavior was represented by three ILR-coordinates. ILR-coordinate $z1$ represents the relative importance of one behavior (e.g., MVPA)

relative to the geometric mean of the other behaviors (e.g., sleep, sedentary time, and LPA). With orthogonal rotation, pivot ILR-coordinates for all behaviors were obtained. For MVPA, ILR-coordinates are as follows:

$$z=\begin{pmatrix} \sqrt{\frac{3}{4}}\cdot\ln\frac{\text{MVPA}}{(\text{sleep}\cdot\text{sedentary time}\cdot\text{LPA})^{\frac{1}{3}}}, \\ \sqrt{\frac{2}{3}}\cdot\ln\frac{\text{LPA}}{(\text{sleep}\cdot\text{sedentary time})^{\frac{1}{2}}}, \\ \sqrt{\frac{1}{2}}\cdot\ln\frac{\text{sleep}}{\text{sedentary time}} \end{pmatrix} \quad (1)$$

Linear regression models were created for each movement behavior and outcome. All cardiometabolic outcomes were log-transformed to attain normal distribution for linear regression models, and standardized beta values are reported. In each model, three ILR-coordinates were entered as independent variables along with covariates. We considered gender, smoking (yes/no), alcohol consumption (g/day, based on multiple questions about drinking habits), marital status (married or cohabiting/not married or divorced or widowed), employment (employed/not employed), education (no professional education/vocational education/university or polytechnic degree), and sleep efficiency as possible confounders based on previous studies [3, 20, 23, 33]. All covariates were entered into the models. No significant collinearity between covariates was observed (variance inflation factor <5). No significant autocorrelation (Durbin Watson statistic $1.5 < d < 2.5$) or heteroscedasticity based on the distribution and variance of residuals was observed.

Because occupational PA has been shown to associate with increased CVD risk [34], we conducted a sensitivity analysis by excluding participants reporting heavy manual tasks ($n = 138$) or continuous movements or walking ($n = 316$) occurring in their work often or very often. Because the linear regression model coefficients did not change notably, only results for the whole study sample are presented in results. Both too short and too long sleep have been associated with higher risks for CVD mortality [35], and we tested the possibility of a U-shaped relationship between sleep duration and outcomes by creating linear regression models with only sleep duration and each cardiometabolic biomarker with covariates. The models were rerun with a quadratic term for sleep included. If the linear term of sleep was significant but the quadratic term after adding it to the model was not, the association was considered linear. If the linear term was not significant but the quadratic term was significant ($p < 0.10$), the association was considered non-linear [8, 36]. Only triglycerides showed non-linear association which was confirmed to be U-shaped with scatter plot, and the linear model for that outcome was stratified by mean sleep duration (two groups: <8.5 h/day and ≥8.5 h/day). The mean sleep duration in the study population was 8.5 h and fell between the recommended sleep duration for adults (7–9 h per night) [37].

We then theoretically substituted time spent in one behavior with another and explored the association with cardiometabolic health markers. The mean behavior composition was used as a reference, and regression coefficients were predicted on the adjusted linear regression model. The theoretical time reallocations were composed to display the dose-dependent associations between time spent on behaviors and cardiometabolic outcomes. The reallocated time was subtracted from or added to the mean behavior composition, and regression coefficients were predicted again. The difference in the coefficients between the new time composition and the reference composition was calculated, backtransformed, and presented as percentages. Isotemporal plots were constructed to illustrate the difference of theoretically allocating time between the two behaviors had on each cardiometabolic outcome. We used 15-min intervals (representing an approximately 1% time change in the 24-h time use) from 15 to 60 min between other behaviors and from 15 to 30 min for reallocations from or to MVPA to remain below the average MVPA time and illustrate realistic behavior changes.

## 3 | Results

The characteristics of the participants are presented in Table 1. Compared to the cohort members who were excluded (due to missing data in movement composition or covariates or who had prior CVD or diabetes or were pregnant during data collection), the final study sample had on average 0.09 mmol/L lower triglycerides ($p = 0.006$). The sample did not differ from the non-participants in terms of blood pressure, cholesterol, fasting glucose level, body mass index, waist circumference, VFA, body fat percentage, employment or marital status, alcohol consumption, smoking status, educational level, or gender distribution (all $p > 0.05$, data not shown).

### 3.1 | Associations Between 24-h Movement Behaviors and Cardiometabolic Indicators

The average movement behavior composition was 520 min/day for sleep, 525 min/day for sedentary time, 352 min/day for LPA, and 43 min/day for MVPA. Movement composition was significantly associated with all cardiometabolic markers except DBP (univariate model $p = 0.104$). Gender-adjusted linear regression models are presented in Table 2, and all covariate-adjusted models are provided in Table 3. After accounting for movement composition, gender, sleep efficiency, socio-economic status, and drinking and smoking habits, more MVPA time was associated with lower body fat and VFA, a smaller waist circumference, and higher HDL cholesterol, while more sedentary time was associated with the same outcomes in the opposite direction. More sleep time was associated with higher HDL cholesterol and a lower waist circumference (Table 3). More MVPA time was associated with lower LDL cholesterol and more sedentary time with higher fasting glucose, but these associations did not reach statistical significance.

Triglycerides yielded a U-shaped association with sleep duration, and the model was stratified by mean sleep duration (Table 4). When sleep time was less than 8.5 h/day, more MVPA and longer sleep time were associated with lower triglyceride levels, and more sedentary time was associated with a higher triglyceride level.

**TABLE 1** | Characteristics of the study population, $n = 1134$.

| | Women ($n = 687$) | Men ($n = 447$) | All ($n = 1134$) |
|---|---|---|---|
| Age (years old) | 33.8 (0.6) | 33.8 (0.6) | 33.8 (0.6) |
| Height (cm) | 165.3 (5.8) | 179.1 (6.3) | 170.7 (9.0) |
| Weight (kg) | 70.6 (14.9) | 83.6 (13.5) | 75.7 (15.7) |
| BMI ($m/kg^2$) | 25.8 (5.2) | 26.1 (3.8) | 25.9 (4.7) |
| Waist circumference (cm) | 84.7 (12.3) | 92.4 (10.5) | 87.8 (12.2) |
| Body fat (%) | 31.9 (9.0) | 20.7 (7.0) | 27.5 (10.0) |
| Visceral fat area ($cm^2$) | 110.5 (58.1) | 80.0 (43.0) | 98.4 (54.7) |
| Alcohol consumption (g/day) | 1.8 (0.6–4.2) | 1.8 (0.6–4.1) | 1.8 (0.6–4.2) |
| HDL cholesterol (mmol/L) | 1.6 (0.3) | 1.4 (0.3) | 1.5 (0.3) |
| LDL cholesterol (mmol/L) | 2.5 (0.7) | 3.0 (0.8) | 2.7 (0.8) |
| Triglycerides (mmol/L) | 0.8 (0.4) | 1.1 (0.7) | 0.9 (0.6) |
| Systolic BP (mmHg) | 106.7 (10.6) | 119.7 (10.9) | 111.8 (12.5) |
| Diastolic BP (mmHg) | 73.2 (8.7) | 75.9 (8.1) | 74.2 (8.6) |
| Fasting glucose (mmol/L) | 4.9 (0.4) | 5.2 (0.4) | 5.0 (0.4) |
| Sleep efficiency (%) | 88.1 (4.3) | 86.0 (4.6) | 87.3 (4.5) |
| Smoker, *n* (%) | 188 (27) | 193 (43) | 381 (34) |
| Married/cohabiting, *n* (%) | 551 (80) | 348 (78) | 899 (79) |
| Employed, *n* (%) | 431 (65) | 368 (86) | 799 (73) |
| Education, *n* (%) | | | |
| No professional education | 43 (6) | 45 (10) | 88 (8) |
| Vocational/college level education | 207 (30) | 175 (39) | 382 (34) |
| Polytechnic/university degree | 437 (64) | 226 (51) | 663 (58) |

*Note:* Values are mean (standard deviation), median (25th–75th percentile), or count (%).
Abbreviations: BMI = body mass index, BP = blood pressure, HDL = high-density lipoprotein, LDL = low-density lipoprotein.

### 3.2 | Estimated Theoretical Effects of Time Reallocations Between 24-h Movement Behaviors

Time reallocation between MVPA and any other behavior was associated in most cases with the largest differences in cardiometabolic outcomes, of which the most significant were for adiposity measures. Time reallocation between all movement behaviors and their association with body fat percentage are presented in Figure 1. Figures S1–S5 illustrate other outcomes (HDL cholesterol, waist circumference, VFA, and triglycerides). No significant differences occurred in the levels of systolic blood pressure, fasting glucose, or LDL cholesterol from time reallocations between any two behaviors.

#### 3.2.1 | Adiposity

Reallocating time from any other behavior to MVPA was associated with lower adiposity. For example, increasing MVPA by 30 min at the expense of sedentary time was associated with 4.9% (95% CI from −6.5% to −3.3%) lower body fat percentage (Figure 1), 1.6% (95% CI from −2.3% to −0.9%) smaller waist circumference (Figure S2), and 7.2% (95% CI from −9.8% to −4.5%) lower VFA (Figure S3). Opposite time reallocation, 30 min from MVPA to sedentary time, revealed a stronger reverse association: 10.5% (95% CI 6.4%–14.8%) higher body fat percentage, 2.9% (95% CI 1.4%–4.5%) larger waist circumference, and 15.7% (95% CI 8.6%–23.3%) higher VFA. A similar trend with smaller beneficial differences in adiposity measures was seen if MVPA was increased at the expense of sleep or LPA and vice versa if MVPA time was reallocated to sleep or LPA; however, reallocating time from sleep to MVPA was not associated with a smaller waist circumference.

Also, reallocating time from sedentary time to LPA associated with lower adiposity level, but with smaller effect size; for example, substituting 30 min of sedentary time with LPA associated with 1.4% (95% CI from −2.1% to −0.7%) smaller body fat percentage (Figure 1), 0.5% (95% CI from −0.8% to −0.2%) smaller waist circumference (Figure S2), and 2.3% (95% CI from −3.5% to −1.2%) lower VFA (Figure S3). Opposite time reallocation from LPA to sedentary time had symmetrical detrimental association with adiposity measures.

**TABLE 2** | Gender-adjusted linear regression models for movement composition and cardiometabolic outcomes, $R^2$, and standardized beta values for each model are presented.

| | | | Sleep | | Sedentary time | | LPA | | MVPA | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Outcome** | **Model $R^2$** | **Model *p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** |
| HDL cholesterol | 0.112 (adj 0.109) | <0.001 | **0.09 (0.01–0.18)** | 0.035 | **−0.18 (−0.28 to −0.09)** | <0.001 | −0.03 (−0.09 to 0.03) | 0.363 | **0.11 (0.05–0.17)** | <0.001 |
| LDL cholesterol | 0.097 (adj. 0.093) | <0.001 | 0.08 (−0.08 to 0.10) | 0.859 | 0.03 (−0.07 to 0.13) | 0.562 | 0.01 (−0.05 to 0.07) | 0.745 | **−0.09 (−0.14 to −0.03)** | 0.004 |
| Systolic BP | 0.271 (adj. 0.269) | <0.001 | 0.01 (−0.07 to 0.09) | 0.791 | −0.08 (−0.17 to 0.01) | 0.079 | 0.05 (−0.01 to 0.10) | 0.116 | 0.01 (−0.04 to 0.07) | 0.589 |
| Waist circumference | 0.141 (adj. 0.138) | <0.001 | **−0.12 (−0.21 to −0.04)** | 0.004 | **0.24 (0.14–0.33)** | <0.001 | 0.03 (−0.04 to 0.09) | 0.417 | **−0.11 (−0.16 to −0.05)** | <0.001 |
| Body fat | 0.358 (adj. 0.356) | <0.001 | −0.02 (−0.10 to 0.05) | 0.554 | **0.16 (0.08–0.25)** | <0.001 | −0.04 (−0.10 to 0.01) | 0.119 | **−0.13 (−0.18 to −0.08)** | <0.001 |
| Visceral fat area | 0.119 (adj. 0.116) | <0.001 | −0.02 (−0.11 to 0.06) | 0.588 | **0.18 (0.08–0.28)** | <0.001 | −0.05 (−0.11 to 0.02) | 0.135 | **−0.13 (−0.19 to −0.07)** | <0.001 |
| Fasting glucose | 0.142 (adj. 0.139) | <0.001 | −0.03 (−0.11 to 0.06) | 0.502 | 0.05 (−0.04 to 0.15) | 0.269 | 0.03 (−0.04 to 0.09) | 0.411 | **−0.06 (−0.12 to 0.00)** | 0.038 |

*Note:* All outcomes are log-transformed. The models are accounted for gender. Significant associations ($p < 0.05$) are bolded.
Abbreviations: BP = blood pressure, HDL = high-density lipoprotein, LDL = low-density lipoprotein, LPA = light physical activity, MVPA = moderate-to-vigorous physical activity.

**TABLE 3** | Adjusted linear regression models for movement composition and cardiometabolic outcomes. $R^2$ and standardized beta values with a 95% confidence interval for each model are presented.

| | | | Sleep | | Sedentary time | | LPA | | MVPA | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Outcome** | **model $R^2$** | **model *p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** | **β (95% CI)** | ***p*** |
| HDL cholesterol | 0.129 (0.120) | <0.001 | **0.10 (0.01–0.19)** | 0.027 | **−0.20 (−0.31 to −0.10)** | <0.001 | 0.01 (−0.07 to 0.06) | 0.847 | **0.09 (0.03–0.15)** | 0.006 |
| LDL cholesterol | 0.109 (0.100) | <0.001 | −0.02 (−0.11 to 0.07) | 0.718 | 0.07 (−0.03 to 0.18) | 0.157 | 0.01 (−0.08 to 0.06) | 0.789 | −0.06 (−0.12 to 0.00) | 0.059 |
| Systolic BP | 0.286 (0.279) | <0.001 | 0.01 (−0.07 to 0.09) | 0.839 | −0.06 (−0.16 to 0.03) | 0.170 | 0.04 (−0.02 to 0.10) | 0.220 | 0.01 (−0.04 to 0.07) | 0.688 |
| Waist circumference | 0.161 (0.152) | <0.001 | **−0.14 (−0.23 to −0.06)** | 0.001 | **0.27 (0.17–0.37)** | <0.001 | 0.01 (−0.05 to 0.08) | 0.699 | **−0.09 (−0.15 to −0.03)** | 0.003 |
| Body fat | 0.370 (0.364) | <0.001 | −0.03 (−0.10 to 0.05) | 0.458 | **0.17 (0.09–0.26)** | <0.001 | −0.04 (−0.10 to 0.01) | 0.139 | **−0.12 (−0.17 to −0.07)** | <0.001 |
| Visceral fat area | 0.141 (0.132) | <0.001 | −0.03 (−0.12 to 0.05) | 0.437 | **0.19 (0.09–0.30)** | <0.001 | −0.05 (−0.12 to 0.01) | 0.120 | **−0.12 (−0.18 to −0.06)** | <0.001 |
| Fasting glucose | 0.157 (0.148) | <0.001 | −0.04 (−0.13 to 0.05) | 0.365 | 0.09 (−0.01 to 0.19) | 0.077 | 0.00 (−0.06 to 0.07) | 0.919 | −0.05 (−0.11 to 0.01) | 0.109 |

*Note:* All outcomes are log-transformed. The models are accounted for gender, marital status, education level, employment, smoking, alcohol consumption, and sleep efficiency. Significant associations ($p < 0.05$) are bolded.
Abbreviations: BP = blood pressure, HDL = high-density lipoprotein, LDL = low-density lipoprotein, LPA = light physical activity, MVPA = moderate-to-vigorous physical activity.

**TABLE 4** | Linear regression models for movement composition and triglycerides. $R^2$ and standardized beta values with a 95% confidence interval for each model are presented.

| Outcome | Model $R^2$ | Model $p$ | Sleep | | Sedentary time | | LPA | | MVPA | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | $\beta$ (95% CI) | $p$ | $\beta$ (95% CI) | $p$ | $\beta$ (95% CI) | $p$ | $\beta$ (95% CI) | $p$ |
| Triglycerides, sleep <8.5 h[a] | 0.107 | <0.001 | **−0.16 (−0.31 to 0.00)** | 0.048 | **0.31 (0.13–0.50)** | <0.001 | 0.04 (−0.08 to 0.15) | 0.539 | **−0.09 (−0.17 to −0.01)** | 0.028 |
| Triglycerides, sleep <8.5 h[b] | 0.119 (0.102) | <0.001 | **−0.16 (−0.33 to 0.00)** | 0.049 | **0.33 (0.13–0.52)** | <0.001 | 0.03 (−0.09 to 0.16) | 0.576 | **−0.10 (−0.19 to −0.01)** | 0.030 |
| Triglycerides, sleep ≥8.5 h[a] | 0.067 | <0.001 | 0.08 (−0.10 to 0.25) | 0.390 | 0.04 (−0.15 to 0.23) | 0.693 | −0.10 (−0.22 to 0.01) | 0.072 | −0.07 (−0.16 to 0.02) | 0.116 |
| Triglycerides, sleep ≥8.5 h[b] | 0.093 (0.074) | <0.001 | 0.04 (−0.14 to 0.22) | 0.641 | 0.06 (−0.14 to 0.26) | 0.571 | −0.08 (−0.20 to 0.04) | 0.194 | −0.06 (−0.15 to 0.03) | 0.214 |

*Note:* All outcomes are log-transformed. Significant associations ($p < 0.05$) are bolded.
Abbreviations: LPA = light physical activity, MVPA = moderate-to-vigorous physical activity.
[a]The models are accounted for gender.
[b]The models are accounted for gender, marital status, education level, employment, smoking, alcohol consumption, and sleep efficiency. $n = 587$ (participants with <8.5 h sleep) and $n = 547$ (participants with ≥8.5 h sleep).

### 3.2.2 | HDL Cholesterol

Time reallocation from sedentary time or LPA to MVPA was beneficially associated with HDL cholesterol, and a detrimental association was observed with reverse time reallocation from MVPA to sedentary time, LPA, or sleep (Figure S1). For example, substituting 30 min of sedentary time with MVPA was associated with 2.4% (95% CI from 1.2% to 3.5%) higher HDL, and with reverse reallocation from MVPA to sedentary time, 4.2% (95% CI from 1.8% to 6.7%) lower HDL cholesterol.

Also, reallocating time from sedentary time to LPA showed beneficial associations with HDL cholesterol but the effect size was smaller; for example, substituting 30 min of sedentary time with LPA associated with 0.7% (95% CI from 0.2% to 1.1%) higher HDL cholesterol (Figure S1). Opposite time reallocation from LPA to sedentary time had symmetrical detrimental association with HDL cholesterol.

### 3.2.3 | Triglycerides

Reallocating time between any two behaviors yielded no significant differences in triglyceride levels when sleep duration was equal or greater than the mean (8.5h/day) (Figure S5). When sleep duration was less than the mean, reallocating time from sedentary time to sleep, LPA or MVPA was associated with lower triglyceride levels (Figure S4). The greatest difference was observed when 60 min of sedentary time were replaced with sleep (−8.6%, 95% CI from −14.7% to −2.1%). When sedentary time or LPA replaced MVPA or when sedentary time replaced sleep, a negative association of similar magnitude was observed; 60 min reallocated from sleep to sedentary time was associated with a 9.5% (95% CI from 1.9% to 17.7%) higher triglyceride level.

## 4 | Discussion

This population-based study demonstrates that device-estimated 24-h movement composition is significantly associated with cardiometabolic health indicators in the adult population. After considering movement composition, gender, sleep efficiency, socioeconomic status, and drinking and smoking habits, more MVPA was associated with lower adiposity and higher HDL cholesterol. A similar but opposite association was observed for more sedentary time. More sleep was associated with higher HDL cholesterol and, from adiposity measures, a smaller waist circumference.

Pairwise isotemporal time substitutions estimating the percentage difference in each cardiometabolic outcome with 15–60 min (1–4% of 24 h) theoretical time changes in the composition of 24-h movement behaviors indicated a significantly lower adiposity and higher HDL cholesterol when MVPA increased at the expense of other awake behaviors. In line with existing literature [8, 16, 18], decreasing MVPA, especially at the expense of sedentary time, led to larger detrimental differences in the same outcomes. Beneficial differences in adiposity and HDL cholesterol were also seen when LPA replaced sedentary time.

Results obtained in this study concerning awake hours movement composition align with earlier findings on cardiometabolic

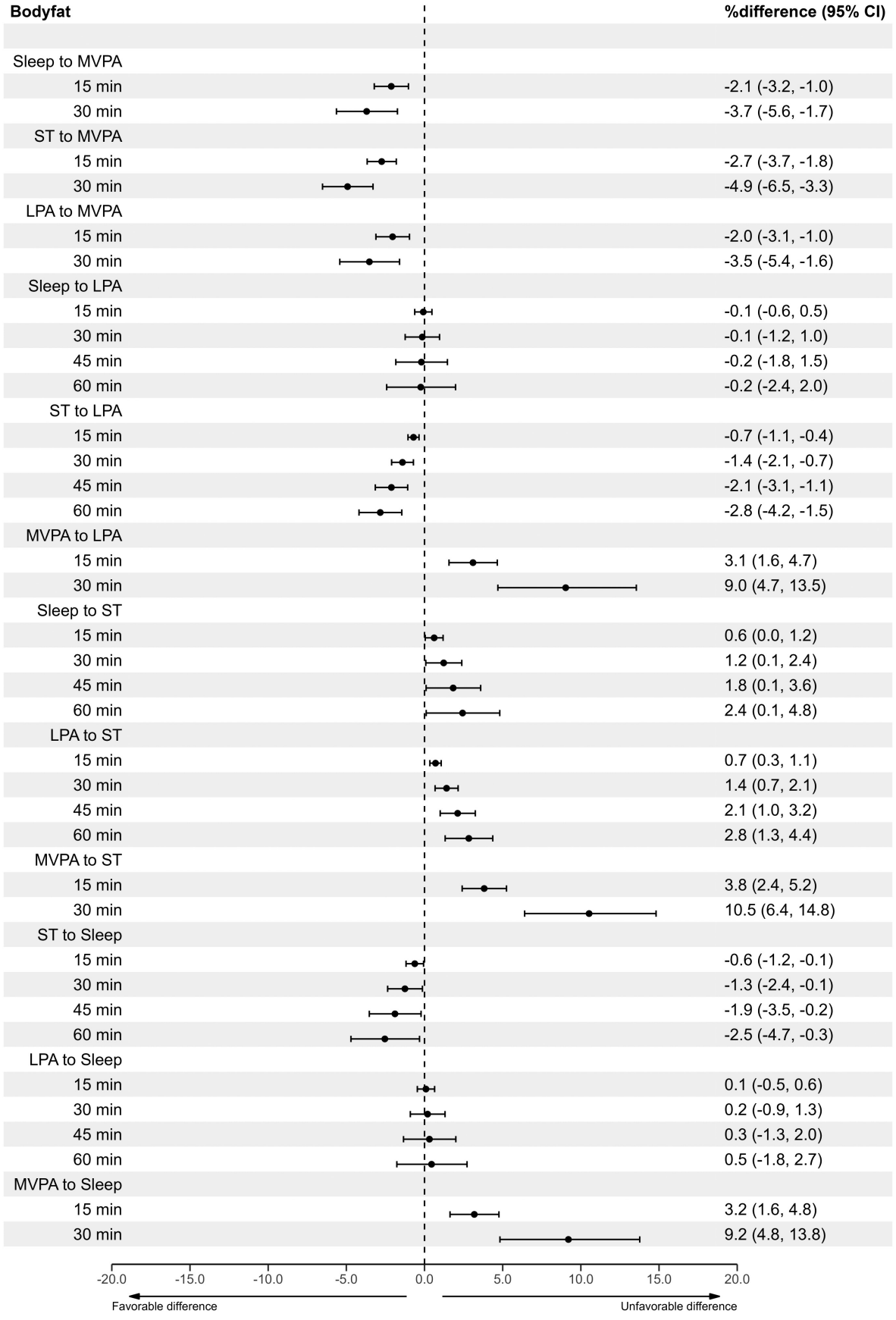


**FIGURE 1** | Forest plot of pairwise time reallocations and association with body fat percentage, difference in percentages, and 95% confidence interval.

health and movement behavior time use [8, 16–18]. For example, beneficial differences of reallocating time to MVPA from sedentary time on cardiometabolic markers and adiposity have been reported widely [17]. Moreover, in one of the first studies utilizing a CoDa approach, Chastin et al. [16] reported beneficial differences in body mass index when MVPA time increased at the expense of sedentary time. Also, beneficial differences in cardiometabolic markers have been reported when LPA time has been increased at the expense of sedentary time [8, 16, 38].

Previous studies have mainly assessed sleep via questionnaires and used proxy measures of time in bed to calculate sleep duration [8, 16, 18, 39], only included awake activities [40] or used wearable thigh sensor recognition of awake hours and expressed sleep duration as the subtracted time between 24 h and awake hours [38]. In addition, none of the above-mentioned studies evaluated sleep quality. Sleep quality is likely to influence the associations between 24-h movement behaviors and health outcomes. For instance, there is evidence from population-based studies suggesting that increased sedentary time is associated with poorer perceived sleep quality and efficiency [41], whereas more time spent in MVPA may contribute to better perceived sleep quality and higher objectively measured sleep efficiency [42].

Our study is the first to examine the associations of device-estimated 24-h movement behaviors with cardiometabolic health, while also accounting for device-estimated sleep efficiency. Due to variety of methods in assessing sleep duration and lack of adjustment for sleep quality in previous studies, direct comparison of our results to earlier research on reallocating time to and from sleep is difficult. Previously, reallocating time from awake behaviors (especially from MVPA or stepping) to time in bed has revealed detrimental or no associations with sugar metabolism, lipids, and adiposity [8, 16, 18, 38]. Small positive differences in blood pressure and C-reactive protein when time was reallocated from awake behaviors to time in bed were reported by Chastin et al. [16] but notably, in their study, most associations were detrimental. We found that replacing sedentary time with sleep was beneficially associated with HDL cholesterol, waist circumference, body fat percentage, VFA, and with triglycerides when sleep time was less than 8.5 h/day. This contradicts the most previous studies showing no positive differences in cardiometabolic health markers with time reallocation from sedentary time to time in bed [8, 18]. Partly in line with our findings, by using a sleep recognition algorithm for accelerometry, Collings and colleagues [43], after accounting for perceived sleep quality, reported positive differences in adiposity among short sleepers (mean sleep 6.6 h/day) when sleep duration increased at the expense of sedentary time. The positive association between time reallocation from sedentary time to sleep and adiposity in our study was not dependent on sleep duration.

Previous research suggests different physiological pathways between short and long sleep durations and cardiometabolic health. Too short sleep effects to immune system and regulation of ghrelin and leptin hormones which can lead to systemic inflammation, imbalanced glycemic control, and increased appetite [11, 12]. Adverse cardiometabolic responses of too long sleep duration have not been explicitly recognized but socioeconomical factors like unemployment, mental health problems like depression, and presence of undiagnosed diseases have been suggested as possible confounding factors at least partly explaining the association [12]. The absolute thresholds for defining too long or too short sleep have not been established and can be outcome-specific and include individual variation [12, 44]. Even though the average sleep duration in our population was rather high, we found adverse differences in cardiometabolic outcomes when the sleep time was increased at the expense on only MVPA, not other wake activities, when adjusting with employment status, educational level, sleep efficiency, and other confounders. Reallocating time from MVPA to sleep showed detrimental differences especially in visceral fat and HDL cholesterol but also in other outcomes. This suggests that MVPA is the most potent health-enhancing activity for cardiometabolic health at least if sleep duration is already sufficient.

Our results highlight the importance of reaching the minimum recommended level of 150 min/week MVPA. The most detrimental differences in all cardiometabolic health markers occurred when MVPA time was theoretically decreased 30 min/day from the mean (decreased to 13 min of MVPA per day i.e., 91 min/week). Decreasing sedentary time by increasing PA, especially MVPA, or sleep is low-risk strategy and accessible for most adults to maintain cardiometabolic health.

The strengths of the present study include a population-based sample of adults and device-estimated 24-h movement composition. Sleep time was determined from a wearable device with a validated algorithm. To account for confounding, we included many covariates known to be associated with cardiometabolic health. The differences in cardiometabolic outcomes showed clear dose-dependent associations with time-use; the more the time was reallocated between movement behaviors, the higher the differences were obtained.

This study has some limitations. Diet, a known factor in cardiometabolic health, was not considered. Due to the cross-sectional study design, no causal interpretations can be drawn. Additionally, all time reallocations were theoretical, thus not showing actual behavioral changes in time. The Oura ring has earlier shown limited specificity in detecting wake periods during sleep [27] which can indicate lower accuracy of sleep continuity metrics, including sleep efficiency used in this study. Existing literature indicates that occupational PA may not provide the same health benefits as leisure-time physical activity [34]. However, wearable devices currently lack the capability to measure the context of PA behaviors. To address this limitation, we repeated the analysis by excluding participants who reported having heavy manual jobs, but the linear regression results did not change notably. The study sample had a lower level of triglycerides compared to participants excluded which could suggest some selection bias. Yet, we did not detect other significant differences between the study sample and those excluded. The study population was homogenous in terms of age and nationality, which limits the generalizability of the results to more diverse populations.

## 5 | Conclusions

The findings of the present study strengthen the consensus on the beneficial associations of PA and detrimental associations of

sedentary time with cardiometabolic health in adults after accounting for other movement behaviors. In addition, we found that more sleep, measured by a wearable device with a validated algorithm and considering sleep efficiency, is associated with smaller waist circumference and higher HDL cholesterol levels. Increasing PA and sleep time at the expense of sedentary time should be encouraged in adulthood.

## 6 | Perspective

Movement behaviors throughout the day are constrained to 24 h; thus, increasing one behavior leads to an inevitable decrease in one or multiple others. All movement behaviors are associated with cardiometabolic health. The compositional data analysis approach considers the codependency between movement behaviors. When this methodology was utilized in a population-based cohort with ring-measured 24-h movement behaviors in adults, we found an increase in MVPA and sleep at the expense of other behaviors positively associated with cardiometabolic health, namely adiposity and lipid levels, even after accounting for sleep efficiency and health behavioral and demographical confounders. Time reallocations between movement behaviors indicated asymmetrical dose-dependent associations; more pronounced detrimental associations were detected when sedentary time increased at the expense of PA. Maintaining current levels of PA and sleep or increasing them at the expense of sedentary time is recommended in adulthood for better cardiometabolic health.


### Acknowledgments

We thank all cohort members and researchers who have participated in the study. We also wish to acknowledge the work of the NFBC project center.


### Conflicts of Interest

The authors declare no conflicts of interest. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

### Data Availability Statement

NFBC data are available from the University of Oulu, Infrastructure for Population Studies. Permission to use the data can be applied for research purposes via an electronic material request portal. In the use of data, we follow the EU general data protection regulation (679/2016) and the Finnish Data Protection Act. The use of personal data is based on a cohort participant's written informed consent in their latest follow-up study, which may cause limitations to its use. Please, contact the NFBC project center (NFBCprojectcenter@oulu.fi) and visit the cohort website (www.oulu.fi/nfbc) for more information.

### References


1. G. A. Roth, D. Abate, K. H. Abate, et al., "Global, Regional, and National Age-Sex-Specific Mortality for 282 Causes of Death in 195 Countries and Territories, 1980–2017: A Systematic Analysis for the Global Burden of Disease Study 2017," *Lancet* 392, no. 10159 (2018): 1736–1788, https://doi.org/10.1016/S0140-6736(18)32203-7.

2. M. Fernström, U. Fernberg, and A. Hurtig-Wennlöf, "The Importance of Cardiorespiratory Fitness and Sleep Duration in Early CVD Prevention: BMI, Resting Heart Rate and Questions About Sleep Patterns are Suggested in Risk Assessment of Young Adults, 18–25 Years," *BMC Public Health* 20, no. 1 (2020): 1715, https://doi.org/10.1186/s12889-020-09801-3.

3. J. Perk, G. de Backer, H. Gohlke, et al., "European Guidelines on Cardiovascular Disease Prevention in Clinical Practice (Version 2012): The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (Constituted by Representatives of Nine Societies and by Invited Experts)," *European Heart Journal* 33, no. 13 (2012): 1635–1701, https://doi.org/10.1093/eurheartj/ehs092.

4. M. Hamer and E. Stamatakis, "Physical Activity and Risk of Cardiovascular Disease Events: Inflammatory and Metabolic Mechanisms," *Medicine and Science in Sports and Exercise* 41, no. 6 (2009): 1206–1211, https://doi.org/10.1249/MSS.0b013e3181971247.

5. W. E. Kraus, K. E. Powell, W. L. Haskell, et al., "Physical Activity, All-Cause and Cardiovascular Mortality, and Cardiovascular Disease," *Medicine and Science in Sports and Exercise* 51, no. 6 (2019): 1270–1281, https://doi.org/10.1249/MSS.0000000000001939.

6. A. Z. LaCroix, J. Bellettiere, E. Rillamas-Sun, et al., "Association of Light Physical Activity Measured by Accelerometry and Incidence of Coronary Heart Disease and Cardiovascular Disease in Older Women," *JAMA Network Open* 2, no. 3 (2019): e190419, https://doi.org/10.1001/jamanetworkopen.2019.0419.

7. S. Amagasa, M. Machida, N. Fukushima, et al., "Is Objectively Measured Light-Intensity Physical Activity Associated With Health Outcomes After Adjustment for Moderate-To-Vigorous Physical Activity in Adults? A Systematic Review," *International Journal of Behavioral Nutrition and Physical Activity* 15, no. 1 (2018): 65, https://doi.org/10.1186/s12966-018-0695-z.

8. V. Farrahi, M. Kangas, R. Walmsley, et al., "Compositional Associations of Sleep and Activities Within the 24-h Cycle With Cardiometabolic Health Markers in Adults," *Medicine and Science in Sports and Exercise* 53, no. 2 (2021): 324–332, https://doi.org/10.1249/MSS.0000000000002481.

9. L. A. Brocklebank, C. L. Falconer, A. S. Page, R. Perry, and A. R. Cooper, "Accelerometer-Measured Sedentary Time and Cardiometabolic Biomarkers: A Systematic Review," *Preventive Medicine* 76 (2015): 92–102, https://doi.org/10.1016/j.ypmed.2015.04.013.

10. U. Ekelund, W. J. Brown, J. Steene-Johannessen, et al., "Do the Associations of Sedentary Behaviour With Cardiovascular Disease Mortality and Cancer Mortality Differ by Physical Activity Level? A Systematic Review and Harmonised Meta-Analysis of Data From 850 060 Participants," *British Journal of Sports Medicine* 53, no. 14 (2019): 886–894, https://doi.org/10.1136/bjsports-2017-098963.

11. H. Owens, B. Christian, and B. Polivka, "Sleep Behaviors in Traditional-Age College Students: A State of the Science Review With Implications for Practice," *Journal of the American Association of Nurse Practitioners* 29, no. 11 (2017): 695–703, https://doi.org/10.1002/2327-6924.12520.

12. C. Krittanawong, A. Tunhasiriwet, Z. Wang, et al., "Association Between Short and Long Sleep Durations and Cardiovascular Outcomes: A Systematic Review and Meta-Analysis," *European Heart Journal Acute Cardiovascular Care* 8, no. 8 (2019): 762–770, https://doi.org/10.1177/2048872617741733.

13. J. Aitchison, "The Statistical Analysis of Compositional Data," *Journal of the Royal Statistical Society: Series B: Methodological* 44, no. 2 (1982): 139–160, https://doi.org/10.1111/j.2517-6161.1982.tb01195.x.

14. J. A. Martín-Fernández, J. Daunis-i-Estadella, and G. Mateu-Figueras, "On the Interpretation of Differences Between Groups for Compositional Data. SORT-Statistics and Operations Research," *Transactions* 39, no. 2 (2015): 231–252, https://raco.cat/index.php/SORT/article/view/302261.

15. D. Dumuid, T. E. Stanford, J. A. Martin-Fernández, et al., "Compositional Data Analysis for Physical Activity, Sedentary Time and Sleep

Research," *Statistical Methods in Medical Research* 27, no. 12 (2017): 3726–3738, https://doi.org/10.1177/0962280217710835.

16. S. Chastin, J. Palarea-Albaladejo, M. L. Dontje, and D. A. Skelton, "Combined Effects of Time Spent in Physical Activity, Sedentary Behaviors and Sleep on Obesity and Cardio-Metabolic Health Markers: A Novel Compositional Data Analysis Approach," *PLoS One* 10, no. 10 (2015): e0139984, https://doi.org/10.1371/journal.pone.0139984.

17. I. Janssen, A. E. Clarke, V. Carson, et al., "A Systematic Review of Compositional Data Analysis Studies Examining Associations Between Sleep, Sedentary Behaviour, and Physical Activity With Health Outcomes in Adults," *Applied Physiology, Nutrition, and Metabolism* 45, no. 10 (2020): S248–S257, https://doi.org/10.1139/apnm-2020-0160.

18. K. Suorsa, N. Gupta, T. Leskinen, et al., "Modifications of 24-h Movement Behaviors to Prevent Obesity in Retirement: A Natural Experiment Using Compositional Data Analysis," *International Journal of Obesity* 47, no. 10 (2023): 922–930, https://doi.org/10.1038/s41366-023-01326-0.

19. M. P. Hoevenaar-Blom, A. M. W. Spijkerman, D. Kromhout, J. F. van den Berg, and W. M. M. Verschuren, "Sleep Duration and Sleep Quality in Relation to 12-Year Cardiovascular Disease Incidence: The MORGEN Study," *Sleep* 34, no. 11 (2011): 1487–1492, https://doi.org/10.5665/sleep.1382.

20. L. X. Qian, L. Xudong, D. Han-Bing, et al., "Sleep Quality, Sleep Duration, and the Risk of Coronary Heart Disease: A Prospective Cohort Study With 60,586 Adults," *Journal of Clinical Sleep Medicine* 14, no. 1 (2023): 109–117, https://doi.org/10.5664/jcsm.6894.

21. M. Fabbri, A. Beracci, M. Martoni, D. Meneo, L. Tonetti, and V. Natale, "Measuring Subjective Sleep Quality: A Review," *International Journal of Environmental Research and Public Health* 18, no. 3 (2021): 1082, https://doi.org/10.3390/ijerph18031082.

22. A. D. Krystal and J. D. Edinger, "Measuring Sleep Quality," *Sleep Medicine* 9, no. Suppl 1 (2008): S10–S17, https://doi.org/10.1016/S1389-9457(08)70011-X.

23. S. R. Yiallourou and M. J. Carrington, "Improved Sleep Efficiency is Associated With Reduced Cardio-Metabolic Risk: Findings From the MODERN Trial," *Journal of Sleep Research* 30, no. 6 (2021): e13389, https://doi.org/10.1111/jsr.13389.

24. B. Yan, J. Yang, B. Zhao, Y. Fan, W. Wang, and X. Ma, "Objective Sleep Efficiency Predicts Cardiovascular Disease in a Community Population: The Sleep Heart Health Study," *Journal of the American Heart Association* 10, no. 7 (2021): e016201, https://doi.org/10.1161/JAHA.120.016201.

25. M. R. Järvelin, P. Elliott, I. Kleinschmidt, et al., "Ecological and Individual Predictors of Birthweight in a Northern Finland Birth Cohort 1986," *Paediatric and Perinatal Epidemiology* 11, no. 3 (1997): 298–312, https://doi.org/10.1111/j.1365-3016.1997.tb00007.x.

26. "University of Oulu," Northern Finland Birth Cohort 1986, accessed May 4, 2023, http://urn.fi/urn:nbn:fi:att:f5c10eef-3d25-4bd0-beb8-f2d59df95b8e.

27. M. de Zambotti, L. Rosas, I. M. Colrain, and F. C. Baker, "The Sleep of the Ring: Comparison of the ŌURA Sleep Tracker Against Polysomnography," *Behavioral Sleep Medicine* 17, no. 2 (2019): 124–136, https://doi.org/10.1080/15402002.2017.1300587.

28. M. Asgari Mehrabadi, I. Azimi, F. Sarhaddi, et al., "Sleep Tracking of a Commercially Available Smart Ring and Smartwatch Against Medical-Grade Actigraphy in Everyday Settings: Instrument Validation Study," *JMIR mHealth and uHealth* 8, no. 10 (2020): e20465, https://doi.org/10.2196/20465.

29. H. Kinnunen, A. Rantanen, T. Kenttä, and H. Koskimäki, "Feasible Assessment of Recovery and Cardiovascular Health: Accuracy of Nocturnal HR and HRV Assessed Via Ring PPG in Comparison to Medical Grade ECG," *Physiological Measurement* 41, no. 4 (2020): 04NT01, https://doi.org/10.1088/1361-6579/ab840a.

30. A. Henriksen, F. Svartdal, S. Grimsgaard, G. Hartvigsen, and L. A. Hopstock, "Polar Vantage and Oura Physical Activity and Sleep Trackers: Validation and Comparison Study," *JMIR Formative Research* 6, no. 5 (2022): e27248, https://doi.org/10.2196/27248.

31. H. Niela-Vilen, I. Azimi, K. Suorsa, et al., "Comparison of Oura Smart Ring Against ActiGraph Accelerometer for Measurement of Physical Activity and Sedentary Time in a Free-Living Context," *CIN: Computers, Informatics, Nursing* 40, no. 12 (2022): 856–862, https://doi.org/10.1097/CIN.0000000000000885.

32. E. Kristiansson, J. Fridolfsson, D. Arvidsson, A. Holmäng, M. Börjesson, and U. Andersson-Hall, "Validation of Oura Ring Energy Expenditure and Steps in Laboratory and Free-Living," *BMC Medical Research Methodology* 23, no. 1 (2023): 50, https://doi.org/10.1186/s12874-023-01868-x.

33. A. E. Bauman, J. F. Sallis, D. A. Dzewaltowski, and N. Owen, "Toward a Better Understanding of the Influences on Physical Activity: The Role of Determinants, Correlates, Causal Variables, Mediators, Moderators, and Confounders," *American Journal of Preventive Medicine* 23, no. 2, Supplement (2002): 5–14, https://doi.org/10.1016/s0749-3797(02)00469-5.

34. A. Holtermann, P. Schnohr, B. G. Nordestgaard, and J. L. Marott, "The Physical Activity Paradox in Cardiovascular Disease and All-Cause Mortality: The Contemporary Copenhagen General Population Study With 104 046 Adults," *European Heart Journal* 42, no. 15 (2021): 1499–1511, https://doi.org/10.1093/eurheartj/ehab087.

35. C. Sabanayagam and A. Shankar, "Sleep Duration and Cardiovascular Disease: Results From the National Health Interview Survey," *Sleep* 33, no. 8 (2010): 1037–1042, https://doi.org/10.1093/sleep/33.8.1037.

36. U. Simonsohn, "Two Lines: A Valid Alternative to the Invalid Testing of U-Shaped Relationships With Quadratic Regressions," *Advances in Methods and Practices in Psychological Science* 1, no. 4 (2018): 538–555, https://doi.org/10.1177/2515245918805755.

37. M. Hirshkowitz, K. Whiton, S. M. Albert, et al., "National Sleep Foundation's Sleep Time Duration Recommendations: Methodology and Results Summary," *Sleep Health* 1, no. 1 (2015): 40–43, https://doi.org/10.1016/j.sleh.2014.12.010.

38. C. Powell, L. D. Browne, B. P. Carson, et al., "Use of Compositional Data Analysis to Show Estimated Changes in Cardiometabolic Health by Reallocating Time to Light-Intensity Physical Activity in Older Adults," *Sports Medicine* 50, no. 1 (2020): 205–217, https://doi.org/10.1007/s40279-019-01153-2.

39. D. E. McGregor, V. Carson, J. Palarea-Albaladejo, P. M. Dall, M. S. Tremblay, and S. F. M. Chastin, "Compositional Analysis of the Associations Between 24-h Movement Behaviours and Health Indicators Among Adults and Older Adults From the Canadian Health Measure Survey," *International Journal of Environmental Research and Public Health* 15, no. 8 (2018): 1779, https://doi.org/10.3390/ijerph15081779.

40. G. J. H. Biddle, C. L. Edwardson, J. Henson, et al., "Associations of Physical Behaviours and Behavioural Reallocations With Markers of Metabolic Health: A Compositional Data Analysis," *International Journal of Environmental Research and Public Health* 15, no. 10 (2018): 2280, https://doi.org/10.3390/ijerph15102280.

41. A. Hofman, T. Voortman, M. A. Ikram, and A. I. Luik, "Time Spent in Physical Activity, Sedentary Behavior, and Sleep: Associations With Self-Rated Sleep Quality in Middle-Aged and Older Adults," *Sleep Health* 8, no. 6 (2022): 701–704, https://doi.org/10.1016/j.sleh.2022.06.009.

42. M. A. Kredlow, M. C. Capozzoli, B. A. Hearon, A. W. Calkins, and M. W. Otto, "The Effects of Physical Activity on Sleep: A Meta-Analytic Review," *Journal of Behavioral Medicine* 38, no. 3 (2015): 427–449, https://doi.org/10.1007/s10865-015-9617-6.

43. P. J. Collings, A. Backes, G. A. Aguayo, G. Fagherazzi, L. Malisoux, and Group the OL Study, "Substituting Device-Measured Sedentary Time With Alternative 24-Hour Movement Behaviours: Compositional

Associations With Adiposity and Cardiometabolic Risk in the ORISCAV-LUX 2 Study," *Diabetology and Metabolic Syndrome* 15, no. 1 (2023): 70, https://doi.org/10.1186/s13098-023-01040-x.

44. T. A. Dejenie, M. T. G/Medhin, F. T. Admasu, et al., "Impact of Objectively-Measured Sleep Duration on Cardiometabolic Health: A Systematic Review of Recent Evidence," *Frontiers in Endocrinology* 13 (2022): 13, https://doi.org/10.3389/fendo.2022.1064969.

## Supporting Information

Additional supporting information can be found online in the Supporting Information section.