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Compositional association of 24-h movement behavior with incident major adverse cardiac events and all-cause mortality

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Cardiovascular disease (CVD) causes a high disease burden. Physical activity (PA) reduces CVD morbidity and mortality. We aimed to determine the relationship between the composition of moderate-to-vigorous PA (MVPA), light PA (LPA), sedentary behavior (SB), and sleep during midlife to the incidence of major adverse cardiac events (MACE) and all-cause mortality at a 7-year follow-up. The study population consisted of Northern Finland Birth Cohort 1966 members who participated in the 46-year follow-up in 2012 and were free of MACE ($N = 4147$). Time spent in MVPA, LPA, and SB was determined from accelerometer data. Sleep time was self-reported. Hospital visits and deaths were obtained from national registers. Participants were followed until December 31, 2019, or first MACE occurrence (acute myocardial infarction, unstable angina pectoris, stroke, hospitalization due to heart failure, or death due to CVD), death from another cause, or censoring. Cox proportional hazards model was used to estimate hazard ratios of MACE incidence and all-cause mortality. Isotemporal time reallocations were used to demonstrate the dose–response association between time spent in behaviors and outcome. The 24-h time composition was significantly associated with incident MACE and all-cause mortality. More time in MVPA relative to other behaviors was associated with a lower risk of events. Isotemporal time reallocations indicated that the greatest risk reduction occurred when MVPA replaced sleep. Higher MVPA associates with a reduced risk of incident MACE and all-cause mortality after accounting for the 24-h movement composition and confounders. Regular engagement in MVPA should be encouraged in midlife.

KEYWORDS
cardiovascular disease, compositional data analysis, physical activity, population-based cohort, sedentary time

1 | INTRODUCTION

Cardiovascular disease (CVD) causes over 17-million deaths per year. It is the leading cause of death globally.[1] Cardiovascular health in adults has improved in recent years, but disease incidence keeps increasing due to an aging and growing population.[1,2] Lifestyle factors, such as smoking, physical inactivity, excessive sedentary time, and an unhealthy diet are associated with CVD development. It has been estimated that more than three-quarters of all CVD deaths could be prevented with adequate lifestyle changes.[3]

Achieving recommended levels of physical activity (PA) reduces cardiovascular morbidity and mortality, mainly due to positive effects of PA on CVD risk factors, such as blood pressure, lipid profile, and body composition.[4] Some of these positive effects seem to act via aerobic fitness level, which could be improved or maintained by performing enough PA.[5] Meeting PA recommendations for moderate-to-vigorous physical activity (MVPA) has the most significant influence on CVD risk reduction.[4] Likewise, lower than recommended level of MVPA, compared to total inactivity, exert positive effects on CVD risk factors, such as insulin and triglyceride levels.[4,6,7]

Excessive time spent in sedentary behaviors increases CVD morbidity[8] and mortality in a dose–response manner.[9] In contrast, PA seems to have a protective, mediating role concerning the association between sedentary time and CVD mortality.[10] Sleep duration has also been shown to be related to the risk of CVD; however, results have been controversial. Short sleep duration has been found to increase blood pressure.[11] Additionally, only short[12] or only long[13] or both short and long durations of sleep[14,15] have been associated with an increased risk for CVD or CVD mortality. Nevertheless, for the precise CVD risk assessment, all movement and nonmovement behaviors over the course of a 24-h period should be considered.

Daily movement and nonmovement behaviors are interrelated and codependent. Analyzing them as independent behaviors neglects their codependent nature and may lead to biased findings. Due to finite time, increasing the amount of time spent in one behavior automatically decreases the amount of time spent in another or a combination of other behaviors. Compositional data analysis (CoDa) considers the codependence of different behaviors and reflects the results as a time exchange between two or more behaviors. Previous studies utilizing CoDa have found that replacing sedentary time with MVPA or LPA is associated with decreased all-cause mortality risk.[16] Furthermore, replacing sedentary time with MVPA or LPA seems to be favorable for a person's cardiometabolic risk profile.[17] In a previous pooled analysis, higher time spent in MVPA compared to other behaviors was found to be associated with a lower risk for all-cause mortality, whereas sleep duration was not associated with mortality.[18] Time-allocated associations with health are not always symmetrical, especially concerning MVPA: An increase in MVPA by a certain amount did not reduce the risk of CVD and all-cause mortality to the same extent than similar decrease in MVPA increased the risk.[16]

Many studies utilizing CoDa or similar statistical approaches for estimating CVD and all-cause mortality risk have included only waking behaviors, namely sedentary time, LPA, and MVPA.[16,19–21] The association of sleep duration with all-cause mortality has been inconclusive.[22] Reallocating time from MVPA to sleep was related to increased mortality risk and vice versa.[22] The association of time reallocations of awake behaviors and sleep with mortality was suggested to be dependent on sleep duration; if sleep duration is insufficient, increasing it at the expense of other waking behaviors is associated with decreased mortality risk.[22]

Prior studies have not investigated the association of time composition, including 24-h movement behaviors in their entirety and the incidence of major adverse cardiac events. This population-based study aimed to determine the association of early midlife time composition, including time spent in LPA, MVPA, SB, self-reported sleep duration, and incidence of major adverse cardiac events and all-cause mortality during a 7-year follow-up.

2 | METHODS

2.1 | Study population

Data used in the present study come from a population-based study called the Northern Finland Birth Cohort 1966 (NFBC1966).[23] The NFBC1966 included all newborns from Northern Finland whose birth was expected in 1966 ($N = 12\,058$ live births). Since then, the birth cohort participants were followed prospectively on a regular basis. The Ethical Committee of the Northern Ostrobothnia Hospital District (94/2011) 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. Detailed information about the NFBC1966 has been described elsewhere.[24]

Participants included in the present study consisted of those NFBC1966 members who participated in the most recent data collection during 2012–2014, provided valid PA data, had covariate information available and were free of prior hospital-treated MACE ($N = 4147$). The data collection methods included questionnaires

about health and behavior, clinical measurements, and records of PA for two weeks with a waist-worn accelerometer.

2.2 | Clinical measurements and questionnaires

During the clinical measurements, participants' weight and height were measured, and BMI was calculated as weight (kg) divided by height squared (m^2). In the laboratory, venous blood samples were drawn after overnight fasting for the analysis of triglycerides, serum total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) cholesterol levels, which were determined by using enzymatic assay methods (Advia 1800; Siemens Healthcare Diagnostics Inc.). The samples were analyzed in NordLab Oulu, a testing laboratory (T113) accredited by the Finnish Accreditation Service (EN ISO 15189). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in addition to heart rate three times while in a seated position after 15 min of rest (the latter two measurements were averaged; Omron M10, Omron 124 Healthcare, Kyoto, Japan).

The participants filled in questionnaires about their background, health, behavior, and work life. From these questionnaires, information was obtained about gender, smoking (yes/no), heavy alcohol consumption (men ≥ 40 g/day, women ≥20 g/day, composed from multiple questionnaire items, as a reference, one standard drink contains 12 g of alcohol), marital status (married/unmarried), education (no professional education/vocational or college level/university degree), and employment status (employed/unemployed). Sleep duration was self-reported based on this question: "How many hours do you sleep on average per night?" The participants reporting less than 4 h of sleep were excluded from the analysis ($N = 7$).

2.3 | Measurement of physical activity

Physical activity was measured with a waist-worn accelerometer (Hookie AM20; Traxmeet Ltd). The participants were asked to wear the accelerometer for all waking hours except for any water-based activities for 14 consecutive days. The PA data collection and analysis have been described elsewhere in detail.[25] Raw acceleration data collected at 100 Hz was segmented into 6-s epochs, and mean amplitude deviation (MAD) values were computed.[26,27] From MAD values, monitor non-wear time was detected using a widely used approach for count-based data with the modification of a shorter window size for handling the artifactual acceleration (30 s instead of 2 min).[28] The remaining wear time epochs were cross-referenced with self-reported sleep times (captured with two questions about typical times for going to bed and waking up) to account potential monitor wear during sleep, and all accelerometer data that overlapped with a sleep interval were discarded. Wear time was then classified into sedentary (activities with < 1.5 metabolic equivalents [MET] in sitting or lying posture), standing still (activities with < 1.5 MET in standing posture), LPA (1.5–3.0 MET), or MVPA (≥3 MET) based on MAD values and a recently validated algorithm for posture detection from hip accelerometer data.[27,29] The posture detection algorithm enables further differentiation between standing still and sitting or lying from hip-based raw acceleration data based on constant Earth's gravity vector and upright walking posture, and it has shown good-to-excellent accuracy when compared with thigh-worn posture classification as ground truth under free-living conditions in adults.[29] Standing still was considered as LPA and added to total LPA time. Thus, sedentary time included only time spent sitting or lying down. Time spent in each intensity category (min/day) was calculated, and averages were formed over all valid days. The participants were included in the analyses, if they provided at least four valid days, where valid days are defined as at least 10 h of device wear time.[30] Accelerometry data were analyzed with MATLAB (version R2019b; MathWorks).

2.4 | Morbidity and mortality

Hospital visit information was obtained from the Finnish Care Register for Health Care, including the discharge diagnoses of hospitalized patients. These data were collected from all the hospitals maintained by local authorities, municipal federations and the central government, and from large private hospitals.

New cases of MACE were defined as the first admission or outpatient visit after the end of PA recording due to acute myocardial infarction (ICD-10 codes: I21, I22), unstable angina pectoris (I20), stroke (I63), hospitalization due to heart failure (I50), or CVD death (I00-I99). Information on deaths, including CVD deaths and deaths from all natural causes (A00-R99), was obtained from Statistics Finland. The definitions of incident cases and deaths were based on the principal diagnosis. Participants were followed from the first day after their PA recording until their date of hospital admittance due to MACE, date of death, or censoring at December 31, 2019.

2.5 | Statistical analysis

The descriptive data are presented in counts, proportions, means, and standard deviations (SDs). Statistical significance was set to 0.05. Compositional data analysis was performed using R version 4.1.2 and "compositions" packages. Statistical analyses were performed with R and SPSS (IBM SPSS Statistics 27, IBM Corp., Armonk, USA).

CoDa was performed in accordance with previous literature that have utilized it for movement behavior data.[31,32] The compositional means were determined by rescaling the geometric mean of each behavior to add up to 24 h. For each participant, sleep duration and time spent in SB, LPA, and MVPA were transformed into isometric log-ratio (ilr) coordinates. Using four-part composition, each behavior was represented by three ilr-coordinates: z1, z2, and z3. Ilr-coordinate z1 represents the relative importance of one behavior (e.g., MVPA) relative to the geometric mean of the other behaviors (e.g., SB, LPA, and sleep). For MVPA, ilr-coordinates are as follows:

$$z = \left(\sqrt{\frac{3}{4}} \bullet \ln \frac{\text{MVPA}}{(\text{sleep} \bullet \text{SB} \bullet \text{LPA})^{1/3}}, \sqrt{\frac{2}{3}} \bullet \ln \frac{\text{LPA}}{(\text{sleep} \bullet \text{SB})^{1/2}}, \sqrt{\frac{1}{2}} \bullet \ln \frac{\text{sleep}}{\text{SB}} \right) \quad (1)$$

With orthogonal rotation, pivot ilr-coordinates for other behaviors were obtained. Cox proportional hazards models were conducted to estimate hazard ratios (HRs) of MACE incidence and all-cause mortality with 95% confidence intervals (CIs). Cox proportional hazard models were created for each movement behavior. In each model, three ilr-coordinates were entered as independent variables along with covariates. We considered smoking, heavy alcohol consumption, marital status, education, employment status, BMI ($kg \cdot m^{-2}$), SBP (mmHg), resting heart rate (RHR) (bpm), LDL cholesterol (mmol/L), and glycated hemoglobin (mmol/mol) as possible confounders based on previous studies.[3,33] Only covariates significantly associated with survival were entered into the models. Because biological variables (here BMI, SBP, RHR, LDL cholesterol, and glycated hemoglobin) can mediate the association between activity composition and outcome,[4] we evaluated their mediating role in preliminary analysis using Cox proportional hazards model adjusted with significant covariates and with stepwise approach adding each biological variable separately to the model and calculating the proportional HR difference.[34] We did not notice major mediating effect (each biological variable explained less than 1.6% of the association, data not shown) and only fully adjusted models are presented. Models were stratified by gender to fulfill the proportional hazard assumption. To account for a potential U-shaped association between sleep duration and outcome,[15] we repeated the Cox proportional hazard models stratified with mean sleep duration.

To account potential reverse causation, we conducted two separate sensitivity analyses. We excluded events occurring during first two years of the follow-up ($N = 23$) and then repeated the Cox proportional hazard models. In the second model, we included all events but further adjusted with self-reported conditions which could have affected baseline activity behavior, namely angina (chest pain during exertion within the last 12 months, yes/no) and musculoskeletal pain (affecting PA at work or leisure time within the last 12 months, scale from 0 to 10, 0 = no pain, 10 = worst possible pain, sum of work and leisure time, i.e., total score from 0 to 20).

Isotemporal plots were constructed to explore the associations of pairwise time reallocations among 24-h movement behaviors. Mean behavior composition was used as a reference, and HR was predicted based on the adjusted Cox regression model. To assist meaningful interpretation of results, time reallocation among movement behaviors ranged from 15 min (representing approximately 1% time change in the 24-h time use) to 60 min (representing approximately 4% time change in the 24-h time use). Time reallocation from MVPA to other movement behaviors was constrained to 30-min to remain below the sample mean for MVPA. While in agreement with previous CoDa studies,[16,18] this range for time reallocations would also allow for displaying the dose–response association between time spent in behaviors and HR for MACE and all-cause mortality as well as interstudy comparison.

3 | RESULTS

Altogether 4147 participants were included in the study. Flowchart of the participant inclusion process is presented in Figure 1. Compared to the participants who were excluded due to missing data in one or multiple covariates or prior MACE, the study sample included in the final analyses had on average 1.1 kg/m^2 lower BMI ($p < 0.001$). The groups did not differ in terms of gender distribution, employment status, prevalence of heavy alcohol consumption, SBP, RHR, LDL cholesterol, or glycated hemoglobin (all $p > 0.05$, data not shown). The average follow-up time was 2477 days, during which 86 events occurred. The characteristics of the participants with and without a MACE event or naturally caused death are presented in Table 1. On average, participants provided 13 (SD 2) valid days of

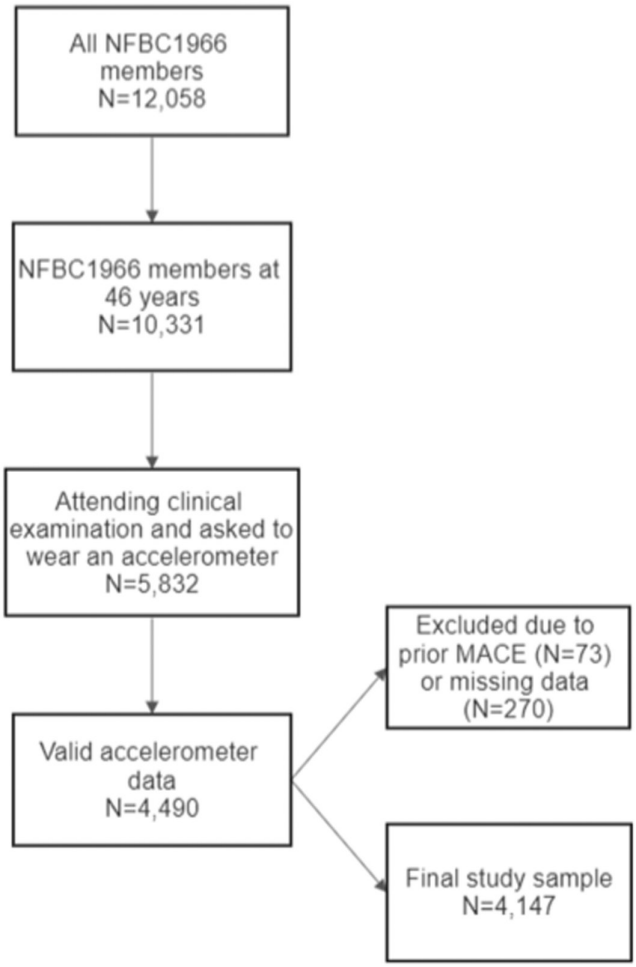

FIGURE 1 Flowchart of the participant inclusion process

accelerometer wear. The composition average for sleep was 517 min/day, sedentary time was 494 min/day, LPA time was 384 min/day, and MVPA time was 45 min/day.

Movement composition was significantly associated with the risk of MACE and all-cause mortality. Cox proportional hazard models showed that more time spent in MVPA relative to other behaviors was associated with a lower risk for MACE and all-cause mortality in the crude model and after adjustments. More time allocated to sleep at the expense of other behaviors was associated with increased risk of MACE and all-cause mortality in the crude model but became nonsignificant after adjustments (Table 2). After stratifying with mean sleep duration (two groups: ≤540 min and >540 min), the association between sleep and outcome became nonsignificant (Table 3). Results were similar after excluding events occurring during first 2 years or after further adjusting for self-reported symptoms (Table S1).

Using the average composition as a reference, we conducted isotemporal time substitutions for up to 60 min between two behaviors. The plots showed that the HRs were associated with each 15-min time increment/decrement between two behaviors (Figure 2). Increasing MVPA time at the expense of sleep (Figure 2, top panel) showed the highest risk reduction of incident MACE and all-cause mortality. Similarly, increasing MVPA time at the expense of sedentary time or LPA was associated with a lower risk for MACE and mortality. Increasing any other behavior at the expense of decreasing MVPA time was related to a highly increased risk of MACE and all-cause mortality. Increasing LPA time at the expense of sleep was associated with a minor decrease in the risk of an event (Figure 2, second top panel). Furthermore, time allocation between LPA and sedentary time was related to a minor decrease in incident MACE and all-cause mortality. Increasing sleep time at the expense of any other behavior was associated with an increased risk but only marginally when this behavior was sedentary time or LPA (Figure 2, bottom panel).

4 | DISCUSSION

This population-based study demonstrated that 24-h movement composition was significantly associated with MACE incidence and all-cause mortality during a 7-year follow-up of a middle-aged population. Higher MVPA was associated with a lower risk of incident MACE and all-cause mortality after accounting for the 24-h movement composition and confounders. Higher sleep duration was associated with increased risk in the univariate model but became nonsignificant after accounting for confounders. Additionally, the 95% confidence intervals had a wide span suggesting a lower precision of the risk estimate and higher variation of study population. Sensitivity analyses showed no significant reverse causation. Pairwise isotemporal time substitutions estimating the risks associated with 1–4% (15–60 min) time change in the composition of 24-h movement behaviors showed a significant reduction in the risk of an event when MVPA time was increased at the expense of any other behavior. However, decreasing MVPA time at the expense of any other behavior led to an even higher risk for incident MACE and all-cause mortality.

Our results are in concordance with previous studies where CoDa highlighted the importance of MVPA in offering protection from adverse cardiac events and premature death.[16–19] Based on our isotemporal time substitutions, maintaining the current level of MVPA or increasing it at the expense of other waking behaviors or sleep seems to be the most beneficial approach for maintaining cardiovascular health. We did not find noticeable changes in the risk of MACE and all-cause mortality when SB was replaced by LPA or vice versa. This finding differs from previous findings where LPA increments at the expense of SB

TABLE 1 Characteristics of the study population ($N = 4147$) according to major adverse cardiac events. Values are the mean (SD) unless otherwise stated.

	Major adverse cardiac event or naturally caused death		
	All ($N = 4147$)	Yes ($n = 86$)	No ($n = 4061$)
Follow-up time, number of days	2477 (273)	1474 (667)	2498 (212)
Female, *n* (%)	2386 (58)	41 (48)	2345 (58)
BMI ($kg \cdot m^{-2}$)	26.7 (4.8)	27.8 (5.1)	26.6 (4.8)
Smoker, *n* (%)	1126 (27)	19 (22)	1107 (28)
Heavy alcohol user, *n* (%)	332 (8)	12 (14)	320 (8)
Married, *n* (%)	3311 (80)	68 (79)	3243 (80)
Education, *n* (%)			
No professional education	106 (3)	4 (5)	102 (3)
Vocational/college level	2701 (67)	58 (72)	2643 (67)
University degree	1211 (30)	19 (24)	1192 (30)
Employed, *n* (%)	3762 (91)	70 (81)	3692 (91)
Systolic blood pressure (mmHg)	125 (16)	125 (16)	132 (18)
Resting HR (bpm)	70 (11)	70 (12)	70 (11)
Low-density lipoprotein cholesterol (mmol/L)	3.44 (0.93)	3.75 (1.02)	3.44 (0.92)
Glycated hemoglobin (mmol/mol)	5.48 (0.51)	5.55 (0.52)	5.48 (0.51)

TABLE 2 Hazard ratios (95% confidence interval), standard errors, and *p*-values for major adverse cardiac events and all-cause mortality using isometric log-ratios for MVPA, LPA, SB, and sleep combinations. Univariate and adjusted Cox regression models.

	Univariate model			Adjusted model[a]		
	HR (95% CI)	SE	*p*-Value	HR (95% CI)	SE	*p*-Value
MVPA vs. other behaviors	**0.46 (0.33–0.63)**	0.16	<0.001	**0.49 (0.34–0.69)**	0.18	<0.001
LPA vs. other behaviors	0.85 (0.37–1.99)	0.43	0.715	0.97 (0.41–2.28)	0.44	0.937
SB vs. other behaviors	0.49 (0.18–1.30)	0.50	0.150	0.53 (0.20–1.40)	0.50	0.201
Sleep vs. other behaviors	**5.25 (1.26–21.88)**	0.73	0.023	4.00 (0.95–16.97)	0.74	0.060

Note: The models were stratified by gender. Significant associations at level $p < 0.05$ are shown in bold.

Abbreviations: CI, confidence interval; HR, hazard ratio; LPA, light physical activity; MVPA, moderate-to-vigorous physical activity; SB, sedentary behavior; SE, standard error.

[a]Adjusted for systolic blood pressure, employment status, resting heart rate, body mass index, low-density lipoprotein cholesterol, glycated hemoglobin, and heavy alcohol consumption.

decreased CVD mortality risk[19] or were positively associated with markers of cardiometabolic health.[35] The age of the study population might partly explain these divergent findings. Dohrn et al.[19] reported that participants who died during follow-up had a mean age of above 74 years with, on average, 22 min/day less MVPA than our study population. Thus, for some portion of the elderly population, activities with moderate or higher intensities might be unrealistic and beneficial effects on cardiovascular health could be seen already with lighter intensities of PA.

Longer sleep duration relative to waking behaviors had a significant detrimental association with MACE and all-cause mortality risk in the univariate model, but the association was of borderline significance after adjustments. After repeating the Cox proportional hazard models with stratified mean sleep duration, the association became nonsignificant, indicating a possible nonlinear relationship. Previous results of the association between sleep duration and CVD risk and mortality have been somewhat inconclusive; however, it seems that both ends of the sleep duration spectrum could contribute to higher mortality risk, with long sleep duration being more detrimental than short.[36]

The strengths of the study include the wide population-based sample of middle-aged adults and comprehensive follow-up data obtained from national registers. Physical

TABLE 3 Hazard ratios (95% confidence interval), standard errors, and *p*-values for major adverse cardiac events and all-cause mortality using isometric log-ratios for combinations of MVPA, LPA, SB, and sleep. Univariate and adjusted Cox regression models stratified with mean sleep duration.

	Univariate model			Adjusted model[a]		
	HR (95% CI)	**SE**	***p*-Value**	**HR (95% CI)**	**SE**	***p*-Value**
MVPA vs. other behaviors	**0.46 (0.33–0.64)**	0.16	<0.001	**0.50 (0.35–0.71)**	0.18	<0.001
LPA vs. other behaviors	1.26 (0.45–3.55)	0.53	0.662	1.48 (0.52–4.20)	0.53	0.460
SB vs. other behaviors	0.79 (0.23–2.75)	0.64	0.713	0.91 (0.26–3.11)	0.63	0.875
Sleep vs. other behaviors	2.17 (0.30–15.70)	1.01	0.442	1.50 (0.21–10.83)	1.01	0.688

Note: The models were stratified by gender and mean sleep duration (two groups: ≤540 min and >540 min). Significant associations at level $p < 0.05$ are shown in bold.

Abbreviations: CI, confidence interval; HR, hazard ratio; LPA, light physical activity; MVPA, moderate-to-vigorous physical activity; SB, sedentary behavior; SE, standard error.

[a]Adjusted for systolic blood pressure, employment status, resting heart rate, body mass index, low-density lipoprotein cholesterol, glycated hemoglobin, and heavy alcohol consumption.

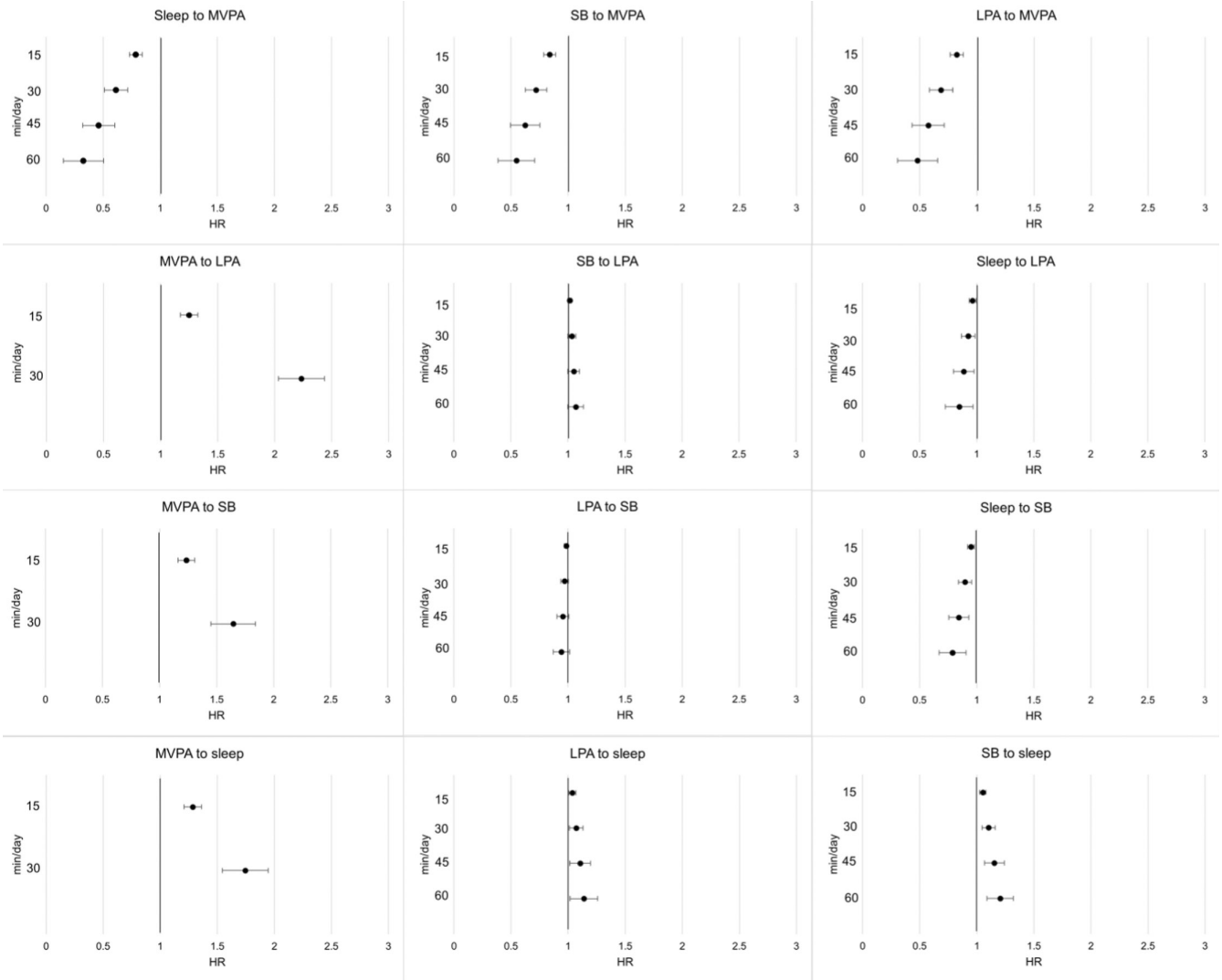

FIGURE 2 Hazard ratios and 95% confidence intervals for MACE incidence and all-cause mortality from time allocating between one and another movement behaviors. The reference behavior is the composite mean (sleep 517 min/day, SB 494 min/day, LPA 384 min/day, and MVPA 45 min/day).

activity was captured with accelerometers worn during all waking hours. Utilizing CoDa with 24-h movement composition considers the full spectrum of movement and nonmovement behaviors, including sleep time, to precisely estimate the effect of time composition on incident MACE and all-cause mortality during the follow-up period. Additionally, we included a wide range of possible confounders associated with the movement behaviors and the outcome.

The study does not come without limitations. Although device-based measurement of daily activities was captured over a relatively long timeframe (14 days) and with raw accelerometry, using cut-points for translating accelerometer data into SB, LPA, and MVPA may be a limitation.[37] Sleep duration was self-reported and could potentially involve reporting bias.[38] Previous CoDa studies assessing cardiovascular outcomes and all-cause mortality have mainly neglected sleep,[16,19–21] and studies providing a device-based measure of sleep have had significantly smaller study samples and cross-sectional study designs.[35,39] Basically, almost all large-scale studies providing a 24-h movement behavior composition have relied on self-reports of sleep.[17,40] On average, the follow-up duration in this study was 6.8 years; however, a longer follow-up would have provided more cases. Nutrition, a risk factor for CVD, was not considered in the study which can be seen as a limitation. Study sample had a lower BMI compared to participants excluded due to prior MACE or missing data, which could suggest some selection bias. However, we did not detect other significant differences between study sample and those excluded.

The mean age of the study participants at the end of the follow-up was 53 years. Thus, they were relatively young, considering that the risk and prevalence of MACE increase later in life. However, similar follow-up durations and age profiles of populations have been reported in other studies.[20,21] For example, similar to our findings, von Rosen et al.[16] reported almost equal hazard ratios for 20-min time allocations between SB and MVPA and all-cause mortality during a 15-year follow-up. In addition, their study population was nearly the same age as ours. Thus, even with a relatively young study population, we found that 24-h movement composition was significantly associated with incident MACE and all-cause mortality. This finding highlights the importance of maintaining or increasing MVPA time in midlife as a preventive measure for cardiac problems and premature death.

5 | CONCLUSIONS

More time in MVPA was associated with reduced risk of incident MACE and all-cause mortality during a 7-year follow-up after accounting for the 24-h movement composition and other potential confounders. Regular engagement in MVPA should be encouraged in midlife, especially in subpopulations at high risk for CVD.

6 | PERSPECTIVE

Due to finite time during a day, increasing the amount of time spent in one activity behavior automatically decreases the amount of time spent in another or a combination of other behaviors. Compositional data analysis considers this codependence of different behaviors. When compositional data analysis was used in population-based cohort study, we found highest reduction in the risk of incident MACE and all-cause mortality at 7-year follow-up when MVPA time was increased at the expense of any other behavior. Time reallocation associations were not symmetrical; decreasing MVPA time was associated with even higher increments in the risk of event. Maintaining the current level or increasing MVPA time is recommended for reducing the risk of MACE and all-cause mortality in midlife.

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CONFLICT OF INTEREST

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

DATA AVAILABILITY STATEMENT

Research data are not shared.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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