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Physical activity patterns and cardiovascular disease risk in adults with overweight or obesity: a prospective cohort study

Kai Mu[1†], Yanan Qiao[2†], Ruilang Lin[3], Yongfu Yu[3], Min Zhao[4], Costan G. Magnussen[5,6,7] and Bo Xi[2*]

Abstract

Background Cardiovascular disease (CVD) is a major global health burden, particularly among overweight and obese individuals. We aimed to investigate associations between different physical activity patterns (concentrated activity vs. regularly active) and incident CVD, and to compare their effects in this specific population.

Methods Data were from the UK Biobank, with 49,368 overweight and obese individuals (63.50 ± 7.73 years; 51.4% women) included in this study from 2006–2010. Participants were categorized into three physical activity patterns: inactive (< 150 min/week of moderate-to-vigorous physical activity (MVPA)), regularly active (≥ 150 min/week of MVPA, with ≥ 50% of activity distributed over > 2 days/week), and concentrated activity (≥ 150 min/week of MVPA, with ≥ 50% concentrated on 1–2 days/week).

Results In fully adjusted models, both the concentrated activity and regularly active patterns had a similar magnitude of CVD risk reduction compared with inactive individuals, overall CVD (concentrated activity: HR [95% CI]: 0.79 [0.74–0.85]; regularly active: 0.76 [0.70–0.83]), myocardial infarction (concentrated activity: 0.77 [0.67–0.88]; regularly active: 0.64 [0.53–0.76]), atrial fibrillation (concentrated activity: 0.81 [0.73–0.89]; regularly active: 0.84 [0.75–0.94]), heart failure (concentrated activity: 0.60 [0.52–0.70]; regularly active: 0.61 [0.50–0.73]), and stroke (concentrated activity: 0.79 [0.66–0.94]; regularly active: 0.81 [0.65–1.00]).

Conclusions Concentrated MVPA over 1–2 days (the concentrated activity pattern) was associated with reduced CVD risk in individuals with overweight or obesity, with a magnitude of risk reduction similar to that observed for regularly distributed activity.

Keywords Physical activity, Cardiovascular diseases, Obesity

†Kai Mu and Yanan Qiao are equal contributors.

*Correspondence:
Bo Xi
xibo2007@126.com
[1] Department of Pediatrics, The Second Qilu Hospital of Shandong University, School of Public Health, Cheeloo College of Medicine, Shandong University, Jinan, Shandong 250012, China
[2] Department of Epidemiology, The Second Qilu Hospital of Shandong University, School of Public Health, Cheeloo College of Medicine, Shandong University, Jinan, Shandong 250012, China
[3] Department of Biostatistics, Key Laboratory of Public Health Safety of Ministry of Education, NHC Key Laboratory for Health Technology Assessment, School of Public Health, Fudan University, Shanghai 200032, China
[4] Department of Nutrition and Food Hygiene, School of Public Health, Qilu Hospital, Shandong University, Jinan, Shandong 250012, China
[5] Baker Heart and Diabetes Institute, Melbourne, VIC, Australia
[6] Research Center of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland
[7] Centre for Population Health Research, University of Turku and Turku University Hospital, Turku, Finland

Background

Cardiovascular disease (CVD) remains a global health challenge [1, 2], accounting for approximately 19.91 million deaths worldwide in 2021 [3]. Elevated body mass index (BMI), an established modifiable risk factor for CVD, demonstrated an etiological association with 3.69 million attributable deaths in the same year, representing a 46.7% increase compared to 2010 [3]. CVD encompasses subtypes, including myocardial infarction (MI), atrial fibrillation (AF), heart failure (HF), and stroke, with higher prevalence among individuals with overweight or obesity [4, 5].

Physical activity (PA) has been well-established as a critical modifiable determinant in reducing or potentially offsetting excess CVD risk in overweight or obese individuals [6, 7]. The World Health Organization, along with clinical guidelines from both the United States and Europe, recommend engagement of adult populations in a minimum of 150 min of moderate-intensity aerobic PA or 75 min of vigorous-intensity PA per week (or an equivalent combination of both) [8, 9]. These guidelines advocate both progressive dose-escalation of aerobic exercise for enhanced health benefits and consistent distribution of PA throughout the week, ideally over 4–5 days or daily [10]. Despite the recommendations and public health efforts to promote PA, approximately three-quarters of adults exhibit suboptimal adherence [9]. A key barrier to guideline adherence is perceived time constraints [9].

One promising behavioral strategy is the concentrated activity approach [11–13] that is, individuals may engage in structured PA regimens, primarily within 1–2 days. Prior analyses of the UK Biobank have shown that concentrated activity pattern confers similar reductions in CVD risk as regularly distributed activity [14, 15]. Similarly, the NHANES data indicate that concentrated activity pattern can lead to comparable improvements in adiposity markers, including waist circumference and BMI [16]. However, these studies largely excluded or underrepresented individuals with overweight or obesity. Consequently, whether concentrated activity pattern is associated with CVD risk specifically in high-risk population remains unexamined. Addressing this evidence gap is critical, as demonstrating the efficacy of concentrated activity in adults with overweight or obesity could inform more inclusive and adherence-friendly public health strategies.

In this study, we examined the longitudinal association of different PA patterns, with a specific focus on the concentrated activity pattern, with the incidence of CVD among overweight and obese individuals. This prospective cohort study used accelerometer-based PA data from the UK Biobank, providing a more objective and precise assessment of PA levels than self-reported data that is prone to recall bias and measurement error [17].

Methods

We adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines for observational research.

Study design and participants

We analyzed data from the UK Biobank, a large prospective cohort comprising 502,629 participants aged 40–73 years recruited from 2006–2010. The UK Biobank initially invited 9.2 million individuals residing within 25 miles of 22 assessment centers across the United Kingdom, achieving a baseline response proportion of 5.4%. For our analysis, we included participants as overweight/obese per WHO criteria (BMI $\geq$ 25 kg/m^2). Ethics approval was granted by the North–West Multicenter Research Ethics Committee (Reference No. R21/NW/0157), with all participants providing written informed consent. The UK Biobank data processing scripts used for the analyses described in this study are publicly available on the website (https://github.com/shaankhurshid/weekend_warrior_phewas).

Outcomes

The primary outcome was a composite of incident fatal and non-fatal CVD, including MI,AF, HF and stroke. Outcomes were ascertained through linkage to national hospital inpatient records databases (Hospital Episode Statistics for England, Patient Episode Database for Wales, Scottish Morbidity Record) and death registrations (NHS Digital for England/Wales, NHS Central Register and National Records of Scotland). Incident cases were identified using ICD-10 codes (Additional file 1: Table S1). The UK Biobank hospital data have shown > 95% accuracy for cardiovascular outcomes [18, 19]. Outcomes were ascertained exclusively from hospital admissions and death certificates but not from primary care records, which ensured consistent definitions across regions and capturing clinically significant events. Follow-up began at the accelerometer assessment and continued until the first CVD diagnosis, death, or November 30, 2022, whichever came first.

Physical activity measurement

Accelerometry data were collected in a subsample (n = 103, 661) who wore a wrist-based Axivity AX3 (Newcastle upon Tyne, UK) triaxial accelerometer for one week. The accelerometers continuously recorded motion data at 100 Hz with a dynamic range of ± 8 g (units of gravity) [18]. Raw acceleration signals underwent gravitational calibration using manufacturer-specified

algorithms and processed to calculate the mean vector magnitude for 5-s intervals (epochs). Valid accelerometer wear-time was defined by two criteria: (1) a minimum of 72 h (3 days) total wear time with representative 24-h sampling, and (2) < 60 consecutive minutes of zero counts, indicating device non-wear. Non-wear periods were identified using a validated algorithm that detects ≥ 60 consecutive minutes with standard deviation < 13 mg across all three axes.

Time spent in different movement behaviors was estimated using a machine-learning algorithm trained on an independent free-living cohort of 152 individuals [19]. The algorithm previously demonstrated a mean accuracy of 0.88 and a Cohen's kappa of 0.80 [19].

Participants were excluded if they had insufficient valid wear time (n = 6,992), inadequate data for non-wear imputation (n = 1,194), or unrealistic acceleration values (n = 13). Unrealistic values were defined as: single-epoch readings exceeding the device's physical range (± 8 g), or acceleration patterns inconsistent with human movement (e.g., ≥ 12 consecutive hours of zero counts despite device active wear timestamps). These criteria were applied to identify probable device malfunction or data corruption rather than true activity patterns. Although PA measured during a single week may not capture all seasonal or long-term behavioral variation, nor individual year-to-year changes in activity habits, this approach represents the standard protocol in accelerometer-based epidemiological studies and has been validated in the UK Biobank study involving over 100,000 participants [20]. Accelerometer-based assessment provides a more objective measure than self-report, which is susceptible to recall bias and social-desirability bias. Moreover, any non-differential measurement error arising from the single-week assessment would generally bias effect estimate toward the null, meaning that the observed reductions in CVD risk likely represent conservative estimates of the true associations.

Covariate selection and definitions

Covariates were selected based on established prior knowledge and previous analyses of concentrated activity pattern using the UK Biobank data [2, 14]. Detailed definitions for all covariates are provided (Additional file 1: Table S2). The following variables were considered: (1) demographic factors: age at the end of accelerometer wear (years), sex (women and men), and race/ethnicity (White and non-White); (2) socioeconomic factors: Townsend deprivation index (TDI, a composite measure of area-level socioeconomic status) [21], education level (college or university degree, advanced level (A level)/Advanced Subsidiary level (AS level) or equivalent, Ordinary level (O level)/General Certificate of Secondary Education (GCSE) or equivalent, Certificate of Secondary Education (CSE) or equivalent, National Vocational Qualification (NVQ) or Higher National Diploma (HND) or Higher National Certificate (HNC) or equivalent, and others), and employment status (employed and unemployed); (3) lifestyle factors: smoking status (never, ever, and current), alcohol drinking (never, ≤ 1–2 times/week, and ≥ 3 times/week), sleep duration (< 7, 7–8, and > 8 h/day), and a healthy diet score (range 0–7 points, based on intake of fruits, vegetables, fish, processed and unprocessed red meats, whole grains, and refined grains) [22]; (4) cardiometabolic factors: body mass index (BMI, kg/m^2), SBP/DBP (mmHg), glycated hemoglobin (HbA1c, mmol/mol), triglycerides (TG, mmol/L), low-density lipoprotein cholesterol (LDL-C, mmol/L), high-density lipoprotein cholesterol (HDL-C, mmol/L), and use of antihypertensive, antihyperglycemic, and lipid-lowering medications (yes and no). Because cardiometabolic factors (including BMI, SBP, DBP, HbA1c, TG, LDL-C, HDL-C, and use of related medications) are considered potential mediators in the pathway between PA patterns and CVD, they were not included in the primary analyses. Instead, these variables were examined in sensitivity analyses to explore potential mechanistic pathways.

Follow-up and censoring

Follow-up commenced post-accelerometer assessment, with entry time defined as the completion of PA measurements via accelerometers and ended at the first occurrence of a CVD event, death, or the last available follow-up date, whichever was first. We excluded those with prevalent CVD ascertained from baseline records. The follow-up duration varied on the basis of data availability, with the final follow-up date being November 30, 2022.

Categorization of physical activity patterns

For the primary analyses, we applied the guideline-recommended threshold of at least 150 min of MVPA per week. Based on accelerometer-derived MVPA, participants were divided into three groups as follows [14]: the inactive group (MVPA < 150 min/week), the regularly active group (MVPA ≥ 150 min/week with ≥ 50% distributed over > 2 days/week), and the concentrated activity group (MVPA ≥ 150 min/week with ≥ 50% concentrated on 1–2 days/week). We also performed sensitivity analyses to assess the robustness of our findings using additional MVPA thresholds to define PA patterns [14], including ≥ 25th percentile (100.8 min/week), ≥ 50th percentile (201.6 min/week) of the total MVPA volume in our study population.

Statistical analysis

Baseline data are presented as mean (SD) for continuous variables and n (%) for categorical variables. Multivariable Cox proportional hazards models were used to estimate associations between PA patterns and CVD outcomes in overweight or obese individuals with results presented as hazard ratios (HRs) and 95% confidence intervals (CIs). Time-to-event was calculated from accelerometer assessment date until the first occurrence of a CVD endpoint, death, or the end of follow-up (November 30, 2022). Separate Cox proportional hazards models were fitted for the composite CVD outcome and for each subtype (MI, AF, HF, stroke). Three hierarchically adjusted models were constructed using the inactive group as reference. Model 1 was adjusted for age, sex, and race/ethnicity. Model 2 was additionally adjusted for the Townsend Deprivation Index, education, and employment status. Model 3 (primary model) was further adjusted for smoking status, alcohol consumption, sleep duration, and diet quality. Missing covariate data were primarily handled using a complete-case (listwise deletion) analysis, with multiple imputation by chained equations (MICE) with random forest implemented as a sensitivity analysis.

Subgroup analyses were conducted according to the following factors: weight status (overweight vs. obesity), age (< 65 vs. ≥ 65 years), sex (female vs. male), smoking status (never vs. ever/current), alcohol consumption (< 1 time/week vs. ≥ 1 time/week), baseline medication use (antihypertensive, antihyperglycemic, lipid-lowering), presence of baseline metabolic conditions (hypertension, diabetes, dyslipidemia, or any combination thereof), and self-reported health status (excellent/good vs. fair/poor).

Pre-specified sensitivity analyses were performed to assess the robustness of the primary findings: (1) exclusion of CVD events that occurred within the first 2 years after accelerometer assessment; (2) adjusting for potential mediators, BMI, SBP, DBP, LDL-C, HDL-C, HbA1c, antihyperglycemic medication, antihypertensive medication, and lipid-lowering medication; (3) reclassifying those with incomplete accelerometer data to inactive. (4) further stratifying the regularly active group into moderately regular (≥ 150 min/week across 3 days) and highly regular (≥ 150 min/week across ≥ 4 days) subgroups; and (5) calculating E-values to quantify the minimum strength of association, an unmeasured confounder would need to have with both the exposure and the outcome to explain away the observed effects. E-values were derived for both point estimates and the lower limits of the 95% confidence interval, providing a transparent metric for assessing robustness to unmeasured confounding. The required strengths of association were compared with those of known confounders reported in the literature.

Statistical analyses were performed via R software version 4.1.3 (R Foundation for Statistical Computing, Vienna, Austria). The statistical significance was set as $P < 0.05$ (two-sided test).

Results

Study population and follow-up

Following exclusion of those with insufficient wear time (n = 6,992), unrealistic acceleration values (n = 13), missing daily physical activity data (n = 1,194), preexisting cardiovascular disease (n = 5,144), and missing information on covariates (n = 1077), 49,368 participants with overweight or obesity were included in analyses (Additional file 1: Fig. S1). Among these, 15,402 (31.2%) were classified as obese, and 33,966 (68.8%) were classified as overweight. Based on the guideline-recommended threshold of ≥ 150 min/week MVPA, 20,110 (40.7%) were classified as concentrated activity, 10,567 (21.4%) as regularly active, and 18,691 (37.9%) as inactive (Table 1). Median follow-up was 7.94 years (IQR 7.37–8.48). Baseline characteristics showed no significant differences between included and excluded participants (Additional file 1: Table S3).

Physical activity patterns and CVD risk

The inactive group demonstrated the highest cumulative incidence of cardiovascular events, whereas both concentrated activity and regularly active groups presented similar risk reductions (Fig. 1). In fully adjusted model (Model 3), both concentrated activity and regularly active patterns were associated with reduced CVD risk compared with inactive individuals (Fig. 2). For the composite CVD, concentrated activity demonstrated a 21% lower risk (HR 0.79, 95% CI 0.74–0.85) and regularly active individuals a 24% lower risk (HR 0.76, 95% CI 0.70–0.83), with substantially overlapping confidence intervals.

For specific cardiovascular outcomes, both patterns demonstrated significant protective associations. The regularly active group showed a 36% risk reduction for MI compared with 23% for the concentrated activity group. For stroke outcome, although the regularly active group did not show a statistically significant association (HR 0.81, 95% CI 0.65–1.00), the concentrated activity pattern was associated with a significant 21% reduction in risk, suggesting that concentrated PA may be particularly relevant for stroke prevention in this population. A direct comparison of the two active patterns (Additional file 1: Table S4) indicated comparable risks for most outcomes, except for myocardial infarction, where concentrated activity showed a trend toward a higher risk (HR 1.19, 95% CI 1.00–1.42, $P = 0.053$).

Table 1 Baseline characteristics of study population stratified by physical activity pattern

Characteristic	Concentrated activity	Regularly active	Inactive group	*P* value	*SMD*
***N* (%)**	20,110 (40.73)	10,567 (21.40)	18,691 (37.86)		
Age (years)	62.22 (7.63)	61.31 (7.81)	63.18 (7.69)	< 0.001	0.161
Sex				< 0.001	0.302
Female	8705 (43.29)	4536 (42.93)	12,136 (64.93)		
Male	11,405 (56.71)	6031 (57.07)	6555 (35.07)		
Ethnicity				< 0.001	0.048
White	19,579 (97.36)	10,152 (96.07)	18,008 (96.35)		
Other	531 (2.64)	415 (3.93)	683 (3.65)		
Townsend deprivation index	−1.91 (2.70)	−1.29 (3.02)	−1.70 (2.81)	< 0.001	0.145
Education				< 0.001	0.176
College or university degree	8665 (43.09)	4736 (44.82)	6254 (33.46)		
A levels/AS levels or equivalent	2693 (13.39)	1401 (13.26)	2435 (13.03)		
O levels/GCSEs or equivalent	4119 (20.48)	2020 (19.12)	4554 (24.36)		
CSE or equivalent	800 (3.98)	460 (4.35)	983 (5.26)		
NVQ or HND or HNC or equivalent	1206 (6.00)	635 (6.01)	1158 (6.20)		
Others	2627 (13.06)	1315 (12.44)	3307 (17.69)		
Employed status				< 0.001	0.065
Employed	19,192 (95.44)	10,000 (94.63)	17,414 (93.17)		
Unemployed	918 (4.56)	567 (5.37)	1277 (6.83)		
Smoking status				< 0.001	0.06
Never	11,284 (56.11)	5811 (54.99)	10,095 (54.01)		
Ever	7612 (37.85)	4079 (38.60)	7061 (37.78)		
Current	1214 (6.04)	677 (6.41)	1535 (8.21)		
Frequency of alcohol drinking				< 0.001	0.165
Never	831 (4.13)	512 (4.85)	1347 (7.21)		
≤ 1–2 times a week	8984 (44.67)	4673 (44.22)	9834 (52.61)		
≥ 3 times a week	10,295 (51.19)	5382 (50.93)	7510 (40.18)		
Sleep duration				< 0.001	0.087
7–8 h/day	14,504 (72.12)	7560 (71.54)	12,603 (67.43)		
< 7 h/day	4416 (21.96)	2451 (23.19)	4634 (24.79)		
> 8 h/day	1190 (5.92)	556 (5.26)	1454 (7.78)		
Healthy diet score	3.32 (1.31)	3.36 (1.32)	3.28 (1.29)	< 0.001	0.042
BMI (kg/m^2)	28.62 (3.20)	28.59 (3.23)	30.21 (4.47)	< 0.001	0.278
SBP (mmHg)	139.46 (17.31)	138.81 (17.29)	139.74 (17.62)	< 0.001	0.035
DBP (mmHg)	83.91 (9.60)	83.86 (9.63)	84.06 (9.54)	0.169	0.014
HbA1c (mmol/mol)	35.38 (5.29)	35.42 (5.55)	36.50 (6.81)	< 0.001	0.122
HDL cholesterol (mmol/L)	1.40 (0.34)	1.41 (0.35)	1.39 (0.34)	0.010	0.026
LDL cholesterol (mmol/L)	3.69 (0.83)	3.66 (0.84)	3.68 (0.87)	0.006	0.026
Antihypertensive medications use				< 0.001	0.119
No	16,766 (83.37)	8877 (84.01)	14,385 (76.96)		
Yes	3344 (16.63)	1690 (15.99)	4306 (23.04)		
Antihyperglycemic medication use				< 0.001	0.084
No	19,697 (97.95)	10,341 (97.86)	17,899 (95.76)		
Yes	413 (2.05)	226 (2.14)	792 (4.24)		
Lipid-lowering medication use				< 0.001	0.079
No	17,514 (87.09)	9243 (87.47)	15,573 (83.32)		
Yes	2596 (12.91)	1324 (12.53)	3118 (16.68)		

Data are showed as mean (SD) for continuous variables and n (%) for categorized variables

Abbreviations: *AS* Advanced Subsidiary, *GCSEs* General Certificate of Secondary Educations, *CSE* Certificate of Secondary Education, *NVQ* National Vocational Qualification, *HND* Higher National Diploma, *HNC* Higher National Certificate, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HbA1c* hemoglobin A1c, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein, *SMD* standardized mean difference; an SMD ≥ 0.10 is considered to indicate a meaningful imbalance. SMD is the recommended metric for assessing covariate balance in observational studies

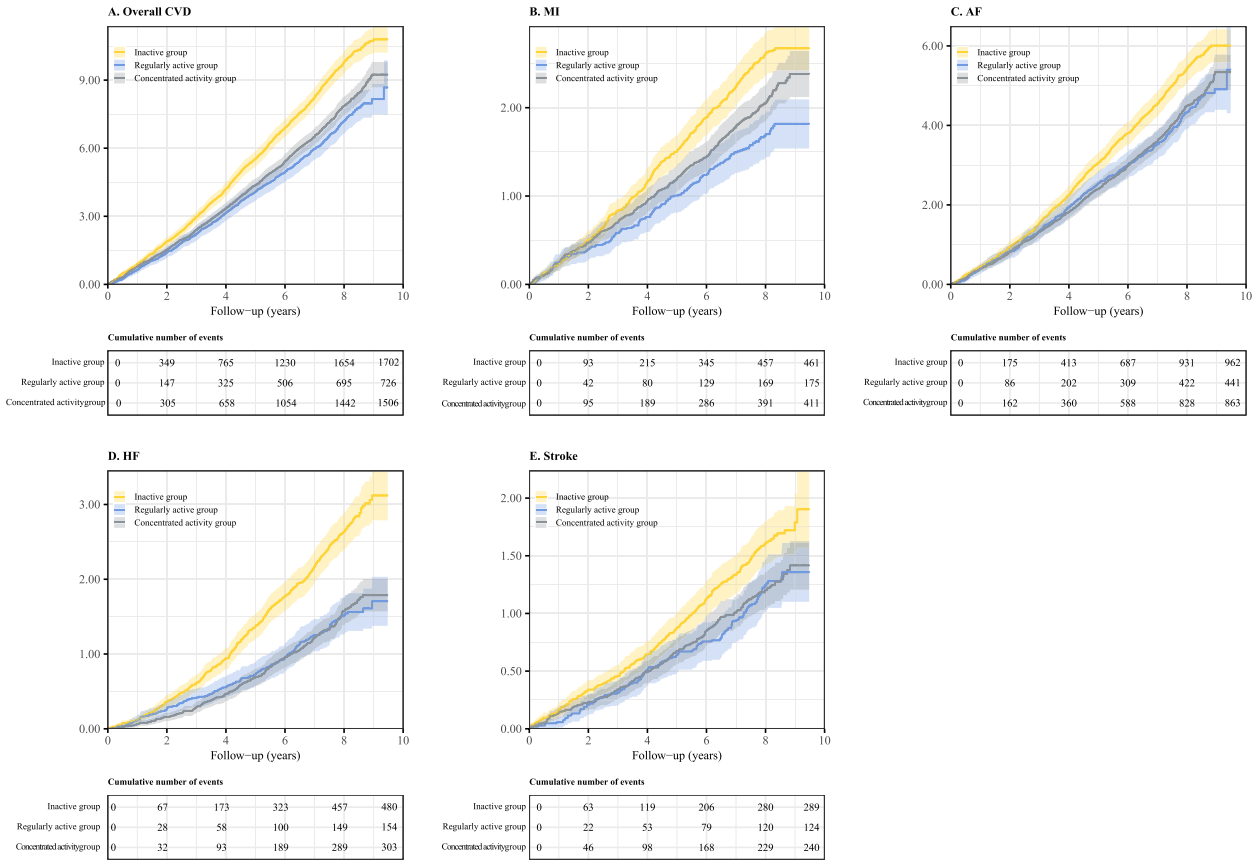

Fig. 1 Cumulative Incidence of Cardiovascular Events by Physical Activity Pattern in Individuals with Overweight or Obesity. *Note*. Kaplan–Meier curves illustrate the cumulative event probability over up to 10 years of follow-up among participants with overweight or obesity. Curves represent three physical activity patterns: inactive (yellow), regularly active (gray), and concentrated activity (blue). Shaded bands indicate 95% confidence intervals. Across all outcomes (**A** Composite CVD, **B** Myocardial infarction, **C** Atrial fibrillation, **D** Heart failure, **E** Stroke), the inactive group shows the highest cumulative incidence. For clinical context, absolute event rates (events per 1,000 person-years) are provided in the table below each panel

Subgroup analyses

Protective associations were consistent across the overweight and obese subgroups (Additional file 1: Table S5) and across key demographic strata including age, sex, smoking status, and alcohol consumption (Table 2). Furthermore, stratified analyses based on baseline medication use (antihypertensive, antihyperglycemic, lipid-lowering), health status (hypertension, diabetes, dyslipidemia), and self-reported health (excellent/good or fair/poor) demonstrated consistent protective associations in all subgroups (Additional file 1: Table S6). These findings indicate that baseline medication use and prevalent metabolic conditions did not substantially modify the observed associations between PA patterns and incident CVD.

Sensitivity analyses

Multiple sensitivity analyses confirmed the robustness of primary findings. First, a lag-period analysis that excluded CVD events within 2 years of accelerometer assessment yielded consistent results (composite CVD: concentrated activity HR 0.79, 95% CI 0.73–0.85; regularly active HR 0.75, 95% CI 0.68–0.83) (Additional file 1: Table S7). Second, analyses using population-specific MVPA thresholds (Additional file 1: Table S8-S9) and examining temporal patterns (consecutive days) (Additional file 1: Table S10) vs. weekend-specific (Additional file 1: Table S11) yielded concordant results. Third, results remained consistent when adjusting for potential mediators and reclassifying incomplete data (Additional file 1: Tables S12-S13). Fourth, sensitivity analysis using multiple imputation yielded results consistent with the complete-case analysis (Additional file 1: Table S14).

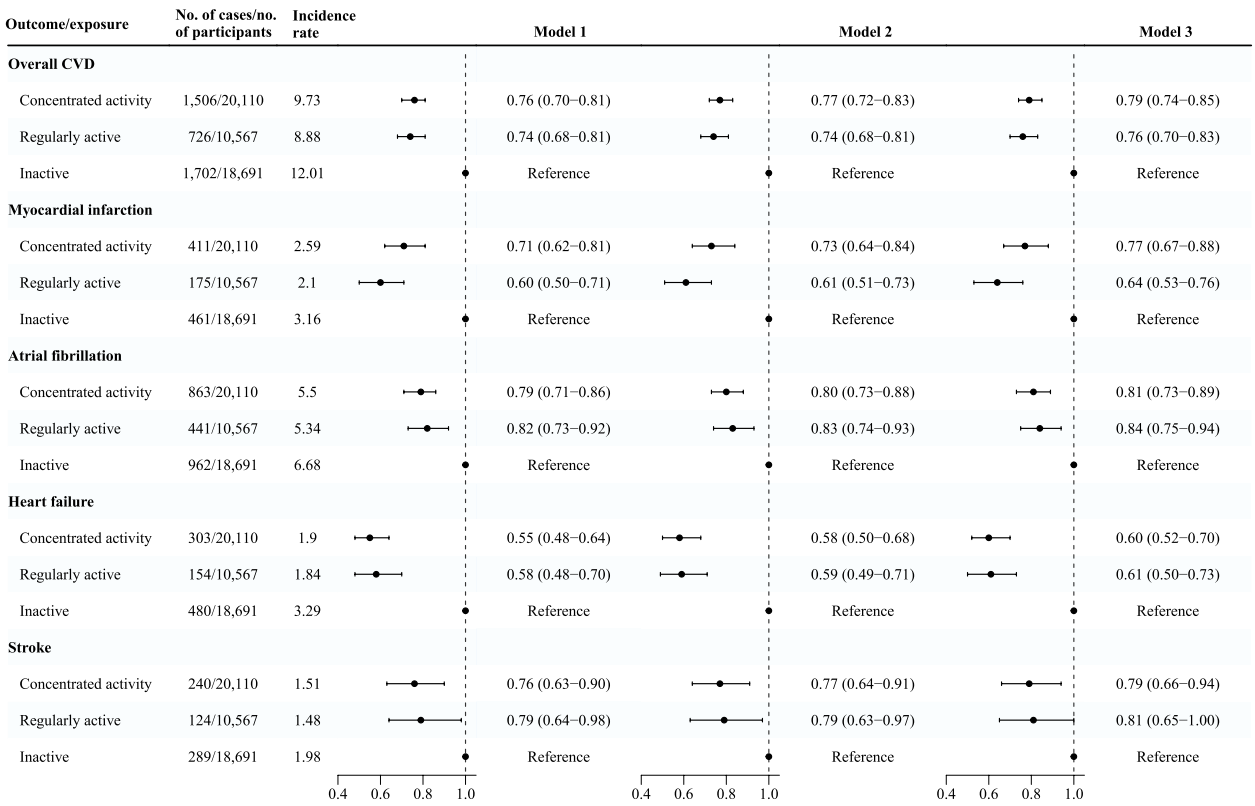

Fig. 2 Association Between Physical Activity Patterns and Incident Cardiovascular Disease in Participants with Overweight or Obesity. *Note*. Forest plot showing hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between physical activity patterns and incident cardiovascular disease outcomes among 49,368 UK Biobank participants with overweight or obesity. The concentrated activity pattern was defined as achieving ≥ 150 min/week of moderate-to-vigorous physical activity (MVPA) with ≥ 50% concentrated within 1–2 days. The regularly active pattern was defined as achieving ≥ 150 min/week of MVPA distributed across ≥ 3 days. The inactive group served as the reference category. Model 1 was adjusted for age, sex, and ethnicity; Model 2 was additionally adjusted for Townsend deprivation index, education, and employment status; Model 3 was further adjusted for smoking status, alcohol consumption, sleep duration, and diet quality. Numbers represent incident cases/total participants at risk. Incidence rates are expressed per 1,000 person-years. For stroke in the regularly active group (Model 3), the 95% CI crosses 1.00, indicating that the association was not statistically significant at $\alpha = 0.05$

Fifth, refined stratification of regularly active pattern showed consistent results (Additional file 1: Table S15).

E-value analysis demonstrated considerable robustness to unmeasured confounding (Additional file 1: Table S16). For the composite CVD endpoint, the E-values for the lower 95% confidence limits were 1.63 for concentrated activity and 1.70 for regularly active individuals. These suggest that an unmeasured confounder would need to be associated with both the PA pattern and incident CVD by relative risks of at least 1.63–1.70 to fully nullify the observed protective associations, a strength of association that exceeds that of most established cardiovascular risk factors.

Discussion

Principal findings

Our analyses revealed that both concentrated activity and regularly active patterns, when meeting guideline-recommended PA levels, are associated with a substantially lower risk of cardiovascular disease compared to inactivity. For the composite CVD outcome, concentrated activity showed a 21% risk reduction (HR 0.79, 95% CI 0.74–0.85), while regularly active individuals showed a 24% risk reduction (HR 0.76, 95% CI 0.70–0.83), with broadly overlapping 95% confidence intervals. Protective associations were observed for all specific cardiovascular outcomes examined: myocardial infarction, atrial fibrillation, heart failure, and stroke. Notably, the risk reduction for myocardial infarction appeared more pronounced in the regularly active group (HR 0.64) than in the concentrated activity group (HR 0.77); this difference approached, but did not reach, conventional statistical significance in direct comparison ($P = 0.053$). For the other outcomes, the magnitude of risk reduction was similar between the two activity patterns. These findings suggest that, in

Table 2 Subgroup analyses of the associations between physical activity pattern and incident cardiovascular disease

CVD outcome/PA pattern	Age		Sex		Smoking status		Alcohol drinking	
	< 65 years	≥ 65 years	Female	Male	Never	Ever or current	< 1 time/week	≥ 1 time/week
Overall CVD								
Concentrated activity	0.79 (0.69–0.89)	0.76 (0.69–0.82)	0.76 (0.68–0.86)	0.81 (0.74–0.89)	0.81 (0.72–0.89)	0.77 (0.70–0.85)	0.72 (0.63–0.84)	0.81 (0.75–0.88)
Regularly active	0.65 (0.55–0.76)	0.78 (0.70–0.87)	0.79 (0.68–0.92)	0.75 (0.68–0.84)	0.78 (0.69–0.89)	0.73 (0.65–0.83)	0.81 (0.68–0.95)	0.75 (0.67–0.83)
Inactive	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
P for interaction		0.029		0.478		0.74		0.089
MI								
Concentrated activity	0.75 (0.60–0.93)	0.74 (0.62–0.89)	0.63 (0.49–0.82)	0.83 (0.71–0.99)	0.82 (0.67–1.00)	0.71 (0.59–0.85)	0.63 (0.47–0.83)	0.80 (0.68–0.94)
Regularly active	0.49 (0.37–0.65)	0.72 (0.58–0.90)	0.51 (0.36–0.74)	0.70 (0.57–0.86)	0.69 (0.54–0.90)	0.57 (0.45–0.74)	0.78 (0.57–1.06)	0.59 (0.47–0.73)
Inactive	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
P for interaction		0.066		0.086		0.425		0.023
AF								
Concentrated activity	0.79 (0.66–0.94)	0.77 (0.69–0.86)	0.79 (0.68–0.93)	0.82 (0.72–0.92)	0.82 (0.71–0.94)	0.79 (0.69–0.90)	0.79 (0.65–0.96)	0.81 (0.73–0.91)
Regularly active	0.67 (0.54–0.83)	0.86 (0.75–0.98)	0.85 (0.70–1.04)	0.83 (0.72–0.96)	0.83 (0.70–0.98)	0.84 (0.72–0.99)	0.86 (0.69–1.09)	0.83 (0.73–0.95)
Inactive	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
P for interaction		0.082		0.902		0.785		0.803
HF								
Concentrated activity	0.70 (0.52–0.94)	0.54 (0.45–0.64)	0.56 (0.43–0.72)	0.63 (0.52–0.76)	0.54 (0.43–0.67)	0.64 (0.53–0.78)	0.60 (0.45–0.80)	0.61 (0.51–0.73)
Regularly active	0.60 (0.42–0.85)	0.57 (0.46–0.71)	0.54 (0.39–0.76)	0.64 (0.51–0.81)	0.56 (0.42–0.74)	0.63 (0.49–0.80)	0.53 (0.36–0.76)	0.64 (0.51–0.79)
Inactive	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
P for interaction		0.142		0.489		0.534		0.786
Stroke								
Concentrated activity	0.75 (0.55–1.03)	0.76 (0.61–0.94)	0.85 (0.65–1.12)	0.74 (0.59–0.94)	0.80 (0.61–1.04)	0.77 (0.60–0.97)	0.61 (0.43–0.88)	0.85 (0.69–1.05)
Regularly active	0.70 (0.49–1.02)	0.80 (0.61–1.04)	0.83 (0.58–1.18)	0.79 (0.60–1.04)	0.90 (0.66–1.24)	0.73 (0.54–0.98)	0.76 (0.51–1.15)	0.83 (0.64–1.07)
Inactive	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
P for interaction		0.789		0.833		0.702		0.138

this high-risk population, the cardiovascular benefits of MVPA were comparable whether the activity is concentrated within 1–2 days or distributed throughout the week.

Our findings contrast with Lars et al. [23] who reported that higher levels of self-reported PA may not fully offset the negative effects on CVD in overweight or obese adults, which differ from the findings of our study. This discrepancy could be due to the methodology used to measure PA (self-report) and the study's relatively small sample size. Compared with self-reported methods, accelerometers usually offer a more precise assessment of PA, as they minimize recall bias and reduce misclassification of activity intensity. With a larger sample size and accelerometer-based PA data, we observed significant associations between PA and a reduced risk of overall CVD and its four subtypes. Additionally, we applied multiple thresholds to test the consistency of our findings,

further enhancing the reliability of the results. Such flexibility is particularly important for encouraging sustained participation and addressing barriers to regular exercise in this high-risk population.

Regression dilution bias may arise if a single week of accelerometer monitoring inadequately captures long-term PA patterns. However, studies using multi-week accelerometer data in middle-aged and older adult populations have demonstrated substantial stability in measurements taken over 4–7 days. For example, Troiano et al. showed that 4–7 days of monitoring provides stable estimates of habitual PA in the NHANES data [24]. Similarly, Masse et al. showed that when four distinct data-processing algorithms were applied to the same single-week accelerometer data, key outcome variables remained reasonably consistent, supporting the ability of a one-week protocol to reflect habitual patterns [25]. This evidence supports the validity of our single-week assessment for classifying activity patterns. Furthermore, any non-differential misclassification resulting from single-week measurement would typically bias risk estimates toward the null, meaning that the observed reductions in CVD risk represent conservative estimates of the true associations. Follow-up duration from accelerometer assessment was shorter than studies examining baseline PA questionnaire data from 2006–2010.

Strengths and limitations

This study has several important strengths. First, objective accelerometer measurement eliminated recall bias and misclassification inherent in self-reported PA. Second, by examining multiple CVD-specific endpoints, including MI, AF, HF and stroke, rather than only composite outcomes, we could assess whether different activity patterns exert distinct effects on individual cardiovascular phenotypes. Third, our focus on adults with overweight or obesity addresses a critical evidence gap in a population that experiences a disproportionate burden of CVD. Fourth, the large sample size (n = 49,368) and substantial median follow-up (7.94 years) allowed precise estimations of associations. Fifth, comprehensive sensitivity analyses, including lag-analysis exclusions, E-value assessments, multiple imputation, and alternative activity thresholds, demonstrated the robustness of the findings.

However, several limitations should be acknowledged. First, as an observational study, our findings demonstrate association and do not establish causality between PA patterns and CVD risk in individual with overweight or obesity. Definitive causal inference would require randomized controlled trials. Second, the UK Biobank cohort is characterized by a substantial selection bias, with a participation rate of only 5.4%. Participants are predominantly health-conscious individuals of higher socioeconomic status and educational attainment [26, 27]. Third, the generalizability of our findings to less health-engaged populations and to other ethnic or socioeconomic groups may be limited. Fourth, PA was assessed using accelerometry over only a single week, which may not fully reflect long-term behavioral patterns or account for seasonal variation. However, this approach represents a standard and well-validated protocol in large-scale population studies. Prior research has demonstrated that 4–7 days of accelerometer wear provides stable estimates of habitual activity in comparable populations [24, 25]. Moreover, any resulting non-differential misclassification would typically bias risk estimates toward the null; thus, the observed reductions in CVD risk are likely conservative. The reliability of this method is supported by the UK Biobank accelerometer substudy, in which a median wear time of 6.9 days proved feasible and reproducible for epidemiological analyses [20]. Although one week of accelerometer monitoring is a well-validated and widely adopted proxy for habitual activity, we acknowledge that longer monitoring periods would further improve the precision of activity pattern classification. Fifth, reverse causation remains a consideration, as undetected subclinical disease could lower PA. However, sensitivity analyses that excluded early events (first 2 years) and stratified by baseline health status showed consistently protective associations, suggesting that reverse causation is unlikely to explain our findings.

Conclusions

In conclusion, our data provides observational evidence that concentrated and regularly active patterns are associated with a similarly reduced risk of CVD in overweight or obese adults. The flexibility of the concentrated activity patterns offers opportunities for the promotion of PA in ways that are feasible and sustainable in this high-risk population. Our results suggest that healthcare providers and physical activity guidelines could consider incorporating flexible exercise schedules, highlighting that health benefits can still be achieved with limited weekly exercise sessions. This approach could be particularly beneficial for individuals who struggle to maintain regular workouts due to work or family commitments, ultimately leading to reduced CVD risk.

Abbreviations

AF	Atrial Fibrillation
AS	Advanced Subsidiary
A level	Advanced level
AS level	Advanced Subsidiary Level
BMI	Body Mass Index
CSE	Certificate of Secondary Education
CVD	Cardiovascular Disease
CSE	Certificate of Secondary Education
DBP	Diastolic Blood Pressure
HbA1c	Hemoglobin A1c

HDL	High-Density Lipoprotein
HDL-C	HDL-Cholesterol
HF	Heart Failure
HNC	Higher National Certificate
HND	Higher National Diploma
HR	Hazard Ratio
HND	Higher National Diploma
HNC	Higher National Certificate
GCSE	General Certificate of Secondary Education
LDL	Low-Density Lipoprotein
LDL-C	LDL-Cholesterol
MI	Myocardial Infarction
MVPA	Moderate-to-Vigorous Physical Activity
NVQ	National Vocational Qualification
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
WHO	World Health Organization

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12916-026-04701-6.

Additional file 1: Figure S1. Flowchart of Inclusion/Exclusion of Study Participants. Table S1. Definitions of cardiovascular diseases based on the 10th Revision of the International Classification of Diseases. Table S2. Definitions of covariates. Table S3. Comparisons of characteristics of included participants in the primary analysis versus those who were excluded because of having incomplete accelerometer data. Table S4. Direct Comparison of Concentrated Activity versus Regularly Active Physical Activity Patterns for Cardiovascular Disease Outcomes. Table S5. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity. Table S6. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity. Table S7. Associations between physical activity pattern and incident cardiovascular disease in study participants with overweight or obesity. Table S8. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity with ≥50% over 1-2 days). Table S9. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity with ≥50% over 1-2 days). Table S10. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity. Table S11. Associations between physical activity pattern and incident cardiovascular disease in participants with overweight or obesity. Table S12. Associations between physical activity pattern and incident cardiovascular disease in study participants with overweight or obesity. Table S13. Associations between physical activity pattern and incident cardiovascular disease in study participants with overweight or obesity. Table S14. Associations between physical activity pattern and incident cardiovascular disease in study participants with overweight or obesity. Table S15. Associations between physical activity patterns and incident cardiovascular disease in participants with overweight or obesity. Table S16. E-value sensitivity analysis for the association between physical activity pattern and incident cardiovascular disease.

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Authors' contributions
B. X. participated in the design of the study and contributed to the manuscript writing. K. M. participated in the data interpretation and manuscript writing. Y.N.Q. contributed to the data cleaning and analyses. R.L. and Y.F.Y. provided statistical consultation and methodological guidance. M.Z. contributed to study conceptualization and critical revision of the manuscript for important intellectual content. C.G.M. provided critical revision and contributed to the interpretation of findings. All authors read and approved the final manuscript. All authors agreed to the author order as presented.

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Data availability
The data supporting this study are available through the UK Biobank resource (https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access). Access requires approval via a standard application process.

Declarations

Ethics approval and consent to participate
This study was performed via the UK Biobank Resource (Application Number: 98410) and all participants signed an informed consent. All methods were performed in accordance with the relevant guidelines and regulations, as approved by UK Biobank Resource.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Naghavi M, Ong KL, Aali A, Ababneh HS, Abate YH, Abbafati C, et al. Global burden of 288 causes of death and life expectancy decomposition in 204 countries and territories and 811 subnational locations, 1990–2021: A systematic analysis for the global burden of disease study 2021. Lancet. 2024;403:2100–32. https://doi.org/10.1016/S0140-6736(24)00367-2.
2. Kany S, Al-Alusi MA, Rämö JT, Pirruccello JP, Churchill TW, Lubitz SA, et al. Associations of "weekend warrior" physical activity with incident disease and cardiometabolic health. Circulation. 2024;150:1236–47. https://doi.org/10.1161/CIRCULATIONAHA.124.068669.
3. Martin SS, Aday AW, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, et al. 2024 heart disease and stroke statistics: A report of US and global data from the American heart association. Circulation. 2024;149(8):e347-913. https://doi.org/10.1161/CIR.0000000000001209.
4. Powell-Wiley TM, Poirier P, Burke LE, Després J-P, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: a scientific statement from the American heart association. Circulation. 2021;143:e984-1010. https://doi.org/10.1161/CIR.0000000000000973.
5. Jia Z, Wang Z, Pan H, Zhang J, Wang Q, Zhou C, et al. Crosstalk between fat tissue and muscle, brain, liver, and heart in obesity: cellular and molecular perspectives. Eur J Med Res. 2024;29:637. https://doi.org/10.1186/s40001-024-02176-w.
6. Chang Q, Zhu Y, Liu Z, Cheng J, Liang H, Lin F, et al. Replacement of sedentary behavior with various physical activities and the risk of all-cause and cause-specific mortality. BMC Med. 2024;22:385. https://doi.org/10.1186/s12916-024-03599-2.
7. Zhao M, Veeranki SP, Magnussen CG, Xi B. Recommended physical activity and all cause and cause specific mortality in US adults: Prospective cohort study. BMJ. 2020:m2031. https://doi.org/10.1136/bmj.m2031.
8. World Health Organization. Global recommendations on physical activity for health. World Health Organization; 2010.

9. Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021;42:3227–337. https://doi.org/10.1093/eurheartj/ehab484.
10. Physical activity guidelines for adults aged 19 to 64. NHS.UK. 2022. https://www.nhs.uk/live-well/exercise/physical-activity-guidelines-for-adults-aged-19-to-64/. Accessed 7 Jan 2025.
11. Vergallo R, Galiuto L. Physical activity patterns and cardiovascular health: 'yes, weekend!' Eur Heart J. 2023;44:4406–7. https://doi.org/10.1093/eurheartj/ehad520.
12. Kunutsor SK, Jae SY, Laukkanen JA. 'weekend warrior' and regularly active physical activity patterns confer similar cardiovascular and mortality benefits: a systematic meta-analysis. Eur J Prev Cardiol. 2023;30:e7-10. https://doi.org/10.1093/eurjpc/zwac246.
13. Ye Z, Liu M, Yang S, Zhang Y, Zhang Y, He P, et al. Association of accelerometer-derived "weekend warrior" moderate to vigorous physical activity, chronic kidney disease and acute kidney injury. Prev Med. 2024;187:108120. https://doi.org/10.1016/j.ypmed.2024.108120.
14. Khurshid S, Al-Alusi MA, Churchill TW, Guseh JS, Ellinor PT. Accelerometer-Derived "Weekend Warrior" Physical Activity and Incident Cardiovascular Disease. JAMA. 2023;330:247. https://doi.org/10.1001/jama.2023.10875.
15. Wang K, Xia F, Li Q, Luo X, Wu J. The associations of weekend warrior activity patterns with the visceral adiposity index in US adults: repeated cross-sectional study. JMIR Public Health Surveill. 2023;9:e41973. https://doi.org/10.2196/41973.
16. Lei L, Li J, Wang W, Yu Y, Pu B, Peng Y, et al. The associations of "weekend warrior" and regularly active physical activity with abdominal and general adiposity in US adults. Obesity. 2024;32:822–33. https://doi.org/10.1002/oby.23986.
17. Cao Z, Min J, Chen H, Hou Y, Yang H, Si K, et al. Author correction: Accelerometer-derived physical activity and mortality in individuals with type 2 diabetes. Nat Commun. 2024;15:9972. https://doi.org/10.1038/s41467-024-54119-y.
18. Strain T, Wijndaele K, Dempsey PC, Sharp SJ, Pearce M, Jeon J, et al. Wearable-device-measured physical activity and future health risk. Nat Med. 2020;26:1385–91. https://doi.org/10.1038/s41591-020-1012-3.
19. Walmsley R, Chan S, Smith-Byrne K, Ramakrishnan R, Woodward M, Rahimi K, et al. Reallocation of time between device-measured movement behaviours and risk of incident cardiovascular disease. Br J Sports Med. 2022;56:1008–17. https://doi.org/10.1136/bjsports-2021-104050.
20. Doherty A, Jackson D, Hammerla N, Plötz T, Olivier P, Granat MH, et al. Large scale population assessment of physical activity using wrist worn accelerometers: the UK biobank study. PLoS ONE. 2017;12:e0169649. https://doi.org/10.1371/journal.pone.0169649.
21. Morris R, Carstairs V. Which deprivation? A comparison of selected deprivation indexes. J Public Health Med. 1991;13:318–26.
22. Zhang Y-B, Chen C, Pan X-F, Guo J, Li Y, Franco OH, et al. Associations of healthy lifestyle and socioeconomic status with mortality and incident cardiovascular disease: Two prospective cohort studies. BMJ. 2021;373:n604. https://doi.org/10.1136/bmj.n604.
23. Lind L, Carlsson AC, Siegbahn A, Sundström J, Ärnlöv J. Impact of physical activity on cardiovascular status in obesity. Eur J Clin Invest. 2017;47:167–75. https://doi.org/10.1111/eci.12722.
24. Troiano RP, Berrigan D, Dodd KW, Mâsse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. Med Sci Sports Exerc. 2008;40:181–8. https://doi.org/10.1249/mss.0b013e31815a51b3.
25. Mâsse LC, Fuemmeler BF, Anderson CB, Matthews CE, Trost SG, Catellier DJ, et al. Accelerometer data reduction: A comparison of four reduction algorithms on select outcome variables. Med Sci Sports Exerc. 2005;37:S544-554. https://doi.org/10.1249/01.mss.0000185674.09066.8a.
26. van Alten S, Domingue BW, Faul J, Galama T, Marees AT. Reweighting UK biobank corrects for pervasive selection bias due to volunteering. Int J Epidemiol. 2024;53:dyae054. https://doi.org/10.1093/ije/dyae054.
27. Swanson JM. The UK biobank and selection bias. Lancet. 2012;380:110. https://doi.org/10.1016/S0140-6736(12)61179-9.

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