Do associations of physical activity and sedentary behaviour with cardiovascular disease and mortality differ across socioeconomic groups? A prospective analysis of device-measured and self-reported UK Biobank data

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ABSTRACT

Objectives: To examine if individual-level and area-level socioeconomic status (SES) modify the association of moderate-to-vigorous physical activity (MVPA), domain-specific physical activity and sedentary behaviour with all-cause mortality (ACM) and incident cardiovascular disease (CVD).

Methods: We used self-reported (International Physical Activity Questionnaire (IPAQ) short form) and device-measured (accelerometer (ACCEL)) physical activity and sedentary behaviour data from the UK Biobank. We created an individual-level composite SES index using latent class analysis of household income, education, and employment status. Townsend index was the measure of area-level SES. Cox proportional hazards regression models stratified across SES were used.

Results: In 328,228 participants (mean age 55.9 (8.1) years, 45% men) with an average follow-up of 12.1(1.4) years, 18,033 deaths and 98,922 incident CVD events occurred. We found an increased ACM risk for low physical activity and high sedentary behaviour and an increased incident CVD risk for low device-measured MVPA and high sitting time. We observed statistically significant interactions for all exposures in ACM analyses by individual-level SES ($p<0.05$) but only for screen time in area-level SES–ACM analysis ($p<0.001$). Compared to high IPAQ_MVPA, adjusted ACM hazard ratios (HRs) for low IPAQ_MVPA were 1.14(1.05-1.25), 1.15(1.06-1.24) and 1.22(1.13-1.31) in high, medium, and low individual-level SES, respectively. There were clear patterns of higher detrimental associations of low ACCEL_MVPA with decreasing area-level SES for both outcomes. High screen time had higher deleterious associations with ACM in low area-level SES. Effect modification by SES indices was unclear for domains and sitting time.

Conclusion: We found modest evidence suggesting that the detrimental associations of low MVPA and high screen time with ACM and incident CVD are accentuated in low SES groups.

Keywords: socioeconomic status, mortality, CVD, physical activity, sedentary behaviour

What is already known on this topic?

- Low SES groups have a higher prevalence of unhealthy lifestyles and may suffer disproportionate harm.
- Studies incorporating composite SES index, multiple domains of physical activity, sedentary behaviour and use of both self-report and device-measured assessments are limited.

What are the new findings?

- Our results suggest that there is a stronger inverse association of self-reported MVPA with all-cause mortality in low compared to high individual-level SES groups.
- We found higher detrimental associations of the low ACCEL_MVPA with all-cause mortality and incident CVD in low area-level SES; patterns were less clear for individual-level SES.
- The detrimental associations of high self-reported screen time with all-cause mortality were stronger in low area-level SES.
- Effect modification by SES was less clear for physical activity domains and sitting time.

How might it impact on clinical practice in the future?

- We recommend primary prevention interventions that tackle physical inactivity and excessive sedentary behaviour tailored to the needs of low SES groups.
- Considering the variability in the interaction effects across SES measures, it may be important to target both low individual-level and area-level SES groups.

BACKGROUND

Socioeconomic inequalities in health are a global challenge[1, 2]. They signify a range of differences in socioeconomic status (SES) as determined by an individual's economic and social position in relation to others, based on income, education, employment status or occupation, and ethnicity[1, 3]. Generally, individuals of low SES or those living in low socioeconomic areas have a higher prevalence of detrimental health-related behaviours[4] and may have less favourable health outcomes such as higher morbidity and mortality[1, 5-8]. Even for a similar level of exposure to risk factors, low SES groups may suffer worse overall health outcomes (a phenomenon termed as vulnerability hypothesis)[9, 10]. Overall, low SES may increase both exposure to chronic disease risk factors and increase the vulnerability of morbidity and impaired health upon exposure[11, 12].

The relationships between individual- and area-level SES, physical activity and sedentary behaviour have been extensively researched. Self-reported leisure-time physical activity (LTPA) is positively associated with high individual (education[13], employment[14], income[13]) and area-level SES[13, 15]. Studies using device-measured physical activity, which captures leisure time as well as occupational and incidental physical activity, have shown both direct[16, 17] and inverse[18] associations between physical activity and SES. Of the various SES measures used, some of the most consistent positive associations with physical activity are reported for education[19]. The detrimental associations of physical inactivity and sedentary behaviour with higher risks of cardiovascular diseases (CVD) and premature mortality are also well established[20-22].

In considering how to reduce socioeconomic inequalities in health, it is important to understand the interaction between SES and health behaviours in jointly determining future health outcomes[23, 24]. The scant evidence on the association between SES, physical activity, sedentary behaviour and health outcomes is unclear[5, 6] and less consistent between studies employing self-report and objective physical activity measures[25]. For example, a previous study reported more consistent and stronger associations of education and occupational social class with device-measured physical activity than with self-report[25]. In a UK Biobank analysis, Foster et al.[5] found a significant interaction between a composite lifestyle behaviour score and area-level SES (Townsend index) for risk of ACM and CVD mortality, but not CVD incidence[5]. Compared to the most healthy lifestyle, the association of the least healthy lifestyle with ACM was more pronounced in lower area-level SES[5]. Another recent study reported lower ACM and CVD risk among groups with healthy lifestyles, with stronger associations among low individual-level SES[6]. Both studies used composite lifestyle scores comprising multiple behavioural factors (e.g., alcohol, smoking, diet)[5, 6]. The physical activity component was limited to self-reported moderate-to-vigorous physical activity (MVPA)[5] or LTPA[6], and sedentary behaviour was limited to television viewing time[5], which is a poor proxy of overall sedentary time[26].

Social patterning (differences across the SES spectrum) in physical activity is more prominent for physical activity domains (e.g., transportation, occupational, household, and leisure-time) than for total physical activity [27, 28]. For example, European adults from high SES participate mostly in LTPA[28]. In contrast, adults from low SES mostly participated in occupational physical activity, while no variations by SES were observed for total physical activity and active commuting[28]. Another study reported higher device-measured sedentary behaviour and lower television viewing among higher SES[29]. No studies, to our knowledge, have examined how SES modifies the association of multiple domains of self-reported and device-measured physical activity and sedentary behaviour with mortality and incident CVD. Differential reporting bias could be more crucial in the context of SES, with another UK cohort (Whitehall II) reporting a weaker correlation between self-reported and device-measured physical activity data in low SES than in high SES groups and for moderate-intensity activities than vigorous activities[30]. In another study, Gorzelitz et al. concluded that discordance between self-reported and device-measured physical activity data was inversely correlated to educational level[31]. Accelerometry devices can capture very short bouts of MVPA as well as lower-intensity activities performed in any domain and overcome other important limitations of self-report measurements (e.g., recall or social desirability bias)[32, 33]. However, motion sensor devices such as accelerometers cannot capture domain-specific activities and can be logistically challenging to implement in low-resource settings due to higher time and resource requirements[34]. Using both self-reported and device-measured physical activity is recommended for a more complete understanding of the associations of physical activity with prospective health outcomes[34]. Further, understanding the role of SES in determining the associations of total physical activity, domains and sedentary behaviour with health outcomes is essential to narrow health disparities, a gap identified by the 2020 WHO Guideline Development Group[34].

The primary aim of this study was to examine whether individual-level SES modifies the association of total and domain-specific physical activity and sedentary behaviour with ACM and incident CVD. The secondary aim was to examine the same effect modification by area-level SES. We hypothesised that the detrimental associations of low physical activity and high sedentary behaviour with outcomes would be stronger in low SES (vulnerability hypothesis).

METHODS

Study design and participants

We used data from the UK Biobank, which recruited 502,656 adults aged 40–69 years between 2006 and 2010[35]. We excluded participants with missing covariates, socioeconomic information, or exposures; poor self-rated health; prevalent CVD (self-reported or hospital admission); or an event (death or CVD event) within two years of recruitment (Supplementary Figures S1 and S2).

Exposures

Supplemental Text S1 provides full descriptions of the exposure variables. Here, we summarise their main attributes:

Questionnaire-based physical activity: Weekly self-reported MVPA (IPAQ_MVPA) was measured using an adaption of the International Physical Activity Questionnaire (IPAQ) short form[36]. It has moderate validity (r = 0.52) for measuring MVPA among adults in the UK compared to accelerometer data[37]. Such correlations with accelerometry are higher than most other self-reported instruments[38]. We calculated total weekly IPAQ_MVPA volume (METs-minutes/week; number of minutes/week*standardised MET value of walking, and moderate and vigorous activities) and categorised participants into three groups: low (< 600 MET-minutes/week), medium (600 - 3000 MET-minutes/week), and high (≥ 3000 MET-min/week)[36].

*Device-measured physical activ*ity*:* Device-measured MVPA (ACCEL_MVPA) was derived in a subsample of participants using data from the Axivity AX3 accelerometer worn on their dominant wrist for 24-hours/day for one week[39]. We used previously established procedures[40, 41] to calibrate data and identify non-wear and only included participants with at least four valid monitoring days (at least one of those days being a weekend). We used a previously validated machine learning activity recognition scheme that uses raw acceleration signals to identify and quantify time spent in different intensities in 10-second windows[42]. Using the total weekly time spent in ACCEL_MVPA, we classified participants into tertiles for this study. The use of tertiles provided the optimal balance between physical activity exposure resolution and exposure group size.

Domain-specific physical activity: Weekly household physical activity volume was based on frequency and duration of light and heavy do-it-yourself activities (such as home maintenance, gardening, digging, carpentry, etc.) and categorised into tertiles. Weekly LTPA volume was based on the frequency and duration of walking for pleasure, other exercises and strenuous sports[43] and categorised into tertiles.

Sedentary behaviour: The study includes two forms of sedentary behaviours: device-measured sitting time and self-reported screen time. We categorised participants into tertiles of total weekly sitting time using the information from the Axivity AX3 accelerometer using the same process defined above. We created 'screen time' tertiles using self-reported daily hours spent watching TV and non-occupational computer use[44].

Outcomes

We examined associations with ACM and incident CVD. Incident CVD was defined as an event (fatal or non-fatal attributed to ICD-10 codes I00–I99) after baseline assessment. Participants were followed until an event or censoring (30th September 2021 for England/Wales and 31st October 2021 for Scotland due to rolling data linkage updates).

Effect modifiers

Supplemental Text S2 and Table S1 provide detailed descriptions of the socioeconomic indices. In brief, we examined effect modification by two composite socioeconomic indices: individual-level SES index and area-level SES using the Townsend index[45]. The individual-level composite SES index was created using latent class analysis of three socioeconomic variables (household income, education, and employment status)[6] and categorised as high, medium and low SES (Supplemental Text S2). Since the model with four latent classes failed to converge, we used the model with three latent classes. "High SES" had a higher proportion of participants with college or university degree and before tax household income of £52,000 or greater (see

Table S1). The proportion of unemployed, those with less than high school education (labelled as 'none' in UK Biobank) and those with household income less than £18,000 were higher in class labelled "low SES". The Townsend index is derived from the respondent's postcode and reflects unemployment, non-car ownership, non-home ownership and household overcrowding[45]. We categorised it into thirds using tertiles, where the lowest third indicated high area-level SES.

Covariates

Supplemental Table S2 provides complete descriptions of the covariates. We selected variables a priori from the relevant literature[5, 6]. We adjusted analyses for sex, ethnicity, sleep score (derived using morning chronotype, sleep duration, insomnia, snoring and daytime sleepiness)[46], dietary pattern score (from the intake of fruits, vegetables, fish, red meat and processed meat)[47], smoking and alcohol consumption.

Statistical analysis

We used multivariable-adjusted Cox proportional hazards regression stratified by socioeconomic indices, with age (scaled in years) as the underlying time scale. To address the impact of reverse causality, we have excluded the initial two years of follow-up and any events within it[5, 39, 48, 49]. The reference groups were the optimum category/tertile of the exposure variables (high physical activity /low sedentary behaviour). Model 1 (main effects) for all exposures was adjusted for the above covariates, Townsend index and education. For IPAQ_MVPA and LTPA analyses, we additionally adjusted for screen time; screen time analyses were adjusted for IPAQ_MVPA; ACCEL_MVPA analyses were adjusted for sitting time and vice versa; Household physical activity analyses were adjusted for LTPA and screen time. There was no evidence of multicollinearity between the variables entered in the model (variation inflation factor (VIF) ≤ 1.16).

Multiplicative interaction terms between exposures and individual-level and area-level SES were included in Model 2 and 3, respectively. We evaluated interactions between exposures (physical activity /sedentary behaviour) and socioeconomic indices using likelihood ratio tests comparing models with and without a cross-product term. P-value for interaction was obtained using continuous variables. Proportional hazard assumption was tested using Schoenfeld residuals[50] and was satisfied. For CVD incidence analyses, we used Fine and Gray subdistribution method[51] to account for competing risks (non-CVD related deaths).

We conducted several sensitivity analyses. First, we additionally adjusted ACM models stratified by individual-level SES for body mass index (BMI). Second, we repeated ACM models for physical activity exposures by adjusting for self-rated health instead of excluding them. Third, we excluded first three years of follow-up and events within these years to reduce potential reverse causation[6]. To further check the sensitivity of the estimates, we calculated E-values that indicate the strength of association an unmeasured confounder would need to have with exposure and outcome to explain away the observed exposure-outcome association[52]. All analyses were performed using STATA/MP 17.0 (StataCorp, TX, USA), with two-sided *p* values< .05 considered statistically significant. Study reporting conforms to STROBE guidelines[53] (see Supplemental STROBE Checklist).

Patient and public involvement

Patients and the public were not involved in the design or conduct of this study.

RESULTS

Sample characteristics

We analysed data from 328,228 participants (mean age 55.9 (8.1) years, 45% men). The low, medium, and high IPAQ_MVPA levels consisted of 15%, 48.6% and 36.4% participants. Over the mean follow-up period of 12.2 (1.4) years (3,922,258 person-years), 18,033 deaths and

98,922 incident CVD events occurred. Participant characteristics across IPAQ_MVPA and ACCEL_MVPA levels are presented in Table 1 and Supplementary Table S3. Supplementary Table S4 shows the distribution of exposure variables across individual-level SES.

Table 1:Baseline characteristics of participants stratified by level of self-reported MVPA (n=328,228)

Characteristics	**Total population (n= 328,228)**	**IPAQ_MVPA (n=310,499)**			
		High (n=113,053)	**Medium (n=150,763)**	**Low (n=46,683)**	**p-value**
Mean age (SD) (years)	55.9 ± 8.1	56.2 ± 8.2	55.6 ± 8.1	55.1 ± 7.8	<0.001
Men	148,522 (45.2%)	52,285 (46.2%)	68,212 (45.2%)	21,873 (46.9%)	<0.001
White ethnicity or race	313,783 (95.6%)	108,619 (96.1%)	144,313 (95.7%)	44,393 (95.1%)	<0.001
Household income (£)					<0.001
Less than 18,000	65,250 (19.9%)	25,634 (22.7%)	25,367 (16.8%)	7,469 (16.0%)	
18,000 to 30,999	82,782 (25.2%)	31,830 (28.2%)	35,434 (23.5%)	10,351 (22.2%)	
31,000 to 51,999	88,932 (27.1%)	30,340 (26.8%)	41,676 (27.6%)	13,256 (28.4%)	
52,000 to 100,000	71,789 (21.9%)	20,335 (18.0%)	37,255 (24.7%)	12,388 (26.5%)	
Greater than 100,000	19,475 (5.9%)	4,914 (4.3%)	11,031 (7.3%)	3,219 (6.9%)	
Education					<0.001
None	43,483 (13.2%)	18,350 (16.2%)	14,578 (9.7%)	4,640 (9.9%)	
O/CSE or equivalent	88,309 (26.9%)	33,348 (29.5%)	37,505 (24.9%)	12,432 (26.6%)	
A/NVQ/professional or equivalent	77,006 (23.5%)	27,462 (24.3%)	34,761 (23.1%)	11,006 (23.6%)	
College/University	119,430 (36.4%)	33,893 (30.0%)	63,919 (42.4%)	18,605 (39.9%)	
Employment					<0.001
Employed	311,760 (95.0%)	107,396 (95.0%)	143,745 (95.3%)	44,382 (95.1%)	
Unemployed	16,468 (5.0%)	5,657 (5.0%)	7,018 (4.7%)	2,301 (4.9%)	
Townsend Index tertile					<0.001
First	111,076 (33.8%)	36,884 (32.6%)	52,896 (35.1%)	16,539 (35.4%)	
Second	110,210 (33.6%)	38,274 (33.9%)	50,534 (33.5%)	15,760 (33.8%)	
Third	106,942 (32.6%)	37,895 (33.5%)	47,333 (31.4%)	14,384 (30.8%)	
Smoking status					<0.001
Never	182,037 (55.5%)	61,552 (54.4%)	85,281 (56.6%)	25,936 (55.6%)	
Previous	113,664 (34.6%)	39,835 (35.2%)	52,203 (34.6%)	15,601 (33.4%)	
Current	32,527 (9.9%)	11,666 (10.3%)	13,279 (8.8%)	5,146 (11.0%)	
Alcohol status					<0.001
Never	11,384 (3.5%)	3,859 (3.4%)	4,634 (3.1%)	1,742 (3.7%)	

Previous	9,530 (2.9%)	3,520 (3.1%)	3,893 (2.6%)	1,357 (2.9%)	
Current	307,314 (93.6%)	105,674 (93.5%)	142,236 (94.3%)	43,584 (93.4%)	
Sleep pattern					<0.001
Poor	22,062 (6.7%)	7,168 (6.3%)	9,361 (6.2%)	3,752 (8.0%)	
Intermediate	185,713 (56.6%)	62,458 (55.2%)	84,495 (56.0%)	28,091 (60.2%)	
Healthy	120,453 (36.7%)	43,427 (38.4%)	56,907 (37.7%)	14,840 (31.8%)	
Diet pattern					<0.001
Poor	20,120 (6.1%)	6,314 (5.6%)	8,370 (5.6%)	4,150 (8.9%)	
Reasonable	201,082 (61.3%)	66,724 (59.0%)	92,368 (61.3%)	30,747 (65.9%)	
Good	107,026 (32.6%)	40,015 (35.4%)	50,025 (33.2%)	11,786 (25.2%)	
Body mass index					<0.001
Normal weight	112,801 (34.4%)	41,601 (36.8%)	53,460 (35.5%)	12,892 (27.6%)	
Overweight	141,884 (43.2%)	49,178 (43.5%)	65,555 (43.5%)	19,801 (42.4%)	
Obese	73,543 (22.4%)	22,274 (19.7%)	31,748 (21.1%)	13,990 (30.0%)	
Self-rated health					
Excellent	61,350 (18.7%)	24,460 (21.6%)	29,240 (19.4%)	5,966 (12.8%)	<0.001
Good	201,826 (61.5%)	69,432 (61.4%)	94,153 (62.5%)	27,827 (59.6%)	
Fair	65,052 (19.8%)	19,161 (16.9%)	27,370 (18.2%)	12,890 (27.6%)	

Participants' physical activity (IPAQ_MVPA) measured using the International Physical Activity Questionnaire was categorised as low (< 600 metabolic equivalent (MET)-min/week), medium (600 to < 3000 MET-min/week), and high (≥ 3000 MET-min/week). Townsend index (including measures of unemployment, non-car ownership, non-home ownership and household overcrowding), derived from respondent's postcode was used as an indicator of area-level SES. We categorised Townsend index into tertiles where the lowest score indicated the least socioeconomic deprivation. Employment status is categorised as employed (includes paid employment or self-employed, retired, paid or voluntary work or student) and unemployed (includes looking after home and/or family, unable to work and unemployed). Sleep pattern is derived using sleep duration, chronotype, insomnia, snoring and dozing. Diet pattern is derived using intake of fruits and vegetables, fish (oily and non-oily), red meat (beef, pork and lamb) and proceeded meat intake. BMI is categorised as normal weight (18.5 to <25 kg/m2), overweight (25.0 to <30 kg/m2) and obesity (≥ 30 kg/m2).

Values in the table are frequencies and percentages unless otherwise stated. Differences between groups was tested using one-way ANOVA for age and using chi-square test for other variables.

All-cause mortality

Whole sample

We found detrimental associations of low IPAQ_MVPA (HR: 1.15 (95% CI: 1.10-1.20)), low ACCEL_MVPA (1.62 (1.39-1.89)) and low household physical activity (1.06 (1.01-1.12)) with ACM (Supplementary Table S5). The HRs for mortality were higher among participants in medium and lowest tertile of LTPA, compared to those in the highest LTPA tertile. Participants in the highest screen time and sitting time tertile were at 12% (9%-17%) and 19% (2%-39%)

higher hazard of mortality than those in the lowest tertile, respectively (Supplementary Table S5). For individual-level SES, we observed significant likelihood ratio tests (p<0.05) for all exposures. The multiplicative interaction term was only significant for screen time (p-value for screen time*area-level SES <0.001).

Stratified by individual-level SES

Figure 1 shows the stratified association of MVPA and domain-specific physical activity with ACM across individual-level SES. There was no statistically significant association of medium IPAQ_MVPA and ACCEL_MVPA with ACM across all levels of individual-level SES. However, there was a stronger detrimental association of low IPAQ_MVPA with ACM in low SES. For example, compared with high IPAQ_MVPA, ACM HRs for low IPAQ_MVPA were 1.14 (1.05-1.25) in high SES, 1.15 (1.06-1.24) in medium SES and 1.22 (1.13-1.31) in low SES. We observed no clear individual-level SES gradient in the associations of ACCEL_MVPA with ACM, though there was a slightly more pronounced detrimental association of low ACCEL_MVPA in low SES. HRs for low ACCEL_MVPA were 1.80 (1.33-2.43) in low SES, 1.47 (1.13-1.91) in medium SES and 1.67 (1.28-2.08) in high SES. Low LTPA was inversely associated with mortality in all groups, with less clear SES patterning. We observed some evidence of higher mortality HRs of medium LTPA among low and medium SES groups only (1.07 (0.99-1.16) in high SES, 1.12 (1.04-1.20) in medium SES and 1.08 (1.01-1.15) in low SES). There was no association of household physical activity with ACM across SES groups (Figure 1).

We found no evidence of association of sitting time with ACM across all individual-level SES groups (except the highest tertile in medium SES (HR: 1.33(1.02-1.73)) (Figure 2). High screen time was detrimentally associated with ACM only among low and high SES groups, with a more pronounced association in high SES. For example, compared to low screen time, ACM

HRs for high screen time were 1.10 (1.04-1.17) in low SES, 1.04 (0.98-1.11) in medium SES and 1.19 (1.11-1.28) in high SES (Figure 2).

Results were largely consistent with the main models when we further adjusted individual-level SES models of physical activity (Figure S3) and sedentary behaviour (Figure S4) for BMI. When we adjusted the main physical activity models for self-rated health (instead of excluding participants with poor self-rated health), the detrimental associations of low IPAQ_MVPA and low LTPA with ACM were attenuated in medium and high-SES (Figure S5). Removing the first three years of follow-up did not appreciably change the results obtained in the main analysis (Figure S6).

Stratified by area-level SES

Low IPAQ_MVPA and ACCEL_MVPA were associated with higher ACM risk in all area-level SES groups (Figure S7). We observed higher ACM HRs of low ACCEL_MVPA in low and medium SES. For example, HRs for low ACCEL_MVPA were 1.78 (1.36-2.29), 1.71 (1.31-2.25) and 1.41 (1.08-1.84) in low, medium, and high area-level SES groups, respectively. The detrimental associations of medium and low tertiles of LTPA were more pronounced in medium SES. We found clear detrimental associations of low household physical activity in the low SES group only (Figure S7).

We observed a clear gradient of stronger detrimental associations of screen time with ACM with decreasing area-level SES (Figure S8). For example, compared to the lowest screen time tertile, ACM HRs for high screen time were 1.07 (1.01-1.14) in high, 1.13 (1.06-1.20) in medium and 1.22 (1.15-1.29) in low SES groups. There was no association of sitting time with ACM across all area-level SES groups.

Incident CVD

Whole sample

Compared to high ACCEL_MVPA, participants in medium (HR: 1.11 (1.05-1.17)) and lowest tertile (1.14 (1.07-1.21)) were at an increased incident CVD risk. Our results showed detrimental associations of the highest sitting time tertile (1.11 (1.05-1.18)) with incident CVD (Supplementary Table S5). We did not find statistically significant associations of self-reported physical activity and sedentary behaviour exposures with incident CVD. The multiplicative interaction term was not significant for all exposures.

Stratified by individual-level SES

Figure 3 shows the stratified association of MVPA and domain-specific physical activity with incident CVD across individual-level SES. The individual-level SES patterns of the association of IPAQ_MVPA with incident CVD was less clear. We observed clear detrimental associations of the lowest tertile of ACCEL_MVPA in medium and high SES and that of medium tertile in high SES only. For example, HRs for low ACCEL_MVPA were 1.13 (0.99-1.28) in low SES, 1.14 (1.04-1.25) in medium SES and 1.15 (1.06-1.26)) in high SES, respectively. There was no association of LTPA and household physical activity with incident CVD across SES groups (Figure 3).

Sitting time (except highest tertile in high SES) and screen time were not associated with incident CVD across all individual-level SES groups (Figure S9). Compared to participants in the lowest sitting time tertile, high SES participants in the highest tertile were at 13% higher hazard of incident CVD (HR: 1.13 (1.03-1.23)).

Stratified by area-level SES

We observed a clear SES gradient of association of low ACCEL_MVPA with incident CVD; the detrimental associations became stronger with decreasing area-level SES. For example, compared to high ACCEL_MVPA, HRs of low ACCEL_MVPA were 1.20 (1.09-1.32)), 1.13 (1.03-1.24) and 1.14 (0.98-1.32) in low, medium, and high area-level SES, respectively.

IPAQ_MVPA, LTPA and household physical activity were not associated with incident CVD across all SES groups (Figure S10).

The deleterious association of high sitting time tertile with incident CVD was observed in medium SES only (Figure S11). Screen time was not associated with incident CVD across all area-level SES strata.

We have provided E-values for all significant associations in Table S6. More than half of all e-values for significant associations in the main analysis had an HR>1.50. For example, an unmeasured confounder would have to have an association of 3.00 with the exposure and outcome to explain away the observed HR of 1.80 of low ACCEL_MVPA and ACM association in low individual-level SES, but weaker confounding could not do so.

DISCUSSION

This study investigated if SES modifies the association of physical activity and sedentary behaviour with ACM and incident CVD. We found detrimental associations of low MVPA with ACM and incident CVD and of high screen time with ACM, with some evidence of stronger detrimental associations in low SES groups. Our findings suggested some variability in the interaction effects of SES on exposure-outcome associations depending on the SES and physical activity measure we tested. SES patterns were clearer for individual-level SES while using self-reported MVPA and for area-level SES while using device-measured MVPA. These findings may inform public health policy and practice by identifying vulnerable individuals and priority target groups for physical inactivity and sedentary behaviour interventions.

SES may influence an individual's access to health information, treatment choices, compliance to treatment regimens, quality of care and social support, resulting in differential prognosis for similar risk factors or health conditions[54]. Previous studies have suggested that low socioeconomic groups may suffer disproportionate harm from unhealthy behaviours such as smoking[10, 55] and alcohol consumption[56]. However, there is limited evidence on the interaction of SES and physical activity and sedentary behaviour for prospective health outcomes[5]. Studies using a single individual-level SES measure have shown inconsistent results. For example, Moore et.al[57] found a stronger beneficial association of higher LTPA with mortality among those with a college education than those with high school or less education (HRs: 0.62(0.59-0.65) vs 0.57(0.54-0.59)). In contrast, Arem et al. reported no interaction of education and LTPA for mortality risks[20]. In our study, the detrimental associations of low physical activity and high sedentary behaviour were more pronounced in low SES, suggesting that SES may interact with physical activity and sedentary behaviour for mortality and incident CVD risks. This finding supports the vulnerability hypothesis, which suggests unhealthy lifestyles may inflict more harm in low socioeconomic groups[5, 10] and is consistent with studies on other unhealthy behaviours such as smoking[10, 55] and alcohol consumption[56].

We found some gradient of stronger detrimental associations of self-reported MVPA with ACM in low individual-level SES, but the patterns were not clear for incident CVD. Though there was detrimental association of low self-reported MVPA in all SES groups, we found some evidence of more pronounced detrimental association in low SES. For example, participants of low, medium, and high individual-level SES with low MVPA were at 22%, 15% and 14% higher hazard of ACM, respectively, compared to those with high MVPA (with approximately 50% overlap in the 95%CI of the low and high SES). These findings are in line with previous studies that have shown more consistent and stronger detrimental associations of unhealthy lifestyles in low SES than their affluent counterparts[5, 6]. A previous UK Biobank analysis showed a higher mortality risk among those with the least healthy lifestyles in the most deprived fifth compared to the least deprived one (HR: 2.47 (2.04-3.00) vs 1.65 (1.25–2.19))[5]. Besides a higher prevalence of unhealthy lifestyle factors[5, 56], potential other explanations for these higher ACM hazards in low SES include exposure to chronic stressors, poor access to information, lower levels of social support[4, 5, 58], and limited health literacy. Targeted primary prevention interventions aimed at increasing physical activity in low SES groups may partly address socioeconomic inequalities in health. Combining approaches such as 'high-risk strategy' (focusing on those who are physically inactive and/or highly sedentary) and 'vulnerable population approach' (focusing on lower SES groups) might be useful[59].

Regarding device-measured physical activity, our results showed higher ACM and incident CVD risk of low device-measured MVPA, and these associations were accentuated with decreasing area-level SES. Effect modification by individual-level SES was less clear for device-measured physical activity, where the high SES group had more pronounced detrimental associations with incident CVD. The differential findings between self-reported and device-measured physical activity exposures may be due to differential measurement properties of the two approaches[60], the selective nature of self-reported physical activity instruments (e.g. capturing bouts lasting at least 10 continuous minutes and mostly LTPA), and a weaker correlation of these two measurement approaches in low-SES[30, 31].

The socioeconomic patterning of the physical activity domains-mortality association was unclear. Our findings are in agreement with a previous study[20], which found no statistically significant interaction (p=0.090) by education in the LTPA-mortality associations. At the same time, it contradicts another study[57] that reported stronger beneficial associations of LTPA with mortality among those with higher education. These inconsistencies in the literature highlight the complex role of SES in physical activity domains-outcome associations and suggest the need for future research to better understand the interaction effects of SES and any underlying mechanisms. We observed no association of LTPA and household physical activity with incident CVD across SES groups (for both individual- and area-level SES), which could partly be due to the lack of overall association between these domains and incident CVD in our study (Table S5). For sedentary behaviour, we found detrimental associations of high screen time with ACM, and these associations became stronger with decreasing area-level SES. In contrast, the effect modification of SES on the associations of sitting time with both outcomes were less clear.

Our results indicated variability in the interaction effects based on the SES measure used. SES patterns were clearer for individual-level SES (self-reported MVPA) and for area-level SES (device-measured MVPA). A possible explanation is that area level SES is more reflective of total movement as captured by accelerometry while individual level SES reflects better leisure time PA, which is what questionnaires capture mostly. Previous studies have also shown mixed results depending upon the SES measure used. Foster et al.[5], in their previous UK Biobank analysis, reported a higher disproportionate risk of a least healthy lifestyle on ACM in low individual- and area-level SES[5]. In contrast, Zhang et. al.[6] reported stronger lifestyles-mortality associations for individual-level SES than that for area-level SES and attributed this to less sensitivity of postcode-derived SES to social causes of health, individual differences, confusion with environmental health determinants and low reliability for heterogenous and mobile communities[6]. However, area-level SES might also contribute to health inequalities through differential access to material resources (physical activity infrastructures, health facilities, etc.), crime, overcrowding and differences in individual-level SES (e.g., limited access to quality schools)[23]. Our findings further add nuance to the literature and highlight the complex role of SES in health behaviours-outcome associations. Taken together, interventions targeting physical inactivity and high sedentary behaviour in low SES groups (individual-level and area-level) might provide the greatest return. We recommend incorporating both individual- and area-level SES measures in future studies to better understand this relationship.

Strengths and limitations

To our knowledge, this is the first study examining the interaction effect of area- and individual-level socioeconomic indices and domain-specific physical activity and sedentary behaviour with ACM and incident CVD using both self-reported and device-measured data. Using two SES indicators (individual and area-level) provided a comprehensive understanding of possible interaction effects. We accounted for competing risks using a sub-distribution hazard model and excluded underweight participants and those with poor self-rated health with possible undiagnosed, subclinical conditions. E-values indicated that it is less likely that the associations we observed are due to unmeasured confounding.

UK Biobank has a low response rate (5.5%) and a higher prevalence of affluent participants of white ethnic background than the general UK population[61]. However, recent evidence shows that physical activity estimates of long-term health outcomes (including ACM and CVD mortality) are not materially affected by poor representativeness and low response rates[62]. Possible misreporting of physical activity participation[31] and covariates between high and low SES might have affected our results. Greater misreporting of physical activity participation in low SES participants[31] might have attenuated the associations, suggesting possibility of even stronger real associations. Despite extensive measures we took (excluding participants with poor self-rated health, prevalent CVD, or an event (death or CVD event) within up to three years of recruitment), reverse causality is still a possibility and this study's observational nature limits inferences about causality.

CONCLUSION

Compared to higher SES groups, low SES groups showed modest evidence of more pronounced inverse associations of MVPA with all-cause mortality and incident CVD, and direct association of screen time with all-cause mortality. Our results suggested some variability in the interaction effects based on the SES and physical activity measures we tested.

We observed consistent and clear interactions of individual-level SES in the association of self-reported MVPA with all-cause mortality. In comparison, area-level SES showed some evidence of interactions in the associations of device-measured MVPA with both outcomes and of screen time with all-cause mortality. Results were less clear for physical activity-domains and device-measured sitting time. Public health interventions targeting physical activity and sedentary behaviour might need to focus on both low SES individuals as well as low SES areas for greater returns. Further research is needed to establish this evidence and better understand the mechanisms underlying these findings.

DECLARATIONS

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Figure titles and legends:

Figure 1: Association of physical activity with all-cause mortality across individual-level socioeconomic status

Small squares denote point estimates of the hazard ratio, and the bars indicate 95% confidence interval.
Reference: High physical activity. Y axis is in log-scale.
SES= Socioeconomic status, IPAQ_MVPA: Self-reported moderate vigorous physical activity (MVPA), ACCEL_MVPA: Device-measured MVPA, LTPA: Leisure-time physical activity
Individual-level SES was created using latent class analysis of three socioeconomic factors (household income, education, and employment status) and categorised into low, medium, and high.
IPAQ_MVPA: Participants physical activity measured using the International Physical Activity Questionnaire was categorised as low (< 600 metabolic equivalent (MET)-min/week), medium (600 to < 3000 MET-min/week), and high (≥ 3000 MET-min/week).
Low SES: High IPAQ_MVPA (2,882/32,501), Medium IPAQ_MVPA (2,751/30,856;1.01 (0.96-1.06)), Low IPAQ_MVPA (997/9,208; 1.22 (1.13-1.31))
Medium SES: High (2,088/46,120), Medium (2,447/53,386; 1.01 (0.96-1.08)), Low (892/17,658; 1.15(1.06-1.24))
High SES: High (1,280/34,432), Medium (2,426/66,521; 1.04(0.97-1.11)), Low (784/19,817; 1.14(1.05-1.25))

ACCEL_MVPA: Device-measured total physical activity was measured using the Axivity AX3 triaxial accelerometer worn on participant's dominant wrist for a 7-day period. Total minutes spent on MVPA (a sum of moderate and vigorous activities) was extracted and categorised into tertile-based thirds. 'Low' indicated the first tertile, 'Medium' indicated second tertile and 'High' indicated third tertile.
Low SES: High ACCEL_MVPA (70/2,695), Medium ACCEL_MVPA (109/2,884; 1.30(0.95-1.77)), Low ACCEL_MVPA (194/3,407; 1.80(1.33-2.43)
Medium SES: High (103/6,461), Medium (129/6,521; 1.03(0.79-1.35)), Low (211/6,275; 1.47(1.13-1.91))
High SES: High (121/9,330), Medium (142/8,699; 1.10(0.85-1.41)), Low (229/7,726; 1.67(1.27-2.08))

LTPA was calculated using the frequency and duration of walking for pleasure, other exercises, and strenuous sports in the last 4 weeks and categorised into tertile-based thirds.
Low SES: High LTPA (1,811 /21,186), Medium LTPA (1,816/ 20,970; 1.08(1.01-1.15)), Low LTPA (2,041/ 22,726; 1.14(1.07-1.22))
Medium SES: High (1,430/ 33,481), Medium (1,606/ 35,277; 1.12(1.04-1.20)), Low (1,671/ 36,695; 1.17(1.09-1.25))
High SES: High (1,365/ 39,621), Medium (1,428/ 39,546; 1.07(0.99-1.16)), Low (1,252/ 33,835; 1.13(1.05-1.22))

Household PA was assessed by asking participants the frequency and duration of light and heavy do-it-yourself activities in the last four weeks and categorised into tertile-based thirds.
Low SES: High household physical activity (1,419/ 15,351), Medium household physical activity (1,323/14,910; 1.04(0.95-1.14)), Low household physical activity (1,578/ 16,931; 1.09(1.00-1.19))
Medium SES: High (1,349/ 26,268), Medium (1,266/ 27,675; 1.02(0.94-1.11)), Low (1,185/ 26,505; 1.05(0.96-1.14))
High SES: High (1,153/ 27,564), Medium (1,175/ 31,809; 1.04(0.96-1.14)), Low (1,006/ 28,341; 1.09(0.99-1.19))

All analyses were adjusted for sex, ethnicity, sleep score, dietary pattern score, smoking and alcohol consumption. IPAQ_MVPA and LTPA analyses were additionally adjusted for screen time (derived using daily hours of TV viewing and non-occupational computer use), ACCEL_MVPA for device-measured sitting time and household physical activity analyses for LTPA and screen time.

Figure 2: Association of sedentary behaviour with all-cause mortality across individual-level socioeconomic status

Small squares denote point estimates of the hazard ratio, and the bars indicate 95% confidence interval.
'Low' indicated the first tertile, 'Medium' indicated second tertile and 'High' indicated third tertile.
Reference: Lowest/first tertile, Y axis is in log-scale.
SES= Socioeconomic status, ,
Individual-level SES was created using latent class analysis of three socioeconomic factors (household income, education, and employment status) and categorised into low, medium, and high.
Sitting time: Device-measured sitting time was measured using the Axivity AX3 triaxial accelerometer worn on participant's dominant wrist for a 7-day period. Total minutes of sitting time was extracted and categorised into tertile-based thirds.
Low SES: Low sitting time (79/2,735), Medium sitting time (107/2,820; 1.03(0.77-1.39)), High sitting time (187/3,431; 1.15(0.86-1.53))
Medium SES: Low (101/7,026), Medium (140/6,340; 1.19 (0.91-1.55)), High (202/5,891; 1.33(1.02-1.73))
High SES: Low (114/8,394), Medium (146/8,918; 0.97 (0.76-1.25)), High (232/8,443; 1.18 (0.92-1.51))

Screen time: Screen time was derived using daily hours spent watching TV and non-occupational and categorised into tertile-based thirds.
Low SES: Low screen time (2,488/30,194), Medium screen time (1,628/17,857; 1.01(0.94-1.08)), High screen time (3,493/33,018; 1.10(1.04-1.17))
Medium SES: Low (2,278/55,968), Medium (1,401/ 29,164; 1.04(0.97-1.12)), High (2,096/ 38,388; 1.04(0.98-1.11))
High SES: Low (2,251/ 71,359), Medium (984/23,852; 1.09(1.01-1.18)), High (1,404/28,317; 1.19(1.11-1.28))

All analyses were adjusted for sex, ethnicity, sleep score, dietary pattern score, smoking and alcohol consumption. Sitting time analyses were additionally adjusted for device-measured MVPA and screen time analyses for self-reported MVPA.

Figure 3: Association of physical activity with incident CVD across individual-level socioeconomic status

Small squares denote point estimates of the sub-hazard ratio, and the bars indicate 95% confidence interval. Reference: High physical activity, Y axis is in log-scale.
SES= Socioeconomic status, IPAQ_MVPA: Self-reported moderate vigorous physical activity (MVPA), ACCEL_MVPA: Device-measured MVPA, LTPA: Leisure-time physical activity
Individual-level SES was created using latent class analysis of three socioeconomic factors (household income, education, and employment status) and categorised into low, medium, and high.
IPAQ_MVPA: Participants physical activity measured using the International Physical Activity Questionnaire was categorised as low (< 600 metabolic equivalent (MET)-min/week), medium (600 to < 3000 MET-min/week), and high (≥ 3000 MET-min/week).
Low SES: High IPAQ_MVPA (9,612/29,796), Medium IPAQ_MVPA (9,155/28,142; 1.01(0.98-1.04)), Low IPAQ_MVPA (2,672/8,276; 1.00(0.95-1.04))
Medium SES: High (13,948/43,887), Medium (15,994/50,705; 0.99(0.97-1.01)), Low (5,375/16,783;1.00 (0.97-1.03))
High SES: High (10,658/33,107), Medium (20,215/64,038; 0.97(0.95-0.99)), Low (6,083/19,105; 0.97(0.94-1.00))

ACCEL_MVPA: Device-measured total physical activity was measured using the Axivity AX3 triaxial accelerometer worn on participant's dominant wrist for a 7-day period. Total minutes spent on MVPA (a sum of moderate and vigorous activities) was extracted and categorised into tertile-based thirds. 'Low' indicated the first tertile, 'Medium' indicated second tertile and 'High' indicated third tertile.
Low SES: High ACCEL_MVPA (617/2,534), Medium ACCEL_MVPA (794/2,686; 1.11(0.97-1.28)), Low ACCEL_MVPA (1,045/3,099; 1.13(0.99-1.28))
Medium SES: High (1,281/6,234), Medium (1,023/6,238; 1.09(0.99-1.19)), Low (1,693/5,942; 1.14(1.04-1.25))
High SES: High (5,942/9,080), Medium (1,633/8,402; 1.13(1.04-1.23)), Low (1,723/7,366; 1.15(1.06-1.26))

LTPA was calculated using the frequency and duration of walking for pleasure, other exercises, and strenuous sports in the last 4 weeks and categorised into tertile-based thirds.
Low SES: High LTPA (6,360/19,384), Medium LTPA (6,198/19,180; 0.98(0.95-1.02)), Low LTPA (6,581/20,802; 0.96(0.93-0.99))
Medium SES: High (10,034/31,895), Medium (10,712/33,502; 1.01(0.98-1.04)), Low (11,114/34,965; 1.00(0.98-1.03))
High SES: High (12,152/38,123), Medium (12,158/38,075; 0.99(0.97-1.02)), Low (10,339/32,599; 0.98(0.96-1.01))

Household PA was assessed by asking participants the frequency and duration of light and heavy do-it-yourself activities in the last four weeks and categorised into tertile-based thirds.
Low SES: High household physical activity (4,576/14,106), Medium High household physical activity (4,377/13,657; 0.98(0.94-1.03)), Low High household physical activity (5,024/15,399; 1.01(0.96-1.05))
Medium SES: High (7,880/24,847), Medium (8,378/26,283; 0.99(0.96-1.02)), Low (7,979/25,241; 0.99(0.96-1.02))
High SES: High (8,364/26,384), Medium (9,666/30,612; 0.99(0.96-1.02)), Low (8,780/27,288; 1.02(0.99-1.05))

All analyses were adjusted for sex, ethnicity, sleep score, dietary pattern score, smoking and alcohol consumption. IPAQ_MVPA and LTPA analyses were additionally adjusted for screen time (derived using daily hours of TV viewing and non-occupational computer use), ACCEL_MVPA for device-measured sitting time and household physical activity analyses for LTPA and screen time. Deaths due to other causes were treated as competing risks.