

# Deaths potentially averted by small changes in physical activity and sedentary time: an individual participant data meta-analysis of prospective cohort studies



Ulf Ekelund, Jakob Tarp, Ding Ding, Miguel Adriano Sanchez-Lastra, Knut Eirik Dalene, Sigmund A Anderssen, Jostein Steene-Johannessen, Bjarne H Hansen, Bente Morseth, Laila A Hopstock, Edvard Sagelv, Peter Nordström, Anna Nordström, Maria Hagströmer, Ing-Mari Dohrn, Keith M Diaz, Steven Hooker, Virginia J Howard, I-Min Lee, Morten W Fagerland

## Summary

**Background** The effects of small, realistic changes in physical activity and sedentary behaviour on population-level mortality are unclear. We aimed to estimate the proportion of deaths preventable by 5-min and 10-min incremental increases in moderate-to-vigorous intensity physical activity (MVPA) and 30-min and 60-min reductions in daily sedentary time.

**Methods** We did an individual participant data meta-analysis of prospective cohort studies. We included studies with device-measured physical activity and sedentary time. We estimated the proportion of deaths prevented (potential impact fractions; PIFs) by changes in (1) the approximately 20% least active participants (high-risk approach) and (2) all participants except the approximately 20% most active (population-based approach). We calculated PIFs from adjusted hazard ratios estimated for 5-min and 10-min increases in MVPA and 30-min and 60-min reductions in sedentary time from observed levels across the activity distribution.

**Findings** We included seven cohorts from Norway, Sweden, and the USA ( $n=40\,327$ ; 4895 deaths). Data from the UK Biobank ( $n=94\,719$ ; 3487 deaths) were analysed separately. A 5-min/day increase in MVPA in the least active participants might prevent 6.0% (95% CI 4.3–7.4) of all deaths. A similar increase in MVPA in all participants except the most active might prevent 10.0% (6.3–13.4) of all deaths. Reducing sedentary time by 30 min/day might prevent 3.0% (2.0–4.1) of all deaths in the high-risk approach and 7.3% (4.8–9.6) in the population-based approach. Results from the UK Biobank were of a smaller magnitude but still substantial—eg, reducing sedentary time by 30 min/day in all except the most active participants was associated with preventing 4.5% (2.8–6.1) of total deaths.

**Interpretation** Small and realistic increases in MVPA of 5 min/day might prevent up to 6% of all deaths in a high-risk approach and 10% of all deaths in population-based approach. Reducing sedentary time by 30 min/day might prevent a smaller, but still meaningful, proportion of deaths in the two risk scenarios.

**Funding** None.

**Copyright** © 2026 Elsevier Ltd. All rights reserved, including those for text and data mining, AI training, and similar technologies.

## Introduction

Physical inactivity is estimated to cause as much as 7–9% of global mortality.<sup>1–3</sup> However, these estimates are based on self-reported physical activity, which is known to be inaccurate and underestimate the magnitude of the physical activity mortality association compared with device-measured physical activity.<sup>4</sup> The benefits of physical activity on population mortality might therefore also be underestimated. Data from devices have expanded the understanding of the health benefits of physical activity from focusing solely on moderate-to-vigorous intensity physical activity (MVPA) to suggest that sedentary time,<sup>5</sup> total physical activity,<sup>5,6</sup> and all intensities of physical activity<sup>5–8</sup> are associated with reduced mortality. However, the effect on population mortality has not been estimated.

Estimates of preventable deaths are usually based on calculating the population attributable fraction (PAF),

which represents the theoretical number of deaths averted if all individuals not meeting WHO's physical activity recommendations started meeting these recommendations.<sup>9</sup> This dichotomisation of physical activity for PAF estimates assumes that health benefits can only be achieved by changing from not meeting to meeting physical activity recommendations, which might be unrealistic for many. These studies also did not consider the population distribution of physical activity and how varying increases in population-level physical activity could affect estimates of preventable deaths. Consequently, it is unclear how small but arguably more realistically achievable population-level increases in physical activity (ie, shifting the population distribution more favourably) affect the proportion of preventable deaths.

Therefore, we estimated the proportion of deaths preventable by 5-min and 10-min incremental increases

Published Online  
January 13, 2026  
[https://doi.org/10.1016/S0140-6736\(25\)02219-6](https://doi.org/10.1016/S0140-6736(25)02219-6)

Oslo Research Centre for Physical Activity and Population Health (ORC-PAPH), Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway (Prof U Ekelund PhD, J Tarp PhD, M A Sanchez-Lastra PhD, Prof S A Anderssen PhD, Prof J Steene-Johannessen PhD, Prof B H Hansen PhD, M W Fagerland PhD); Department of Chronic Diseases, Norwegian Institute of Public Health, Oslo, Norway (Prof U Ekelund, K E Dalene PhD); Prevention Research Collaboration, Sydney School of Public Health (Prof D Ding PhD), Charles Perkins Centre (Prof D Ding), The University of Sydney, Camperdown, NSW, Australia; Department of Special Didactics, Faculty of Education and Sports Sciences, University of Vigo, Pontevedra, Spain (M A Sanchez-Lastra); Well-Move Research Group, Galicia-Sur Health Research Institute (SERGAS-UVIGO), Vigo, Spain (M A Sanchez-Lastra); School of Sport Sciences, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway (Prof B Morseth PhD, E Sagelv PhD, Prof A Nordström MD); Department of Health and Care Sciences, UiT The Arctic University of Norway, Tromsø, Norway (Prof L A Hopstock PhD); Department of Public Health and Caring Sciences, Clinical Geriatrics, Uppsala University, Uppsala, Sweden (Prof P Nordström MD); Division of Physiotherapy, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Stockholm, Sweden

(Prof A Nordström, Prof M Hagströmer PhD, I-M Dohrn PhD); Academic Primary Health Care Centre, Region Stockholm, Stockholm, Sweden (Prof M Hagströmer); Department of Medicine, Columbia University Medical Center, New York, NY, USA (K M Diaz PhD); College of Health and Human Services, San Diego State University, San Diego, CA, USA (Prof S Hooker PhD); Department of Epidemiology, School of Public Health, University of Alabama at Birmingham, Birmingham, AL, USA (Prof V J Howard PhD); Department of Epidemiology, Harvard T H Chan School of Public Health, Boston, MA, USA (Prof I-M Lee ScD); Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA (Prof I-M Lee); Oslo Centre for Biostatistics and Epidemiology, Research Support Services, Oslo University Hospital, Oslo, Norway (M W Fagerland)

Correspondence to: Prof Ulf Ekelund, Oslo Research Centre for Physical Activity and Population Health (ORC-PAPH), Department of Sports Medicine, Norwegian School of Sport Sciences, 0806 Oslo, Norway [ulfek@nih.no](mailto:ulfek@nih.no)

See Online for appendix

## Research in context

### Evidence before this study

Estimates of the population attributable fraction, which represent the theoretical number of deaths averted if all individuals currently not meeting WHO's physical activity recommendations became active by meeting recommendations, have several major methodological limitations. These include overlooking the health benefits that can be achieved by increasing physical activity even below the WHO-recommended level, and relying on self-reported physical activity data, which are prone to biases. We searched PubMed and Google Scholar from database inception to Aug 15, 2024, using the terms "physical activity", "mortality", and "population attributable fraction" and found two articles estimating the number of deaths that are potentially averted by increasing levels of device-measured physical activity. One study was restricted to individuals with prevalent cardiovascular diseases, and the other used a small sample from a single US cohort. Therefore, it remains uncertain how minor, yet potentially more achievable, population-level increases in physical activity or decreases in sedentary time might affect the proportion of preventable deaths.

### Added value of this study

Our results are derived from the observed physical activity levels and the non-linear dose-response associations between

physical activity and sedentary time with risk of mortality. This approach extends previous observations that were based on a theoretical elimination of physical inactivity estimated from self-reported data. Access to individual participant data allowed us to use continuous minute-by-minute, device-measured physical activity and sedentary time data, harmonising exposures, covariates, and statistical analyses across cohorts, using consistent inclusion criteria, and minimised loss of information and power.

### Implications of all the available evidence

Considering that it is unlikely for all individuals to achieve the WHO physical activity recommendation of 150 min of moderate-to-vigorous-intensity physical activity (MVPA) weekly, our data underscore the large impact of realistic and achievable behaviour changes on population health. If the least active 80% of participants had increased MVPA by 5 min/day or reduced sedentary time by 30 min/day, 10% and 7% of all deaths, respectively, might have been avoided during the follow-up period.

in MVPA and 30-min and 60-min reductions in daily sedentary time (potential impact fractions [PIFs]). Additionally, we estimated the proportions of preventable deaths associated with 30-min and 60-min increases in total physical activity and in light-intensity physical activity (LPA). Our analytical approach is based on the observed device-measured physical activity levels in large population-based cohorts and considers the non-linear, dose-response associations between physical activity and sedentary time and the risk of mortality.

## Methods

### Study design

We previously systematically searched the literature for prospective cohort studies in which data on physical activity and sedentary time, measured using hip-worn accelerometers, could be linked to mortality records, leading to the establishment of the Adult Accelerometer Consortium.<sup>5,10,11</sup> Five (Activity, Behaviour, Change [ABC], Sweden; the National Health and Nutrition Examination Survey [NHANES], USA; Norwegian National Physical Activity Survey [NNPAS], Norway; the Reasons for Geographical and Racial Differences in Stroke [REGARDS] study, USA; and the Women's Health Study [WHS], USA) of the nine cohorts from the consortium contributed to this individual-level participant data analysis. Following an updated search, we identified and included two additional cohorts (Healthy Aging Initiative [HAI], Sweden, and the Tromsø Study, Norway), which are now

part of the consortium.<sup>11</sup> Altogether, anonymised individual-level participant data from three studies from the USA, two studies from Norway, and two studies from Sweden were pooled. To obtain a more homogeneous sample, we excluded participants younger than 40 years from those cohorts that included younger participants (NHANES, NNPAS, and ABC). Further, to reduce bias due to reverse causation, participants who died during the first 2 years of follow-up (n=556) were excluded. We accounted for the cluster-based sampling and used sample weights provided by the NHANES to generate estimates and measures of uncertainty representative of the civilian non-institutionalised resident population of the USA.<sup>12</sup> The study selection, data extraction, and bias assessment have been described in detail.<sup>5,10,11</sup> Sample sizes, exclusions, and missing data are shown in the appendix (pp 2–3).

Further, we included data from the UK Biobank Study (application number 29717). In the UK Biobank Study, physical activity was assessed in 103712 participants by wrist-worn accelerometers. Of these, 96651 provided valid accelerometer data, of which we included 94719 participants following exclusions of those dying during the first 2 years of follow-up (n=557) or with missing data (appendix p 2). The UK Biobank Study was included to assess transportability of findings from the Adult Accelerometer Consortium (hip-worn devices) to data generated in the UK Biobank Study using wrist-worn devices and alternative data-processing procedures. Different placements and device-wearing protocols

prohibit direct harmonisation of UK Biobank Study accelerometer data with that from the Adult Accelerometer Consortium.

Ethics approval was granted for all individual studies but was not required for this individual participant data meta-analysis. Ethics approval for the UK Biobank Study was obtained from the Northwest Research Ethics Committee and all participants provided informed consent.

The reporting of this study followed STROBE guidelines (appendix pp 8–10).

### Assessment of physical activity exposures

The harmonisation of physical activity exposure variables from the seven studies included in the accelerometer consortium has previously been described in detail<sup>5,10,11</sup> and is summarised in the appendix (pp 6–7).

Physical activity in the UK Biobank Study was assessed using wrist-worn AX3 accelerometers (Axivity, York, UK) for 24 h per day over 7 consecutive days. Following previously applied procedures for the UK Biobank Study,<sup>13</sup> we included all participants with a wear time of more than 72 h (including sleep). The time spent sedentary (minutes per day), in LPA, and in MVPA was assessed using a validated machine learning method.<sup>14,15</sup>

Total physical activity was calculated as the time spent in LPA plus the time spent in MVPA.

### Covariates

Covariates were selected on the basis of previous knowledge of variables associated with exposures and outcomes and availability in the cohorts. Height and weight were self-reported in three studies (ABC, NNPAS, and WHS) and measured during a clinical examination in the remaining five studies. We calculated BMI and defined obesity as a BMI of at least 30 kg/m<sup>2</sup>. Smoking habits, education, and history of cardiovascular disease, cancer, and diabetes were either self-reported and validated from medical records or obtained from registers. We harmonised the data across cohorts for smoking and education, with smoking categorised as never, former, or current, and the highest attained education level categorised as primary school, high school, or university. In the UK Biobank Study, covariates were assessed a median of 5.7 years before the assessment of physical activity. Height and weight were measured during the clinical examination. Smoking habits, education, and history of chronic diseases were self-reported by a computerised questionnaire. All covariates in the UK Biobank Study were categorised

	ABC (n=719), Sweden*		HAI (n=4271), Sweden†		NHANES (n=3871), USA‡		REGARDS (n=7276), USA§	
	Women	Men	Women	Men	Women	Men	Women	Men
Participants	399 (55.5%)	320 (44.5%)	2172 (50.9%)	2099 (49.1%)	1958 (50.6%)	1913 (49.4%)	3964 (54.5%)	3312 (45.5%)
Deaths	31 (7.8%)	47 (14.7%)	33 (1.5%)	58 (2.8%)	524 (26.8%)	617 (32.3%)	729 (18.4%)	918 (27.7%)
Follow-up time, years	14.3 (1.7)	13.9 (2.3)	3.8 (1.9)	3.9 (1.9)	13.5 (3.3)	12.9 (3.8)	9.9 (3.2)	9.6 (3.3)
Age, years	54.6 (9.1)	55.5 (9.1)	70.4 (0.2)	70.4 (0.3)	60.6 (12.9)	60.5 (12.9)	68.2 (8.7)	69.8 (8.3)
Age ≥60 years	117 (29.3%)	103 (32.2%)	2172 (100%)	2099 (100%)	1020 (52.1%)	984 (51.4%)	3298 (83.2%)	2945 (88.9%)
Sedentary time, min/day	509 (96.1)	529 (103)	550 (82.7)	563 (85.8)	498 (112)	511 (125)	693 (82.2)	689 (83.6)
Total physical activity, min/day	369 (101)	365 (110)	293 (74.5)	284 (78.6)	341 (101)	343 (111)	148 (71.8)	161 (76.4)
Light-intensity physical activity, min/day	340 (89.9)	330 (96.9)	263 (68.4)	249 (71.9)	327 (94.2)	320 (99.6)	141 (66.8)	151 (69.9)
Moderate-to-vigorous intensity physical activity, min/day	29.6 (31.6)	35.0 (30.6)	30.4 (24.0)	34.4 (26.4)	13.9 (16.5)	23.6 (24.8)	6.7 (11.6)	10.0 (15.2)
BMI, kg/m <sup>2</sup>	25.6 (3.9)	26.0 (2.9)	26.3 (4.6)	26.7 (3.7)	29.1 (6.6)	28.3 (5.3)	28.8 (6.3)	28.3 (4.7)
BMI ≥30 kg/m <sup>2</sup>	51 (12.8%)	30 (9.4%)	403 (18.6%)	351 (16.7%)	735 (37.5%)	575 (30.1%)	1453 (36.7%)	968 (29.2%)
Smoking								
Never	164 (41.1%)	126 (39.4%)	1077 (49.6%)	959 (45.7%)	1159 (59.2%)	681 (35.6%)	2272 (57.3%)	1350 (40.8%)
Former	121 (30.3%)	131 (40.9%)	934 (43.0%)	1016 (48.4%)	497 (25.4%)	808 (42.2%)	1277 (32.2%)	1619 (48.9%)
Current	114 (28.6%)	63 (19.7%)	161 (7.4%)	124 (5.9%)	302 (15.4%)	424 (22.2%)	415 (10.5%)	343 (10.4%)
Education								
Primary	120 (30.1%)	117 (36.6%)	266 (12.2%)	391 (18.6%)	529 (27.0%)	577 (30.2%)	255 (6.4%)	172 (5.2%)
High school	145 (36.3%)	117 (36.6%)	890 (41.0%)	831 (39.6%)	515 (26.3%)	440 (23.0%)	2069 (52.2%)	1489 (45.0%)
University	134 (33.6%)	86 (26.9%)	1016 (46.8%)	877 (41.8%)	914 (46.7%)	896 (46.8%)	1640 (41.4%)	1651 (49.8%)
History of cardiovascular disease¶	6 (1.5%)	22 (6.9%)	120 (5.5%)	333 (15.9%)	255 (13.0%)	339 (17.7%)	336 (8.5%)	553 (16.7%)
History of cancer¶	12 (3.0%)	8 (2.5%)	816 (37.6%)	481 (22.9%)	255 (13.0%)	216 (11.3%)	222 (12.1%)	314 (14.5%)
Diabetes¶	9 (2.3%)	11 (3.4%)	130 (6.0%)	226 (10.8%)	269 (13.7%)	271 (14.2%)	505 (12.7%)	482 (14.6%)

Data are n (%) or mean (SD). ABC=Activity, Behaviour, Change. HAI=Healthy Aging Initiative. NHANES=National Health and Nutrition Examination Survey. REGARDS=Reasons for Geographical and Racial Differences in Stroke. \*ActiGraph 7164 (lower back). †ActiGraph GT3X+ (right hip). ‡ActiGraph 7164 (right hip). §Actual (right hip). ¶Binary variables (yes or no). ||Percentages calculated out of 1836 women and 2172 men with non-missing observations.

Table 1: Descriptive characteristics of the ABC, HAI, NHANES, and REGARDS cohorts

	WHS (n=16 316), USA*		NNPAS (n=2208), Norway†		Tromsø study (5666), Norway‡		UK Biobank Study (94 719), UK§	
	Women	Men	Women	Men	Women	Men	Women	Men
Participants	16 316 (100%)	0	1168 (52.9%)	1040 (47.1%)	3025 (53.4%)	2641 (46.6%)	53 449 (56.4%)	41 270 (43.6%)
Deaths	1584 (9.7%)	..	37 (3.2%)	77 (7.4%)	97 (3.2%)	143 (5.4%)	1414 (2.6%)	2073 (5.0%)
Follow-up time, years	8.8 (1.6)	..	9.0 (0.7)	8.8 (1.0)	7.1 (0.5)	7.0 (0.7)	8.0 (0.8)	7.9 (0.9)
Age, years	72.0 (5.7)	..	55.8 (11.0)	57.1 (10.7)	62.9 (10.3)	63.4 (10.1)	61.8 (7.7)	63.0 (7.9)
Age ≥60 years	16 316 (100%)	..	415 (35.5%)	418 (40.2%)	2066 (68.3%)	1895 (71.8%)	32 209 (60.3%)	27 327 (66.2%)
Sedentary time, min/day	503 (103)	..	548 (83.2)	579 (86.8)	714 (82.0)	727 (89.6)	547 (104)	586 (111)
Total physical activity, min/day	388 (101)	..	337 (83.0)	314 (88.4)	327 (83.1)	312 (88.8)	360 (102)	325 (105)
Light-intensity physical activity, min/day	353 (87.3)	..	303 (75.7)	277 (80.6)	299 (75.7)	280 (78.5)	325 (95.9)	276 (94.7)
Moderate-to-vigorous intensity physical activity, min/day	35.3 (29.8)	..	33.7 (23.4)	37.2 (26.5)	27.9 (22.0)	31.4 (25.4)	35.2 (30.0)	49.6 (38.5)
BMI, kg/m <sup>2</sup>	26.2 (5.0)	..	25.1 (4.2)	26.4 (3.4)	26.8 (4.7)	27.7 (3.9)	26.3 (4.8)	27.3 (4.0)
BMI ≥30 kg/m <sup>2</sup>	3186 (19.5%)	..	136 (11.6%)	136 (13.1%)	643 (21.3%)	623 (23.6%)	9721 (18.2%)	8602 (20.8%)
Smoking								
Never	8242 (50.5%)	..	535 (45.8%)	451 (43.4%)	1222 (40.4%)	1002 (37.9%)	32 602 (61.0%)	21 602 (52.3%)
Former	7506 (46.0%)	..	397 (34.0%)	426 (41.0%)	1421 (47.0%)	1350 (51.1%)	17 713 (33.1%)	16 307 (39.5%)
Current	568 (3.5%)	..	236 (20.2%)	163 (15.7%)	382 (12.6%)	289 (10.9%)	3134 (5.9%)	3361 (8.1%)
Education								
Primary	0	..	199 (17.0%)	185 (17.8%)	932 (30.8%)	678 (25.7%)	4229 (7.9%)	3598 (8.7%)
High school	8105 (49.7%)	..	443 (37.9%)	408 (39.2%)	800 (26.4%)	801 (30.3%)	26 544 (49.7%)	19 152 (46.4%)
University	8211 (50.3%)	..	526 (45.0%)	447 (43.0%)	1293 (42.7%)	1162 (44.0%)	22 676 (42.4%)	18 520 (44.9%)
History of cardiovascular disease¶	680 (4.2%)	..	55 (4.7%)	118 (11.3%)	206 (6.8%)	403 (15.3%)	1690 (3.2%)	3529 (8.6%)
History of cancer¶	1931 (11.8%)	..	76 (6.5%)	68 (6.5%)	300 (9.9%)	286 (10.8%)	7565 (14.2%)	4119 (10.0%)
Diabetes¶	1482 (9.1%)	..	35 (3.0%)	65 (6.3%)	164 (5.4%)	184 (7.0%)	1283 (2.4%)	1963 (4.8%)

Data are n (%) or mean (SD). NNPAS=Norwegian National Physical Activity Survey. WHS=Women's Health Study. \*ActiGraph GT3X+ (right hip). †ActiGraph GT1M (right hip). ‡ActiGraph wGT3X-BT (right hip). §Activity AX3 (wrist). ¶Binary variables (yes or no).

Table 2: Descriptive characteristics of the WHS, NNPAS, Tromsø study, and UK Biobank Study cohorts

identically to those in the other cohorts (appendix pp 4–5).

**Outcome ascertainment**

Mortality was ascertained using medical records, death certificates, or administrative linkage following study specific procedures (appendix p 4).

**Statistical analysis**

A detailed description of our analytical procedures can be found in the appendix (pp 11–13). We used a two-stage approach for the individual-level participant data meta-analyses. First, each cohort was analysed individually using MVPA, sedentary time, total physical activity, and LPA as exposure variables in separate models. Cubic splines with four knots were created for each exposure variable, with knot locations at percentiles 5, 35, 65, and 95 of the exposure distributions. A Cox proportional hazard regression model was fitted to the spline variables, with mortality as the outcome and follow-up time as the underlying time scale. We adjusted our models for the following potential and available confounders: (1) age, sex, and accelerometer wear time (adjusted for age and sex); and (2) additional adjustments for BMI, smoking, education, cardiovascular disease, cancer, and diabetes (fully adjusted). Because our models included adjustment

for wear time, the estimated association for higher MVPA reflects both the effect of higher MVPA and the effect of some combination of lower sedentary time and light activity. Similarly, associations for lower sedentary time reflect associations for increases in both light physical activity and MVPA. Based on the fitted models, hazard ratios (HRs) with 95% CIs were estimated for increases in physical activity and reductions in sedentary time from observed levels for each exposure. HRs were estimated for 5-min and 10-min daily increases in MVPA; 30-min and 60-min daily increases in LPA and total physical activity; and 30-min and 60-min daily reductions in sedentary time. For example, for a person engaging in 1 min of MVPA per day, we estimated the effect of a hypothetical addition of 5 min per day (ie, from 1 min to 6 min). HRs below 1 indicate a lower mortality risk for increased physical activity (or decreased sedentary time). For each set of HR and CI, we calculated the PIFs with 95% CIs, which provide a measure of the proportion of mortality that could be prevented by changing the exposure level.<sup>16</sup> Next, cumulative PIFs were calculated to mimic hypothetical public health interventions affecting the population distribution of physical activity for (1) the approximately 20% least active participants (high-risk approach) and (2) all participants except the approximately 20% most active (population-based approach), calculated

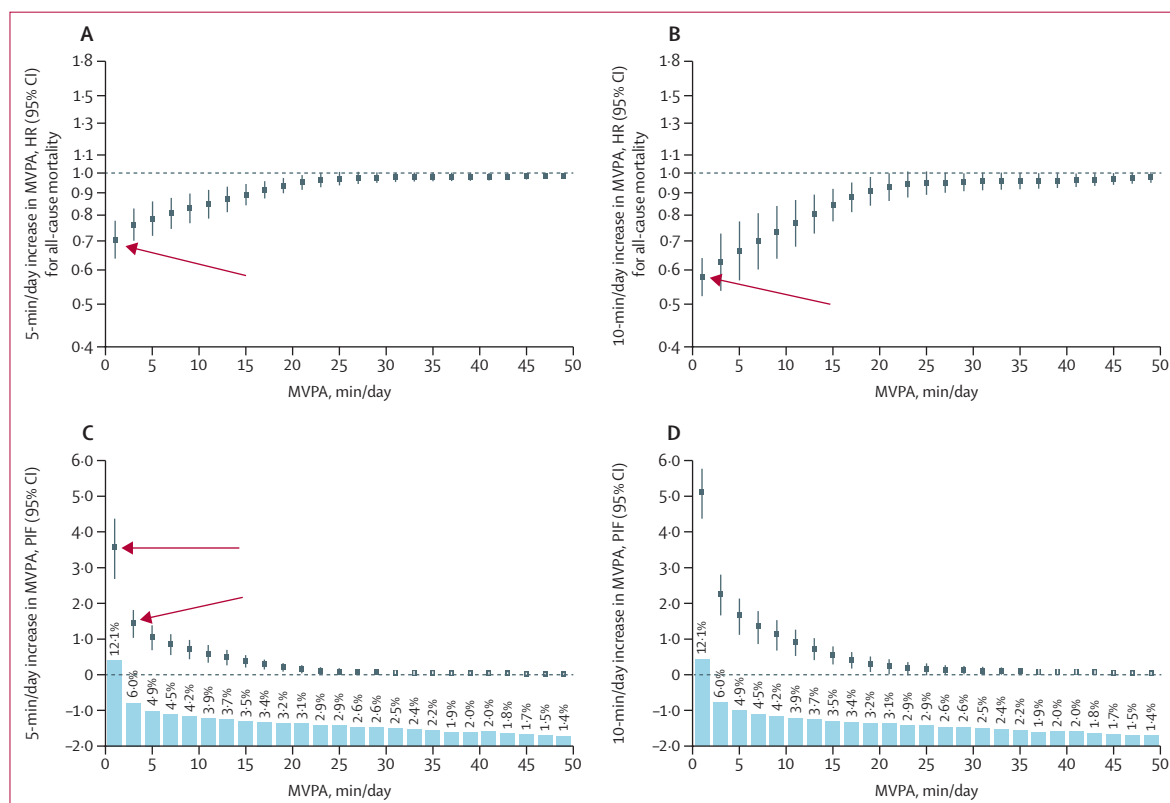
separately for each exposure variable. These contrasts were conceptually informed by Rose's distinction between high-risk and population-based prevention strategies<sup>17</sup> and defined to reflect the shape of the dose-response associations of physical activity and sedentary time with mortality risk (large initial risk reduction with diminishing returns at higher volumes of activity and lower volumes of sedentary time) while maintaining distributional similarity between the individual-level participant data meta-analysis and the UK Biobank Study.

Second, summary HRs with 95% CIs were estimated with a restricted maximum likelihood random-effects model. On the basis of the summary HRs, summary measure PIFs and cumulative PIFs (with 95% CIs) were also calculated. The REGARDS cohort was only included in the stage-two meta-analyses for MVPA because outputs from Actigraph and Actical accelerometers are only comparable for time spent in MVPA.<sup>18</sup>

### Sensitivity analyses

Since age and other demographic factors influence physical activity behaviours and mortality risk, we

stratified our sample by sex, age (<60 years vs ≥60 years), and geographical location (USA vs Scandinavia) in sensitivity analyses. We further reanalysed our data (1) without adjustment for baseline BMI (ie, treating BMI as a mediator instead of a confounder), (2) excluding participants with self-reported mobility limitations in the cohorts (NHANES, and Tromsø, and the UK Biobank Study) where this information was available (to further reduce the possibility of reverse causality), (3) excluding participants with self-reported chronic diseases, and (4) did a one-stage individual-level participant data meta-analysis. Finally, to compare results with previously published studies, we stratified participants into meeting or not meeting the existing WHO physical activity recommendations<sup>9</sup> (operationalised as MVPA ≥22 min/day vs <22 min/day, closely equivalent to the deflection point for the dose-response curve for the association between MVPA and risk for mortality<sup>3</sup>) and estimated the adjusted PAF—ie, the proportion of deaths averted if all participants met the recommendation, using the adjusted HRs derived from the included studies. All sensitivity analyses were performed using



**Figure 1: MVPA and mortality risk in the accelerometer consortium**

Figure shows results of meta-analysis of seven studies. HRs for mortality for 5-min/day (eg, the HR for a 5-min/day increase from 1 min/day to 6 min/day [red arrow]; A) and 10-min/day (eg, the HR for a 10-min/day increase from 1 min/day to 11 min/day [red arrow]; B) increases in MVPA from observed level. Percentage PIF for 5-min/day (eg, a 5-min/day increase in MVPA in those who accumulated ≤2 min/day of MVPA was associated with 3.6% of preventable deaths [red arrow], and a 5-min increase among those accumulating 2–4 min of MVPA per day was associated with 1.4% of preventable deaths [red arrow]; C) and 10-min/day increase in MVPA from observed level (D). Bar charts show distribution and percentages of participants. All results are based on fully adjusted models (age, sex, accelerometer wear-time, BMI, smoking, education, cardiovascular disease, cancer, and diabetes). HR=hazard ratio. MVPA=moderate-to-vigorous-intensity physical activity. PIF=population impact fraction.

MVPA. The statistical analyses were done with Stata/SE 17.0, including the package xblc.<sup>19</sup>

**Role of the funding source**

There was no funding for this study.

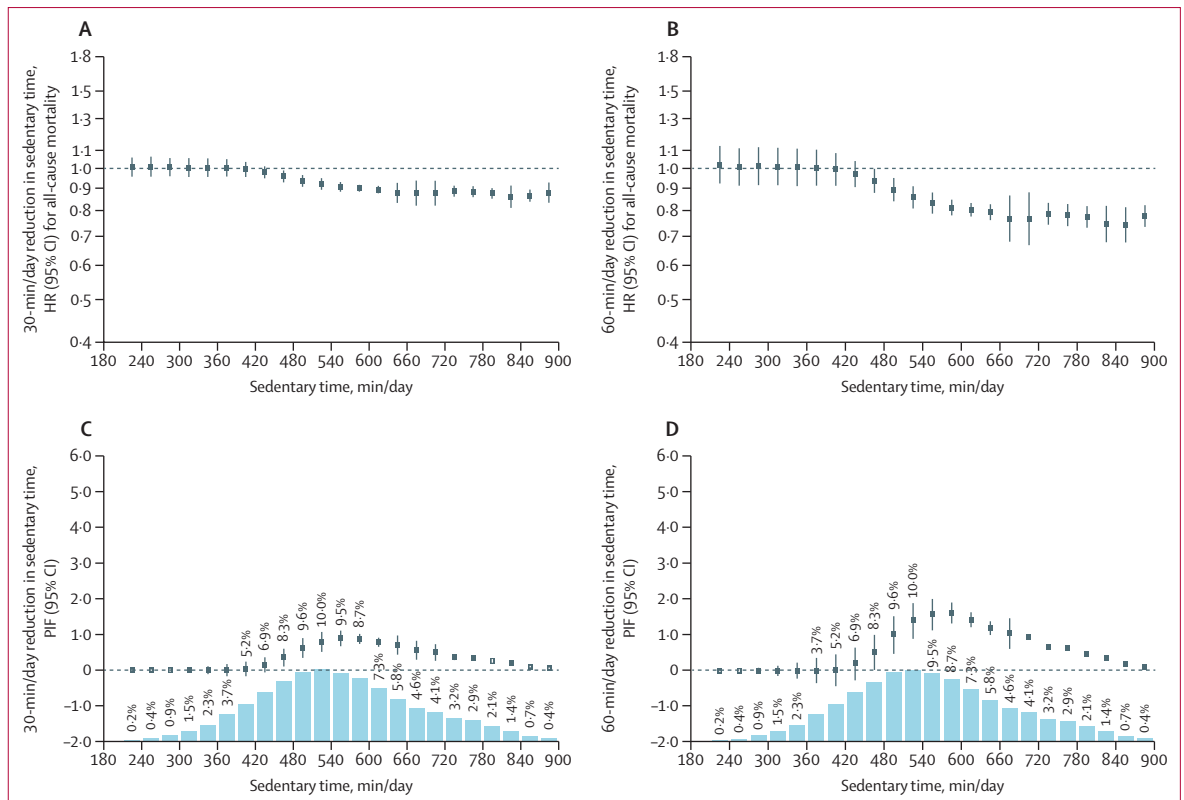
**Results**

We included 135 046 participants (mean age 63.9 years [SD 8.7]; 82 451 [61%] women and 52 595 [39%] men) followed up for a mean of 8.2 years (SD 1.9; tables 1, 2). The appendix shows the distributions (min/day) for MVPA, sedentary time, and total physical activity, by cohort (pp 14–16), and the distribution by cohort and age category (p 3).

Participants in the individual-level participant data meta-analysis (n=40 327; 4895 deaths) spent on average 27.7 min/day (3.1% of wear time) in MVPA. Results from minimally adjusted models are shown in the appendix (pp 17–18). Figure 1 shows the estimated relative changes in risk for mortality associated with 5-min and 10-min increases in MVPA from observed values across the MVPA distribution in the individual-level participant data meta-analysis. For example, a 5-minute increase in MVPA from 1 min/day to 6 min/day

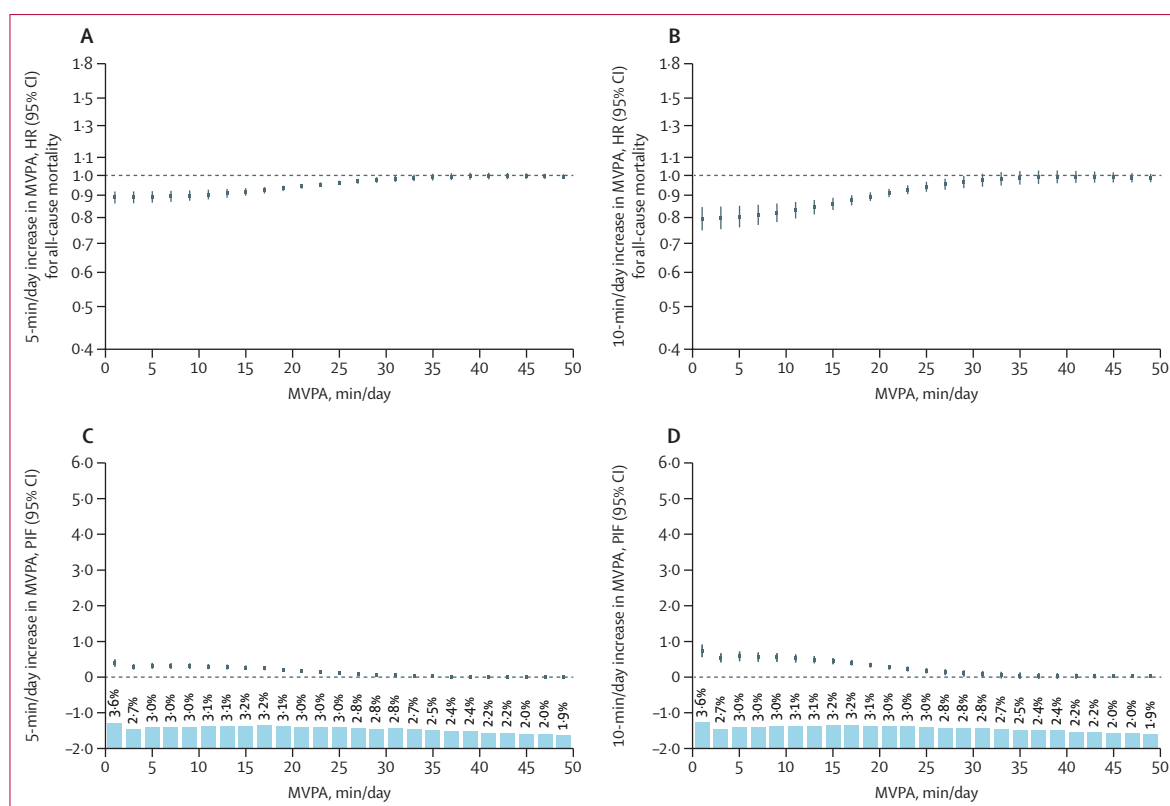
(figure 1A) was associated with an approximately 30% lower mortality risk and a 10-min/day increase from 1 min/day to 11 min/day (figure 1B) with an approximately 42% lower mortality risk. We found diminishing returns on increasing MVPA at higher levels—eg, increasing MVPA from 9 min/day to 14 min/day was associated with an approximately 18% lower mortality risk and from 9 min/day to 19 min/day with an approximately 28% lower mortality risk. Beyond approximately 24 min/day, no clear risk reduction was evident (figure 1A).

We calculated PIFs for 5-min (figure 1C) and 10-minute (figure 1D) increases in MVPA from observed levels (appendix pp 11–12). For example, a 5-min increase in MVPA per day in the least active 12.1% (ie, those who accumulated ≤2 minutes of MVPA per day; n=4865) was associated with 3.6% (95% CI 2.7–4.4) of deaths averted. A 5-min increase among those accumulating 2–4 min of MVPA per day (2438 [6.0%] participants) was associated with 1.4% (1.0–1.8) of deaths averted. The results for the individual-level participant data meta-analysis of 30-min/day and 60-min/day increases in LPA and total physical activity (HRs and PIFs) are shown in the appendix (pp 19–20, 23–24).



**Figure 2: Sedentary time and mortality risk in the accelerometer consortium**

Figure shows results of meta-analysis of six studies. HRs for mortality for 30-min/day (A) and 60-min/day (B) decrease in sedentary time from observed level. Percentage PIF for 30-min/day (C) and 60 min/day (D) decrease in sedentary time from observed level. Bar charts show distribution and percentages of participants. All results are based on fully adjusted models (age, sex, accelerometer wear-time, BMI, smoking, education, cardiovascular disease, cancer, and diabetes). HR=hazard ratio. MVPA=moderate-to-vigorous-intensity physical activity. PIF=population impact fraction.



**Figure 3: MVPA and mortality risk in the UK Biobank Study**

HRs for mortality for 5-min/day (A) and 10-min/day (B) increase in MVPA from observed level. Percentage PIF for 5-min/day (C) and 10-min/day (D) increase in MVPA from observed level. Bar charts show distribution and percentages of participants. All results are based on fully adjusted models (age, sex, accelerometer wear-time, BMI, smoking, education, cardiovascular disease, cancer, and diabetes). HR=hazard ratio. MVPA=moderate-to-vigorous-intensity physical activity. PIF=population impact fraction.

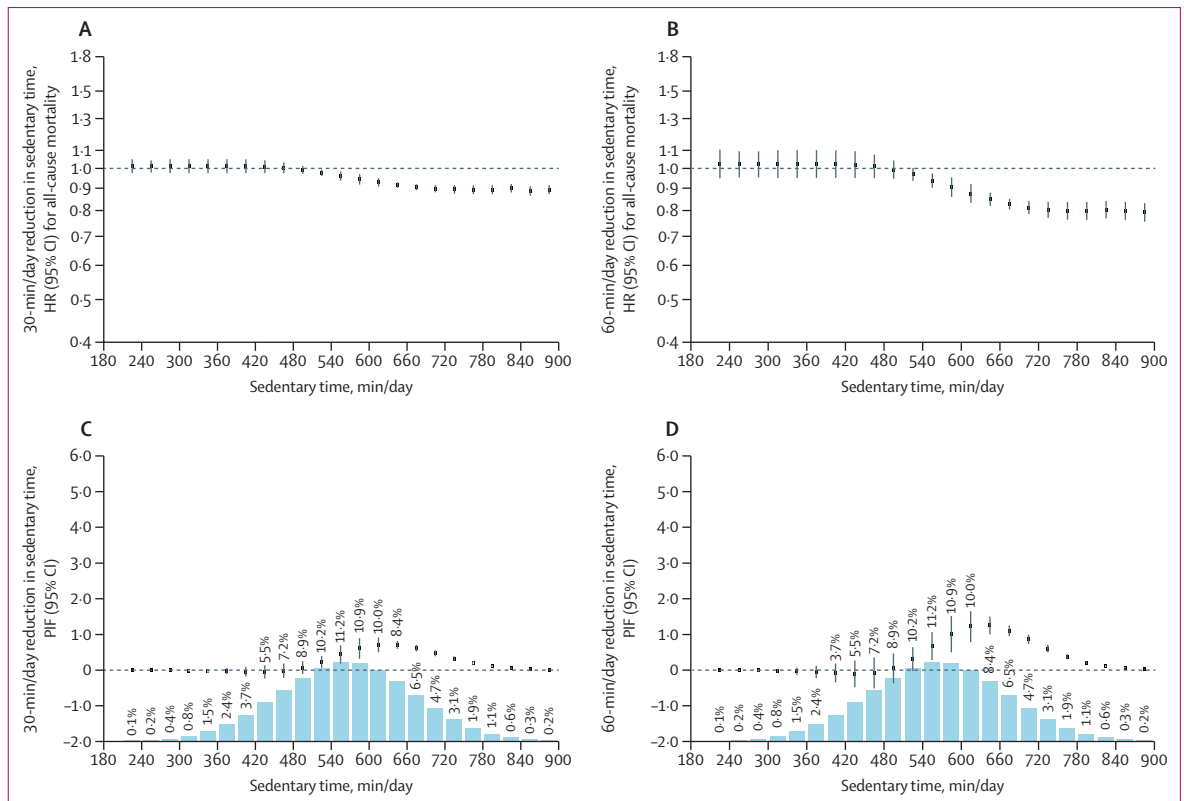
Participants in the individual-level participant data meta-analysis spent an average of 577 min/day (64.5% of wear time) sedentary. We estimated the associations between 30-min (figure 2A) and 60-min (figure 2B) daily reductions in sedentary time and risk for mortality. There was no association between reducing sedentary time and the risk of mortality in those spending less than approximately 480 min/day sedentary (figure 2). By contrast, reducing sedentary time in those who spent more than 480 min sedentary per day, comprising more than 70% of the participants, was associated with a progressively lower risk. For example, in those accumulating at least 660 min/day of sedentary time, reducing sedentary time by 30 min/day was associated with risk reductions of approximately 10%, and reducing sedentary time by 60 min/day was associated with risk reductions of approximately 25% (figure 2C, D). Minimally adjusted results for sedentary time are shown in the appendix (pp 21–22).

Associations and PIFs for higher MVPA and lower sedentary time from the UK Biobank Study ( $n=94719$ ; 3487 deaths) were smaller in comparison (figures 3, 4).

We thereafter estimated the proportion of deaths averted by calculating the cumulative PIFs (appendix

pp 11–12) for defined increases in MVPA, LPA, and total physical activity, and reductions in sedentary time for a high-risk approach and population-based approach (table 3). By targeting the least active participants who spent on average 2.2 min/day in MVPA (high-risk approach), we estimated that 6.0% (95% CI 4.3–7.4) of total deaths in the population could be averted through increasing MVPA by 5 min/day and 8.8% (7.0–10.4) by 10 min/day in this group. By targeting all but the most active 20% (population-based approach), who spent on average 17.4 minutes per day in MVPA, 5-min/day increases in MVPA would prevent 10.0% (6.3–13.4) of deaths in the population and 10-min/day increases in MVPA would prevent 14.9% (9.7–19.3) of deaths in the population (table 2). The corresponding proportion of deaths prevented in the UK Biobank Study was lower, ranging from 2.2% to 6.2% (table 2).

Based on the high-risk approach (approximately 20% of the most sedentary participants, accumulating an average of 721 min/day sedentary), the estimated proportion of mortality that is preventable by reducing sedentary time by 30 min/day was 3.0% (95% CI 2.0–4.1) and by 60 min/day was 5.5% (3.9–6.9); based on the population-based approach (all but approximately the 20% least



**Figure 4: Sedentary time and mortality risk in the UK Biobank Study**  
 HRs for mortality for 30-min/day (A) and 60-min/day (B) decrease in sedentary time from observed level. Percentage PIF for 30-min/day (C) and 60-min/day (D) decrease in sedentary time from observed level. Bar charts show distribution and percentages of participants. All results are based on fully adjusted models (age, sex, accelerometer wear-time, BMI, smoking, education, cardiovascular disease, cancer, and diabetes). HR=hazard ratio. PIF=population impact fraction.

sedentary, accumulating an average of 605 min/day sedentary), the estimated proportion of mortality preventable by reducing sedentary time by 30 min/day was 7.3% (4.8–9.6) and by 60 min/day was 12.6% (8.4–16.4). Corresponding preventable proportions in the UK Biobank Study were between 2.5% (2.1–2.9) and 7.6% (4.4–10.6; table 2). Finally, the estimated proportions of mortality preventable by 60-min/day increases in LPA and total physical activity were roughly similar to the estimates for 5-min increases in MVPA (table 2).

In sensitivity analyses (appendix pp 25–39), results were similar in men and women, but there was little evidence for any meaningful association between increases in MVPA and the risk of mortality in those younger than 60 years; however, statistical power was low because of small numbers (five studies; n=6479; 257 deaths). The results were similar when BMI was excluded from the models, across geographical locations, and when excluding participants with chronic diseases (appendix pp 25–29). We repeated the sensitivity analyses in the UK Biobank Study (sex and age stratified; appendix pp 30–31) and without adjustment for BMI, excluding those with mobility limitations and those with chronic diseases (appendix p 32), and the results were materially

unchanged. We thereafter excluded participants with mobility limitations in the three cohorts (NHANES, Tromsø, and the UK Biobank Study) where this information was available and reanalysed the data. For example, in NHANES, PIFs were attenuated by 1.5 absolute percentage points to 7.3% (95% CI 5.0–9.3) for a 5-min increase in MVPA in the high-risk approach, and by 2.0 absolute percentage points to 11.5% (5.4–16.7) in the population-based approach (appendix pp 33–38).

We also reanalysed the data using a one-stage individual-level participant data meta-analysis, and the results were not materially different from our main results (appendix p 39).

In the adjusted PAF (appendix p 40) scenario, 21.5% (95% CI 18.7–24.0) of all deaths could hypothetically be prevented if all participants spent at least 22 min/day in MVPA, which is equivalent to the minimal level of the current WHO physical activity recommendation of 150 min of MVPA per week (appendix p 40). The corresponding PAF from the UK Biobank Study was 9.2% (7.4–10.9). HRs and PIFs for 5-min and 10-min increases in MVPA from each individual cohort are available in the appendix (p 40).

## Discussion

Our individual-level participant data meta-analyses of seven cohort studies from the USA and Scandinavia illustrated the benefits associated with small increases in physical activity below the recommended target. Our findings suggest that a hypothetical high-risk and population-based intervention resulting in 5-min daily increases in MVPA might reduce mortality in the population by approximately 6.0% and 10.0%, respectively. A 30-min reduction in daily sedentary time might prevent 3.0% of total mortality in the high-risk approach and 7.3% in the population-based approach. The estimated proportions of preventable deaths due to the same increases in MVPA and the reductions in sedentary time in the UK Biobank Study were lower, but still substantial in magnitude.

Reducing sedentary time by 30 min/day, equivalent to 5% of total sedentary time, in our population-based approach, was associated with the prevention of more than 7% of all deaths during follow-up. This 30-min change appears feasible in a real-world setting. Effective interventions to reduce occupational sitting might reduce sitting time by between 40 min and 100 min per 8-h workday.<sup>20</sup> Similarly, interventions aimed to change sedentary behaviour in community-dwelling older adults might reduce sedentary time by 45 min/day.<sup>21</sup> However, in both the high-risk and the population-based approaches, the estimated number of preventable deaths appears greater with increasing MVPA by 5–10 min/day compared with 30-min and 60-min reductions in sedentary time. A 12-week walking intervention in 45–75-year-olds increased time in MVPA by 30–35 min per week,<sup>22</sup> which was sustained after 3 years.<sup>23</sup>

A previous study<sup>24</sup> using accelerometer data from NHANES estimated that adding 10, 20, and 30 min of MVPA per day was associated with a 6.9%, 13.0%, and 16.9% decrease in the number of annual deaths, respectively. Differences between studies might be explained by different analytical approaches, differences in exclusion criteria (eg, age and mobility limitations), and differences in minimum wear time criteria (1 day in the previous study *vs*  $\geq 3$  days in our analyses) yielding different sample sizes. Finally, because of different definitions of MVPA, the average time spent in MVPA was 3.5 times higher in the 2022 NHANES study<sup>24</sup> compared with the data from NHANES included in this study, which might also contribute to the observed differences. Unfortunately, there is no consensus on how to define MVPA from hip accelerometry. Our definition of MVPA (approximately 2000 counts per minute; CPM) corresponds to walking equivalent to around 4–5 km/h (appendix pp 6–7), which is probably the most common type of moderate intensity physical activity in the general population. When we calculated the PIF for a 10-min increase in MVPA using the same intensity threshold as in the earlier study,<sup>24</sup> we estimated that 5.5% of deaths might be preventable in the NHANES cohort. A direct

	High-risk approach*	Population approach†
<b>Moderate-to-vigorous intensity physical activity, 5-min/day increase</b>		
Accelerometer consortium	6.0% (4.3–7.4)	10.0% (6.3–13.4)
UK Biobank Study	2.2% (1.6–2.7)	3.6% (2.4–4.8)
<b>Moderate-to-vigorous intensity physical activity, 10-min/day increase</b>		
Accelerometer consortium	8.8% (7.0–10.4)	14.9% (9.7–19.3)
UK Biobank Study	3.9% (3.0–4.7)	6.2% (4.0–8.2)
<b>Light-intensity physical activity, 30-min/day increase</b>		
Accelerometer consortium	3.3% (2.4–4.1)	5.4% (2.2–8.3)
UK Biobank Study	2.8% (2.3–3.2)	4.0% (2.2–5.7)
<b>Light-intensity physical activity, 60-min/day increase</b>		
Accelerometer consortium	5.5% (4.0–6.7)	8.9% (3.1–13.8)
UK Biobank Study	4.7% (3.9–5.3)	6.0% (2.7–9.2)
<b>Sedentary time, 30-min/day decrease</b>		
Accelerometer consortium	3.0% (2.0–4.1)	7.3% (4.8–9.6)
UK Biobank Study	2.5% (2.1–2.9)	4.5% (2.8–6.1)
<b>Sedentary time, 60-min/day decrease</b>		
Accelerometer consortium	5.5% (3.9–6.9)	12.6% (8.4–16.4)
UK Biobank Study	4.6% (3.8–5.3)	7.6% (4.4–10.6)
<b>Total physical activity, 30-min/day increase</b>		
Accelerometer consortium	3.4% (2.5–4.2)	6.1% (3.1–8.9)
UK Biobank Study	2.5% (2.2–2.9)	4.5% (2.8–6.1)
<b>Total physical activity, 60-min/day increase</b>		
Accelerometer consortium	5.5% (4.1–6.8)	10.6% (5.7–14.9)
UK Biobank Study	4.4% (3.8–5.0)	7.2% (4.0–10.1)

Results are based on meta-analysis of seven (or six; the REGARDS study is only included in the meta-analysis for moderate-to-vigorous intensity physical activity) studies in the accelerometer consortium and a separate analysis of the UK Biobank Study. PIF=potential impact fraction. REGARDS=Reasons for Geographical and Racial Differences in Stroke. \*Cumulative PIF calculated for the approximately 20% least active participants. †Cumulative PIF calculated for all participants except the approximately 20% most active participants.

**Table 3: Cumulative PIF (95% CI) for changes in moderate and vigorous physical activity, light physical activity, sedentary time, and total physical activity**

comparison between the two definitions of MVPA in our dataset (appendix pp 41–42) yielded even larger differences in the estimated percentage of averted deaths, suggesting that results can vary depending on the definition used for MVPA.

Our approach has several strengths. Analyses are based on small and realistic changes in device-measured physical activity and sedentary time and take into account the non-linear shape of the association between activity levels and mortality. The estimated effects of increases in physical activity and reductions in sedentary time are preferable compared with earlier work based on theoretical elimination of physical inactivity,<sup>1,3</sup> as it is unrealistic for all individuals to be able or willing to meet the recommended 150 min of MVPA per week. Similarly, the elimination approach assumes that the health benefits of changing from inactive to active are uniform for all individuals irrespective of their observed activity level, despite the established curve-linear relationships (eg, increasing MVPA from 0 min/week to 30 min/week is associated with significantly more benefits than increasing from 100 min/week to 130 min/week<sup>5</sup>). We

used accelerometer data for our exposures, which are less biased than data from self-reports. Access to individual-level participant data allowed us to use continuous exposure data; harmonise exposures, covariates, and statistical analyses across cohorts using consistent inclusion criteria; and is likely to have minimised loss of information and power.<sup>25</sup> The sample size of our meta-analysis allowed us to conduct several sensitivity analyses, and these results suggest that our main results are robust. However, our findings are limited to participants older than 40 years.

Our results should also be interpreted with the following limitations. We analysed data from Scandinavia, the UK, and the USA, limiting the generalisability beyond these high-income populations. Five of seven cohorts from the consortium were designed to be population representative (ABC, HAI, NHANES, NNPAS, and Tromsø). Reassuringly, the shape of the HRs by observed physical activity levels and the associated PIFs from these cohorts were similar to the results from our meta-analysis combining data from all seven cohorts. The UK Biobank Study is not representative of its source population, and there is evidence of healthy volunteer selection bias,<sup>26</sup> which might be accentuated in the subgroup with accelerometer measurements.<sup>27</sup>

Further, the intensity thresholds (CPM) used for defining light intensity and MVPA were derived from standardised ambulatory activities, which might not mirror all physical activity performed during everyday life. Our indices of physical activity and sedentary time from the UK Biobank Study are not directly comparable to those from the accelerometer consortium. Differences include monitor placement, brands, and algorithms used to define intensity levels. Wrist-worn accelerometers produce higher acceleration signals and thus higher levels of physical activity during everyday conditions compared with waist-worn or hip-worn accelerometers.<sup>28,29</sup> These differences, and differences in definitions of MVPA, probably affect the magnitude of associations between increases in physical activity and risk for mortality and thus the estimated PIFs.

Residual confounding might exist, due to unmeasured or poorly measured confounders. For example, data on mobility limitations were not available in all cohorts. The results were somewhat attenuated (around 15–20% lower) in sensitivity analyses when excluding individuals with mobility limitations. Combined with our exclusion of deaths within the first 2 years of follow-up, this finding reduces the likelihood of bias from reverse causation. However, as in any observational research we cannot fully rule out the possibility that our results reflect some bias from residual or unmeasured confounding and reverse causation. Exposure variables were only measured at baseline; thus, MVPA might not reflect sustained activity levels over time, weakening the consistency assumption and potentially affecting the associations.

Our results can inform public health programmes and policies by illustrating the potentially substantial effects on population health from small, feasible improvements in exposure variables if the population distribution is shifted. A previous individual-level participant data meta-analysis<sup>30</sup> from randomised trials on pharmacological blood pressure lowering suggested that a 5-mm Hg pharmacologically induced reduction in systolic blood pressure reduced the hazard of fatal and non-fatal cardiovascular disease events by 10% in those with and without previous diagnoses of cardiovascular disease, and even at normal or high-normal blood pressure values. A direct comparison between a pharmacological trial and our observational individual-level participant data should be made with caution. However, our findings suggest a 30% reduction in the hazard of all-cause mortality for a 5-min/day increase in MVPA at the lower end of the activity spectrum, equivalent to 3·5% (95% CI 2·8–4·4) of total preventable deaths.

We only investigated all-cause mortality; thus, future research should examine other health outcomes. Additional research using device-measured physical activity is needed in low-income and middle-income countries where the age structure, physical activity levels, and disease burden differ from those included in the present study.

In conclusion, under hypothetical high-risk and population-based intervention scenarios, small and realistic increases in MVPA of 5 min/day might prevent up to 6% and 10% of deaths during follow-up, respectively. Reducing sedentary time by 30 min/day prevented a smaller, but still meaningful, proportion of deaths in the two risk scenarios.

#### Contributors

UE, MWF, and JT contributed to study conception and design. All authors contributed to acquisition of data and funding for individual cohorts. UE, JT, DD, KED, MAS-L, I-ML, and MWF contributed to data interpretation. UE and JT drafted the manuscript. UE and MWF accessed and verified the data. MWF and MAS-L contributed to the statistical analysis. All authors contributed to critical revision of the manuscript for important intellectual content and gave final approval.

#### Declaration of interests

VJH declares payments made to their institution from the National Institute of Neurological Disorders and Stroke (NINDS) and the National Institute on Ageing (NIA). All other authors declare no competing interests.

#### Data sharing

The data underlying this Article were shared by individual cohorts under individual data sharing agreements. Individual study data can be accessed or requested through the following means: Tromsø Study data upon application to the Data and Publication Committee for the Tromsø Study (<https://uit.no/research/tromsostudy>); HAI data upon request to Principal Investigator Prof Anna Nordström ([anna.h.nordstrom@umu.se](mailto:anna.h.nordstrom@umu.se)); NNPAS data upon request to Principal Investigator Prof Sigmund Alfred Anderssen ([sigmundaa@nih.no](mailto:sigmundaa@nih.no)); WHS data upon request to Principal Investigator I-Min Lee ([ileewh.harvard.edu](mailto:ileewh.harvard.edu)); REGARDS data upon request to Virginia Howard ([vjhoward@uab.edu](mailto:vjhoward@uab.edu)); and ABC data upon request to Maria Hagströmer ([maria.hagstromer@ki.se](mailto:maria.hagstromer@ki.se)). UK Biobank Study data are available at <https://www.ukbiobank.ac.uk>. NHANES data are available at <https://www.cdc.gov/nchs/nhanes/>. The codes for our analyses are available on GitHub (<https://github.com/ocbe-uo/deaths-potentially-averted>).

### Acknowledgments

The individual studies contributing to this harmonised meta-analysis were funded from the following sources: the ABC-study was funded by Stockholm County Council, the Swedish National Centre for Research in Sports, and the project ALPHA, which received funding from the European Union in the framework of the Public Health Programme and Folksam Research Foundation, Sweden. The Norwegian National Physical Activity Surveillance Study was supported by the Norwegian Directorate for Public Health and the Norwegian School of Sport Sciences. The REGARDS physical activity study was funded by an investigator-initiated grant (R01-NS061846) from the NINDS of the National Institutes of Health (NIH). The REGARDS parent study was supported by a cooperative agreement (U01-NS041588) co-funded by NINDS and the NIA. Additional funding was provided by an unrestricted research grant from the Coca-Cola Company. The Women's Health Study was funded by NIH grants CA154647, CA047988, CA182913, HL043851, HL080467, and HL099355, and supported in part by the extramural programme at the National Health Institute. The National Center for Health Statistics was not involved in analysing, interpreting, nor necessarily endorses any of the conclusions of the present study. The content is solely the responsibility of the authors.

### References

- Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. *Lancet* 2012; **380**: 219–29.
- Katzmarzyk PT, Friedenreich C, Shiroma EJ, Lee IM. Physical inactivity and non-communicable disease burden in low-income, middle-income and high-income countries. *Br J Sports Med* 2022; **56**: 101–06.
- Strain T, Brage S, Sharp SJ, et al. Use of the prevented fraction for the population to determine deaths averted by existing prevalence of physical activity: a descriptive study. *Lancet Glob Health* 2020; **8**: e920–30.
- Ekelund U, Dalene KE, Tarp J, Lee IM. Physical activity and mortality: what is the dose response and how big is the effect? *Br J Sports Med* 2020; **54**: 1125–26.
- Ekelund U, Tarp J, Steene-Johannessen J, et al. Dose–response associations between accelerometry measured physical activity and sedentary time and all cause mortality: systematic review and harmonised meta-analysis. *BMJ* 2019; **366**: 14570.
- Strain T, Wijndaele K, Dempsey PC, et al. Wearable-device-measured physical activity and future health risk. *Nat Med* 2020; **26**: 1385–91.
- Stamatakis E, Ahmadi MN, Gill JMR, et al. Association of wearable device-measured vigorous intermittent lifestyle physical activity with mortality. *Nat Med* 2022; **28**: 2521–29.
- Lee IM, Shiroma EJ, Evenson KR, Kamada M, LaCroix AZ, Buring JE. Accelerometer-measured physical activity and sedentary behaviour in relation to all-cause mortality: the Women's Health Study. *Circulation* 2018; **137**: 203–05.
- Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. *Br J Sports Med* 2020; **54**: 1451–62.
- Ekelund U, Tarp J, Fagerland MW, et al. Joint associations of accelerometer measured physical activity and sedentary time with all-cause mortality: a harmonised meta-analysis in more than 44 000 middle-aged and older individuals. *Br J Sports Med* 2020; **54**: 1499–506.
- Sagelv EH, Hopstock LA, Morseth B, et al. Device-measured physical activity, sedentary time, and risk of all-cause mortality: an individual participant data analysis of four prospective cohort studies. *Br J Sports Med* 2023; **57**: 1457–63.
- Johnson CL, Paulose-Ram R, Ogden CL, et al. National health and nutrition examination survey: analytic guidelines, 1999–2010. National Center for Health Statistics. *Vital Health Stat* 2013; **2**: 161.
- Doherty A, Jackson D, Hammerla N, et al. Large scale population assessment of physical activity using wrist worn accelerometers: the UK Biobank Study. *PLoS One* 2017; **12**: e0169649.
- Willetts M, Hollowell S, Aslett L, Holmes C, Doherty A. Statistical machine learning of sleep and physical activity phenotypes from sensor data in 96 220 UK Biobank participants. *Sci Rep* 2018; **8**: 7961.
- Walmsley R, Chan S, Smith-Byrne K, et al. Reallocation of time between device-measured movement behaviours and risk of incident cardiovascular disease. *Br J Sports Med* 2021; **56**: 1008–17.
- Barendregt JJ, Veerman JL. Categorical versus continuous risk factors and the calculation of potential impact fractions. *J Epidemiol Community Health* 2010; **64**: 209–12.
- Rose G. Sick individuals and sick populations. *Int J Epidemiol* 2001; **30**: 427–32.
- Duncan S, Stewart T, Bo Schneller M, Godbole S, Cain K, Kerr J. Convergent validity of ActiGraph and actual accelerometers for estimating physical activity in adults. *PLoS One* 2018; **13**: e0198587.
- Orsini N, Greenland S. A procedure to tabulate and plot results after flexible modelling of a quantitative covariate. *Stata J* 2011; **11**: 1–29.
- Nguyen P, Le LKD, Nguyen D, Gao L, Dunstan DW, Moodie M. The effectiveness of sedentary behaviour interventions on sitting time and screen time in children and adults: an umbrella review of systematic reviews. *Int J Behav Nutr Phys Act* 2020; **17**: 117.
- Chastin S, Gardiner PA, Harvey JA, et al. Interventions for reducing sedentary behaviour in community-dwelling older adults. *Cochrane Database Syst Rev* 2021; **6**: CD012784.
- Harris T, Kerry SM, Limb ES, et al. Effect of a primary care walking intervention with and without nurse support on physical activity levels in 45- to 75-year olds: the Pedometer and Consultation Evaluation (PACE-UP) cluster randomised controlled trial. *PLoS Med* 2017; **14**: e1002210.
- Harris T, Kerry SM, Limb ES, et al. Physical activity levels in adults and older adults 3–4 years after pedometer-based walking interventions: long-term follow-up of participants from two randomised controlled trials in UK primary care. *PLoS Med* 2018; **15**: e1002526.
- Saint-Maurice PF, Graubard BI, Troiano RP, et al. Estimated number of deaths prevented through increased physical activity among US adults. *JAMA Intern Med* 2022; **182**: 349–52.
- Riley RD, Lambert PC, Abo-Zaid G. Meta-analysis of individual participant data: rationale, conduct, and reporting. *BMJ* 2010; **340**: c221.
- Fry A, Littlejohns TJ, Sudlow C, et al. Comparison of sociodemographic and health-related characteristics of UK Biobank participants with those of the general population. *Am J Epidemiol* 2017; **186**: 1026–34.
- Dempsey PC, Rowlands AV, Strain T, et al. Physical activity volume, intensity, and incident cardiovascular disease. *Eur Heart J* 2022; **43**: 4789–800.
- Kamada M, Shiroma EJ, Harris TB, Lee IM. Comparison of physical activity assessed using hip- and wrist-worn accelerometers. *Gait Posture* 2016; **44**: 23–28.
- Tudor-Locke C, Barreira TV, Schuna JM Jr. Comparison of step outputs for waist and wrist accelerometer attachment sites. *Med Sci Sports Exerc* 2015; **47**: 839–42.
- Rahimi K, Bidel Z, Nazarzadeh M, et al. Pharmacological blood pressure lowering for primary and secondary prevention of cardiovascular disease across different levels of blood pressure: an individual participant-level data meta-analysis. *Lancet* 2021; **397**: 1625–36.