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Associations of accelerometer-measured light-intensity physical activity with mortality and incidence of cardiovascular diseases and cancers: A prospective cohort study

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Abstract

Background: Although light-intensity physical activity (LPA) has been suggested to be associated with a lower risk of mortality, the minimal and optimal volumes of LPA remain unclear. We aimed to examine the minimal and optimal volumes of LPA associated with the risks of mortality and disease incidence (i.e., cardiovascular diseases and cancer).

Methods: Data were derived from the population-based UK Biobank cohort study, including 69,492 adults aged 43−78 years. Accelerometer-measured LPA was defined using a validated, published machine learning-based Random Forest activity method, which was categorized into four quartile groups. All-cause and cause-specific mortality (cardiovascular disease- and cancer-specific) were determined according to the International Classification of Diseases, 10th version codes. Disease incidence was defined based on primary care, hospitalization, or death records.

Results: During a median follow-up period of 8.04 years, 2024 adults died from all causes, 539 from cardiovascular disease, and 1175 from cancer. For all-cause mortality, compared with participants in the lowest quartile of LPA (<3.9 h/day), the hazard ratios (HRs) and 95% confidence intervals (95% CIs) were 0.82 (95% CI: 0.73–0.93) for those with 3.9–<5.0 h/day, 0.75 (95% CI: 0.66–0.85) for those with 5.0–<6.1 h/day, and 0.77 (95% CI: 0.68–0.88) for those with ≥6.1 h/day, respectively. There was an inverse non-linear dose-response association between LPA and all-cause mortality, with an optimal dose of 5.7 h/day (95% CI: 5.5–6.4; HR = 0.63, 95% CI: 0.56–0.71) and a minimal dose of 3.6 h/day (95% CI: 3.5–8.6; HR = 0.81, 95% CI: 0.78–0.86), with the 5th percentile as the reference. Similar patterns were observed for cause-specific mortality and disease incidence (cardiovascular disease and cancer).

Conclusion: Engaging in LPA for ~3.5 h/day was conservatively associated with lower risk of mortality and disease incidence, with further risk reductions observed up to an optimal dose of ~6.0 h/day. These findings suggest that sufficient LPA offers important health benefits, which can inform the development of future PA guidelines.

Keywords: Light-intensity physical activity; Mortality; Cardiovascular disease; Cancer

1. Introduction

The World Health Organization and American Heart Association guidelines on physical activity (PA) recommended that adults should engage in 150−300 min of moderate-intensity PA weekly (an activity performed at a level between ≥3 and 6

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metabolic equivalents of tasks (METs)), or 75−150 min of vigorous-intensity PA weekly (≥6 METs), or an equivalent combination of both.[1,2] Accordingly, related public health guidelines and most health promotion programs emphasize the necessity for the public to engage in sufficient moderate-to-vigorous-intensity PA (MVPA). Although vigorous PA is time-efficient, it may not be suitable for all adults, especially older adults and those with chronic diseases.[3] The important contribution of light-intensity PA (LPA) (1.6−2.9 METs), which constitutes a major component of daily energy expenditure, has been largely neglected.[4] The promotion of LPA may be a feasible way to increase activity, as it does not require advanced planning or a specific time commitment, and typically includes everyday activities and increased movement during work hours (such as slow walking, low-intensity strolling, standing, cleaning, cooking, and stretching).[5]

Previous evidence has shown the health benefits of LPA, and even 1-min bouts of LPA during prolonged sitting can improve metabolic health.[6] Thirty minutes of objectively measured LPA per day was associated with a 20% reduction in the risk of cardiovascular disease (CVD)-specific mortality and a 14% reduction in the risk of cancer-specific mortality.[7,8] A meta-analysis of six prospective observational studies also showed that spending more time in daily light activity (highest *vs*. lowest amount) reduced the risk of all-cause mortality by 29%.[5] However, prior studies were limited by insufficient statistical power and lacked standardized definitions and operationalization of LPA. The 2018 Physical Activity Guidelines for Americans introduce flexibility regarding "intensity," emphasizing not only moderate-to-vigorous intensity but also the importance of LPA.[9] Similarly, the 2020 World Health Organization guidelines recommended that substituting sedentary behavior with PA of any intensity, even light-intensity, is beneficial for human health.[1] However, these guidelines did not specify the minimal and optimal doses of LPA due to limited evidence available at that time. Understanding this information can offer valuable insights into developing future evidence-based quantitative recommendations on daily LPA.

In this study, we aimed to examine the dose−response associations of device-measured LPA with mortality and disease incidence (i.e., CVD and cancer) in 69,492 adults from the UK Biobank cohort, and to identify the minimal and optimal volumes of LPA associated with reduced risk of mortality and morbidity.

2. Methods

2.1. Study design and sample

We used data from the UK Biobank cohort, which includes 502,629 adults aged 37−73 years who were enrolled between 2006 and 2010.[10] Objective PA measurements were collected from 103,661 participants aged 43−78 years between 2013 and 2015. At enrollment, participants underwent examinations at 1 of 22 assessment centers and were followed up until November 30, 2022. After excluding participants with unusable accelerometer data ($n = 7010$, including those with insufficient wear time (i.e., fewer than 3 valid days, with each hour of the 24-h cycle covered) ($n = 6992$), those whose raw accelerometer data were not well calibrated ($n = 4$), those who had unrealistically high acceleration values ($n = 13$), and those without data on LPA ($n = 1$), those with the diagnosis of CVDs or cancers before accelerometry measurement ($n = 25,928$), and those with missing values for any covariates ($n = 1231$), we finally included 69,492 participants aged 43−78 years with valid wrist accelerometer data (Fig. 1). The basic characteristics of included and excluded participants are shown in

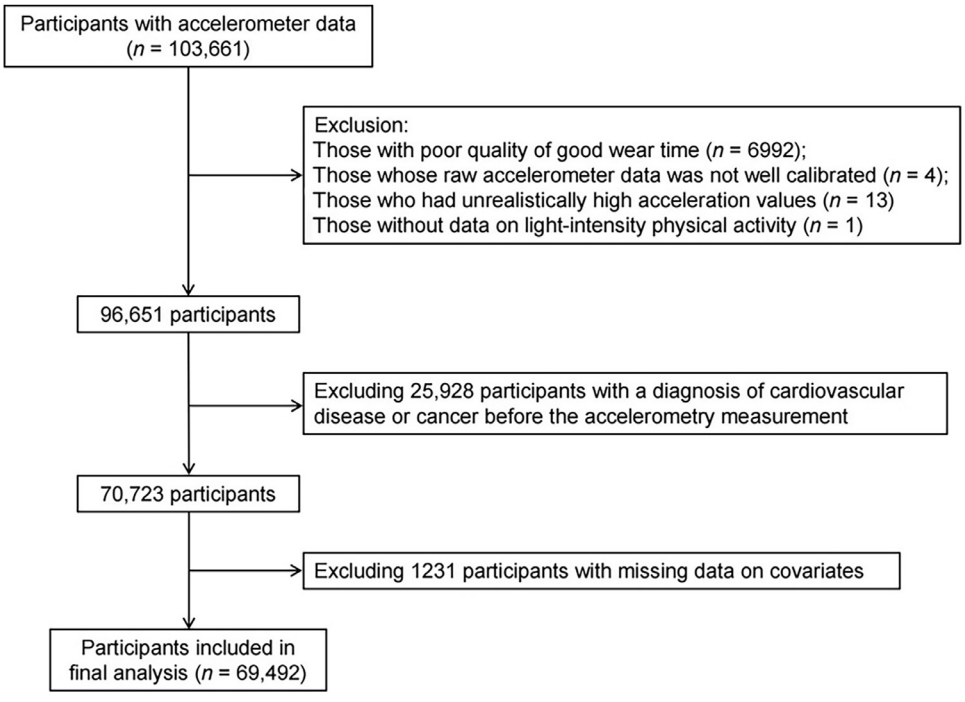

Fig. 1. Flowchart of inclusion/exclusion of study participants.

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Supplementary Table 1. The UK Biobank cohort was reviewed and approved by the North West Multi-Center Research Ethics Committee (R21/NW/0157). All participants provided written informed consent before the baseline assessment.

2.2. Assessment of accelerometer-based LPA

Participants wore an Axivity AX3 wrist-worn triaxial accelerometer (Axivity Ltd., Newcastle upon Tyne, UK) on the dominant wrist for 7 consecutive days, and it recorded continuous acceleration signals calibrated to 100 Hz and a dynamic range of $\pm 8g$.[11] The intensity of PA was identified using a validated, published machine learning-based Random Forest activity method, which includes categories for running, walking, and small utilitarian movements.[12] Detailed descriptions of this method are shown in the Supplementary Method section of Supplementary Materials. For further data analyses, accelerometer-measured LPA was categorized into four groups based on quartiles.

2.3. Morbidity and mortality ascertainment

CVD incidence was identified from primary care, hospital admission, and death records, while cancer incidence was ascertained from cancer registry data, excluding non-melanoma and in situ skin cancers, as well as cancers with ambiguous definitions.[13] In this study, we focused exclusively on a composite cancer outcome that encompassed 13 types of site-specific cancers and 1 type of cancer with multiple sites. We combined all cancer types into a composite to increase the statistical power of our analysis. For mortality, the International Classification of Diseases (ICD), 10th version, was used to code CVD-specific and cancer-specific mortality in the medical record. Specifically, CVD was defined according to the ICD-10 codes (rheumatic heart disease (ICD-10: I01−I09), hypertensive heart disease (I11), hypertensive heart and renal disease (I13), ischemic/pulmonary heart disease and diseases of pulmonary circulation (I20−29), cerebrovascular diseases (I60−69), and other forms of heart disease (I30−51)). The 13 site-specific cancers were coded as C00−C96 (including cancers of the lip, oral cavity, and pharynx (ICD-10: C00−C14), digestive organs (C15−C26), respiratory and intrathoracic organs (C30−C39), bone and articular cartilage (C40−C41), melanoma and other malignant neoplasms of skin (C43−C44), mesothelial and soft tissue (C45−C49), breast (C50−C50), genital organs (C51−C63), urinary tract (C64−C68), eye, brain, and other parts of the central nervous system (C69−C72), thyroid and other endocrine glands (C73−C75), ill-defined, secondary and unspecified sites (C76−C80), stated or presumed to be primary, of lymphoid, hematopoietic, and related tissue (C81−96)). An independent category for cancers of multiple sites was coded as C97.[14] Given the variations in medical visit intervals and follow-up frequency, the first occurrence of CVD or cancer was considered as the endpoint. Therefore, follow-up was terminated for participants on the earliest date of the occurrence of outcomes, death, or the final follow-up on November 30, 2022, whichever occurred first.

2.4. Covariate measurement

Potential covariates in this study were collected from medical history, touch-screen questionnaires, biomedical indices, anthropometric measurements, and accelerometer-measured variables. We adjusted for the following potential confounders: age, sex (males *vs*. females), ethnic/racial groups (White *vs*. non-White), educational attainment (college or university degree, A levels/Advanced Subsidiary (AS) levels or equivalent, O levels/General Certificate of Secondary Education (GCSEs) or equivalent, Certificate of Secondary Education (CSE) or equivalent, National Vocational Qualification (NVQ) or High National Diploma (HND) or High National Certificate (HNC) or equivalent *vs*. others), employment status (employed *vs*. not employed), Townsend Deprivation Index (as a continuous variable), self-reported health (excellent, good, fair *vs*. poor), smoking status (never, ever *vs*. current), frequency of alcohol intake (never, <3 times/week, *vs*. ≥3 times/week), intake of fruits and vegetables (servings/day as a continuous variable), sleep duration (<7 h/day, 7−8 h/day, *vs*. >8 h/day), discretionary screen time (as a continuous variable), duration of accelerometer-measured MVPA (as a continuous variable), accelerometer wear time (as a continuous variable), and season of accelerometer wear (spring, summer, autumn *vs*. winter). We categorized long-standing health problem/disability according to the self-reported question, "*Do you have any long-standing illness, disability, or infirmity*?" with response options of "*yes*" or "*no*." Participants who responded "*yes*" were classified as having a long-standing health problem/disability.

2.5. Statistical analysis

Baseline characteristics were presented as mean ± standard deviation (SD) for continuous variables and number (%) for categorical variables. We used one-way analysis of variance and χ^2 test to assess differences in baseline characteristics across quartiles of accelerometer-derived LPA. We used Cox proportional hazard models, with the completion of accelerometer wear as the start of follow-up, to assess the associations of LPA with mortality (all-cause and cause-specific) and disease incidence (CVD and cancer). Four models were used as follows: Model 1 was adjusted for age and sex; Model 2 was additionally adjusted for ethnic/racial group, educational attainment, employment status, Townsend Deprivation Index score, and self-reported health; Model 3 was additionally adjusted for smoking status, alcohol intake, intake of fruits and vegetables, sleep duration, and discretionary screen time; Model 4 was additionally adjusted for accelerometer wear time, season of accelerometer wear, and MVPA. We used a restricted cubic spline fitted in Cox proportional hazard models to assess the dose−response associations of LPA with mortality and morbidity, adjusting for confounders in Model 4. In our analysis, we evaluated restricted cubic spline models with 3, 4, and 5 knots to balance model flexibility and avoid overfitting. Model selection was based on the Akaike Information Criterion (AIC), where a lower value indicates a better fit after accounting for model complexity. Among the candidate

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models, the 3-knot spline yielded the lowest AIC and was therefore selected as the optimal choice for the final analysis (Supplementary Table 2).[15] The 3 knots were positioned at the 10th, 50th, and 90th percentiles of the LPA distribution, which is an empirical strategy validated in prior epidemiological studies.[16,17] For the primary analyses, the 5th percentile (1114 min/week ≈ 2.7 h/day) was set as the reference to align with conventions for low-exposure anchoring in dose-response analyses. We evaluated both the minimal dose of LPA associated with 50% of the lowest hazard ratio (HR) and the optimal dose corresponding to the lowest HR (i.e., the nadir of the dose curve) for non-linear associations.[18,19] We calculated 95% confidence intervals (95% CIs) for the minimal and optimal doses using bootstrapping with replacement, with 1000 iterations. Based on the prevalence of exposure and the relative risks of exposure with outcomes, the population attributable fraction (PAF) was calculated to evaluate the proportion of cases that could be prevented if all participants achieved the minimal or optimal doses of LPA.[20]

Furthermore, we performed subgroup analyses stratified by age (<60 years *vs.* ≥60 years), sex (males *vs.* females), MVPA (<150 min/week *vs.* ≥150 min/week), sleep duration (<7 h/day, 7−8 h/day, and >8 h/day), discretionary screen time (<3 h/day *vs.* ≥3 h/day, median split), self-reported health status (excellent, good, and fair/poor), educational attainment (college/university degree *vs.* below college/university degree), and socioeconomic level (below *vs.* above the median Townsend Deprivation score of −2.43). We also performed sensitivity analyses to evaluate the robustness of our findings. First, we conducted analyses with multiple imputation for missing values of all confounders. Second, to mitigate potential reverse causality, we excluded outcomes of interest that occurred within 2 or 5 years after baseline. Third, we assessed the dose−response associations using either the minimum value (959 min/week ≈ 2.3 h/day) or the 20th percentile (1567 min/week ≈ 3.7 h/day) as the reference. Fourth, we additionally adjusted for potential mediators in the models, including body mass index, blood pressure, glycated hemoglobin A1c (HbA1c), high-density lipoprotein cholesterol, anti-hypertensive medication use, antihyperglycemic medication use, and lipid-lowering medication use. Fifth, to examine the associations between LPA and mortality risks, we included patients with pre-existing CVDs and cancers at baseline and adjusted for these conditions as covariates in Cox proportional hazard models. Sixth, we further adjusted for self-reported long-standing health problem/disability as an additional confounding factor in Cox proportional hazard models. All analyses were conducted using R software Version 4.0.3 (The R Foundation for Statistical Computing, Vienna, Austria). Two-sided P values < 0.05 were considered statistically significant.

3. Results

3.1. Demographics

This study included 69,492 participants (age = 61.26 ± 7.83 years, mean ± SD; 42.47% males; 96.59% White participants). During the median follow-up of 8.04 years, 2024 participants died from all causes, 539 from CVD, and 1175 from cancer; there were 7731 CVD cases and 6347 cancer cases.

Baseline characteristics of the study participants, categorized by quartiles of LPA (<1656 min/week, 1656−<2090 min/week, 2090−<2574 min/week, and ≥2574 min/week), are shown in Table 1. Participants who engaged in more LPA were more likely to be older, females, non-White, and unemployed, to have lower education attainment, lower socioeconomic level (i.e., higher Townsend Deprivation Index), poor health, current smoking status, alcohol intake ≥3 times/week, to consume more fruits and vegetables, to spend less time on discretionary screen time and MVPA, to have longer wrist accelerometer wear time, and to wear the device more frequently in summer ($P < 0.001$).

3.2. Mortality and disease incidence

3.2.1. Quartile

As shown in Table 2, compared with participants in the lowest quartile of LPA (<1656 min/week), the HRs (95% CIs) for all-cause mortality among those with 1656 to <2090 min/week, 2090 to <2574 min/week, ≥2574 min/week were 0.82 (95% CI: 0.73−0.93), 0.75 (95% CI: 0.66−0.85), and 0.77 (95% CI: 0.68−0.88), respectively (Model 4). The corresponding HRs (95% CIs) for CVD-specific mortality were 0.76 (95% CI: 0.61−0.95), 0.78 (95% CI: 0.62−0.98), and 0.68 (95% CI: 0.53−0.88), respectively; and those for cancer-specific mortality were 0.89 (95% CI: 0.76−1.04), 0.77 (95% CI: 0.65−0.91), and 0.83 (95% CI: 0.70−0.99), respectively. Similar patterns were observed for the incidence of CVD and cancer (Table 2). Additionally, we found that increasing quartiles of LPA were associated with lower cumulative risks of all-cause, CVD-specific, and cancer-specific mortality, as well as the incidence of CVD and cancer (Fig. 2).

3.2.2. Volume

We observed an inverse non-linear dose-response association between LPA and all-cause mortality. The optimal dose was 2404 min/week (95% CI: 2289−2691), with a corresponding HR of 0.63 (95% CI: 0.56−0.71), and the minimal dose was 1506 min/week (95% CI: 1484−3594), with a corresponding HR of 0.81 (95% CI: 0.78−0.86), as compared to the reference of the 5th percentile (1114 min/week) ($p_{\text{nonlinear}} < 0.001$, Fig. 3 and Table 3). Similarly, for CVD-specific mortality, the optimal and minimal doses were 2566 min/week (95% CI: 2253−3640) and 1535 min/week (95% CI: 1480−3499) ($p_{\text{nonlinear}} = 0.017$). For cancer-specific mortality, the corresponding doses were 2457 min/week (95% CI: 2232−3640) and 1533 min/week (95% CI: 1494−3578) ($p_{\text{nonlinear}} = 0.006$) (Fig. 3 and Table 3). For disease incidence, the association between LPA and CVD incidence was non-linear, with an optimal dose of 2929 min/week (95% CI: 2353−3640) and a minimal dose of 1619 min/week (95% CI: 1536−2531). However, the association with cancer incidence was linear, with a minimal dose of 1848 min/week (95% CI: 1538−3151) and no identifiable optimal dose (Fig. 3 and Table 3).

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Table 1
Baseline characteristics of the study population.

Characteristic	Overall	Light-intensity physical activity (min/week)				*P*
		Q1 (<1656)	Q2 (1656−<2090)	Q3 (2090−<2574)	Q4 (≥2574)	
n	69,492	17,375	17,372	17,368	17,377	
Age (year)	61.26 ± 7.83	60.91 ± 8.00	61.42 ± 7.76	61.63 ± 7.76	61.09 ± 7.77	<0.001
Male	29,516 (42.47)	10,571 (60.84)	7875 (45.33)	6064 (34.91)	5006 (28.81)	<0.001
White	67,121 (96.59)	16,835 (96.89)	16,848 (96.98)	16,783 (96.63)	16,655 (95.85)	<0.001
Education						<0.001
College or university degree	31,001 (44.61)	8691 (50.02)	8151 (46.92)	7484 (43.09)	6675 (38.41)	
A levels/AS levels or equivalent	9431 (13.57)	2291 (13.19)	2302 (13.25)	2438 (14.04)	2400 (13.81)	
O levels/GCSEs or equivalent	14,049 (20.22)	2975 (17.12)	3413 (19.65)	3727 (21.46)	3934 (22.64)	
CSE or equivalent	2962 (4.26)	560 (3.22)	636 (3.66)	702 (4.04)	1064 (6.12)	
NVQ or HND or HNC or equivalent	3524 (5.07)	970 (5.58)	840 (4.84)	835 (4.81)	879 (5.06)	
Others	8525 (12.27)	1888 (10.87)	2030 (11.69)	2182 (12.56)	2425 (13.96)	
Employed	65,306 (93.98)	16,464 (94.76)	16,518 (95.08)	16,364 (94.22)	15,960 (91.85)	<0.001
Townsend Deprivation Index	−1.71 (2.83)	−1.49 (2.97)	−1.74 (2.81)	−1.84 (2.75)	−1.77 (2.76)	<0.001
Self-reported health						<0.001
Excellent	16,631 (23.93)	3900 (22.45)	4218 (24.28)	4345 (25.02)	4168 (23.99)	
Good	42,215 (60.75)	10,125 (58.27)	10,617 (61.12)	10,651 (61.33)	10,822 (62.28)	
Fair	9388 (13.51)	2841 (16.35)	2251 (12.96)	2128 (12.25)	2168 (12.48)	
Poor	1258 (1.81)	509 (2.93)	286 (1.65)	244 (1.40)	219 (1.26)	
Smoking status						<0.001
Never	40,973 (58.96)	10,233 (58.89)	10,134 (58.34)	10,169 (58.55)	10,437 (60.06)	
Ever	23,725 (34.14)	5751 (33.10)	6031 (34.72)	6106 (35.16)	5837 (33.59)	
Current	4794 (6.90)	1391 (8.01)	1207 (6.95)	1093 (6.29)	1103 (6.35)	
Frequency of alcohol intake						<0.001
Never	3796 (5.46)	924 (5.32)	838 (4.82)	915 (5.27)	1119 (6.44)	
<3 times a week	31,926 (45.94)	7866 (45.27)	7844 (45.15)	8014 (46.14)	8202 (47.20)	
≥3 times a week	33,770 (48.60)	8585 (49.41)	8690 (50.02)	8439 (48.59)	8056 (46.36)	
Fruits and vegetables (servings/day)	4.08 ± 2.09	3.74 ± 2.01	4.03 ± 2.06	4.20 ± 2.05	4.33 ± 2.21	<0.001
Sleep duration						<0.001
<7 h/day	15,152 (21.80)	3730 (21.47)	3650 (21.01)	3773 (21.72)	3999 (23.01)	
7−8 h/day	50,305 (72.39)	12,482 (71.84)	12,654 (72.84)	12,616 (72.64)	12,553 (72.24)	
>8 h/day	4035 (5.81)	1163 (6.69)	1068 (6.15)	979 (5.64)	825 (4.75)	
Discretionary screen time (h/day)	3.50 ± 2.13	4.01 ± 2.36	3.58 ± 2.10	3.37 ± 2.00	3.05 ± 1.89	<0.001
MVPA (min/week)	300.33 ± 246.97	292.75 ± 253.92	312.20 ± 250.56	305.17 ± 246.06	291.22 ± 236.38	<0.001
Wear time (day)	6.65 ± 0.66	6.63 ± 0.70	6.67 ± 0.64	6.68 ± 0.62	6.65 ± 0.68	<0.001
Wear season						<0.001
Spring	16,006 (23.03)	3968 (22.84)	4091 (23.55)	4065 (23.41)	3882 (22.34)	
Summer	18,392 (26.47)	4396 (25.30)	4429 (25.50)	4573 (26.33)	4994 (28.74)	
Autumn	20,471 (29.46)	5193 (29.89)	5098 (29.35)	5142 (29.61)	5038 (28.99)	
Winter	14,623 (21.04)	3818 (21.97)	3754 (21.61)	3588 (20.66)	3463 (19.93)	

Notes: Data are shown as mean ± SD for continuous variables and number (%) for categorical variables. Percentages might not add up to 100% due to rounding.
Abbreviations: AS = advanced subsidiary; CSE = Certificate of Secondary Education; GCSE = General Certificate of Secondary Education; HNC = High National Certificate; HND = High National Diploma; MVPA = moderate-to-vigorous-intensity physical activity; NVQ = National Vocational Qualification.

3.3. Percent contribution of LPA

The optimal and minimal doses of LPA were estimated to prevent approximately 7.9% (95% CI: 1.6%−14.7%) and 6.2% (95% CI: 3.7%−8.7%) of all-cause deaths, respectively. Similarly, the optimal and minimal doses could prevent approximately 16.6% and 10.4% of CVD deaths, respectively; 3.2% and 4.0% of cancer deaths, and 2.7% and 1.9% of CVD incidence (Supplementary Fig. 1).

3.4. Subgroup analyses

In the subgroup analyses, the protective effects of LPA on mortality and disease incidence were more pronounced in specific populations. Individuals aged ≥60 years who performed more LPA showed significant risk reductions for all-cause and cause-specific mortality, as well as CVD incidence, whereas no significant protective associations were observed in those aged <60 years (Supplementary Table 3). Compared with males, females who performed more LPA demonstrated more statistically significant protective effects on all-cause and cancer-specific mortality as well as CVD and cancer incidence (Supplementary Table 4). Individuals who engaged in <150 min/week of MVPA in addition to more LPA showed significant protective effects across all outcomes, whereas those with MVPA ≥ 150 min/week showed no such benefits (Supplementary Table 5). Individuals who slept 7−8 h/day and performed more LPA showed significant protective effects on all-cause and cause-specific mortality as well as CVD incidence. However, those who slept >8 h/day only had a significant protective effect on CVD mortality. No

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Table 2
Associations of accelerometer-derived light-intensity physical activity categories with mortality and major disease incidence.

Outcome	Light-intensity physical activity category	No. of cases/total participants	Incidence rate per 1000 person-years	Hazard ratio (95% CI)			
				Model 1	Model 2	Model 3	Model 4
All-cause mortality							
	<1656 min/week	676/17,375	4.90	Reference	Reference	Reference	Reference
	1656–<2090 min/week	496/17,372	3.58	0.76 (0.67–0.85)	0.79 (0.70–0.89)	0.80 (0.71–0.90)	0.82 (0.73–0.93)
	2090–<2574 min/week	434/17,368	3.13	0.68 (0.60–0.77)	0.72 (0.63–0.81)	0.73 (0.64–0.83)	0.75 (0.66–0.85)
	≥2574 min/week	418/17,377	3.01	0.71 (0.63–0.81)	0.74 (0.66–0.84)	0.76 (0.67–0.87)	0.77 (0.68–0.88)
CVD mortality							
	<1656 min/week	201/17,375	1.46	Reference	Reference	Reference	Reference
	1656–<2090 min/week	126/17,372	0.91	0.68 (0.54–0.85)	0.72 (0.57–0.90)	0.74 (0.59–0.92)	0.76 (0.61–0.95)
	2090–<2574 min/week	119/17,368	0.86	0.68 (0.54–0.86)	0.73 (0.58–0.92)	0.75 (0.60–0.95)	0.78 (0.62–0.98)
	≥2574 min/week	93/17,377	0.67	0.60 (0.47–0.78)	0.64 (0.49–0.82)	0.67 (0.52–0.87)	0.68 (0.53–0.88)
Cancer mortality							
	<1656 min/week	363/17,375	2.63	Reference	Reference	Reference	Reference
	1656–<2090 min/week	300/17,372	2.17	0.83 (0.71–0.97)	0.86 (0.73–1.00)	0.87 (0.75–1.02)	0.89 (0.76–1.04)
	2090–<2574 min/week	253/17,368	1.82	0.71 (0.60–0.84)	0.73 (0.62–0.86)	0.75 (0.64–0.89)	0.77 (0.65–0.91)
	≥2574 min/week	259/17,377	1.86	0.78 (0.66–0.91)	0.79 (0.67–0.93)	0.82 (0.69–0.97)	0.83 (0.70–0.99)
CVD incidence							
	<1656 min/week	2212/17,375	16.94	Reference	Reference	Reference	Reference
	1656–<2090 min/week	2000/17,372	15.20	0.94 (0.88–1.00)	0.96 (0.91–1.02)	0.97 (0.91–1.03)	0.98 (0.92–1.04)
	2090–<2574 min/week	1806/17,368	13.63	0.87 (0.82–0.93)	0.90 (0.85–0.96)	0.90 (0.85–0.96)	0.92 (0.86–0.98)
	≥2574 min/week	1713/17,377	12.87	0.89 (0.83–0.95)	0.91 (0.85–0.97)	0.92 (0.86–0.98)	0.92 (0.86–0.99)
Cancer incidence							
	<1656 min/week	1707/17,375	13.04	Reference	Reference	Reference	Reference
	1656–<2090 min/week	1633/17,372	12.40	0.97 (0.91–1.04)	0.97 (0.91–1.04)	0.97 (0.91–1.04)	0.97 (0.91–1.04)
	2090–<2574 min/week	1571/17,368	11.90	0.95 (0.89–1.02)	0.96 (0.89–1.02)	0.96 (0.89–1.03)	0.96 (0.89–1.03)
	≥2574 min/week	1436/17,377	10.80	0.91 (0.85–0.98)	0.92 (0.85–0.99)	0.92 (0.85–0.99)	0.92 (0.85–0.99)

Notes: Model 1 was adjusted for age and sex; Model 2 was adjusted for age, sex, ethnic/racial group, educational attainment, employment status, TDI score, and self-reported health; Model 3 was adjusted for age, sex, ethnic/racial group, educational attainment, employment status, TDI, self-reported health, smoking status, alcohol intake, intake of fruits and vegetables, sleep duration, and discretionary screen time; Model 4 was adjusted for age, sex, ethnic/racial group, educational attainment, employment status, TDI, self-reported health, smoking status, alcohol intake, intake of fruits and vegetables, sleep duration, discretionary screen time, accelerometer wear time, season of accelerometer wear, and moderate-to-vigorous intensity physical activity.
Abbreviations: 95% CI = 95% confidence interval; CVD = cardiovascular disease; TDI = Townsend Deprivation Index.

significant protective effects were observed among those who slept <7 h/day (Supplementary Table 6). Individuals with screen time ≥3 h/day who performed more LPA showed significant protective effects across all outcomes, whereas those with screen time <3 h/day showed benefits only for all-cause and cancer-specific mortality (Supplementary Table 7). Compared with individuals with good or excellent health status who performed more LPA, those with fair/poor health status showed more statistically significant protective effects on mortality and disease incidence, particularly at higher activity levels (Quartiles 3–4) (Supplementary Table 8). Individuals with low educational attainment who performed more LPA showed significant risk reductions in all-cause and cause-specific mortality, as well as CVD incidence, whereas those with high education showed benefits only for all-cause and cancer-specific mortality (Supplementary Table 9). Individuals with low socioeconomic levels who performed more LPA exhibited significant protective effects on all-cause and cause-specific mortality as well as cancer incidence, whereas those with high socioeconomic levels showed benefits only for all-cause mortality and CVD incidence (Supplementary Table 10).

3.5. Sensitivity analyses

Sensitivity analyses demonstrated that findings were robust based on data with multiple imputations for variables with missing values (Supplementary Fig. 2); after excluding outcomes of interest that occurred within 2 years (Supplementary Fig. 3); using the minimum value (959 min/week) as the reference (Supplementary Fig. 4) or the 20th percentile (1567 min/week) as the reference (Supplementary Fig. 5); and further adjusting for potential mediators, including body mass index, blood pressure, HbA1c, high-density lipoprotein cholesterol, use of anti-hypertensive medication, use of anti-hyperglycemic medication, and use of lipid-lowering medication (Supplementary Fig. 6); including patients with pre-existing CVDs and cancers at baseline and adjusted for these conditions as covariates in Cox proportional hazard models for examination of association with mortality (Supplementary Table 11); and adjusted for long-standing health problem/disability as an additional confounding factor in Cox proportional hazard models (Supplementary Table 12). However, when the exclusion period was extended to 5 years, the nonlinear associations between LPA and cause-specific mortality and incidence were

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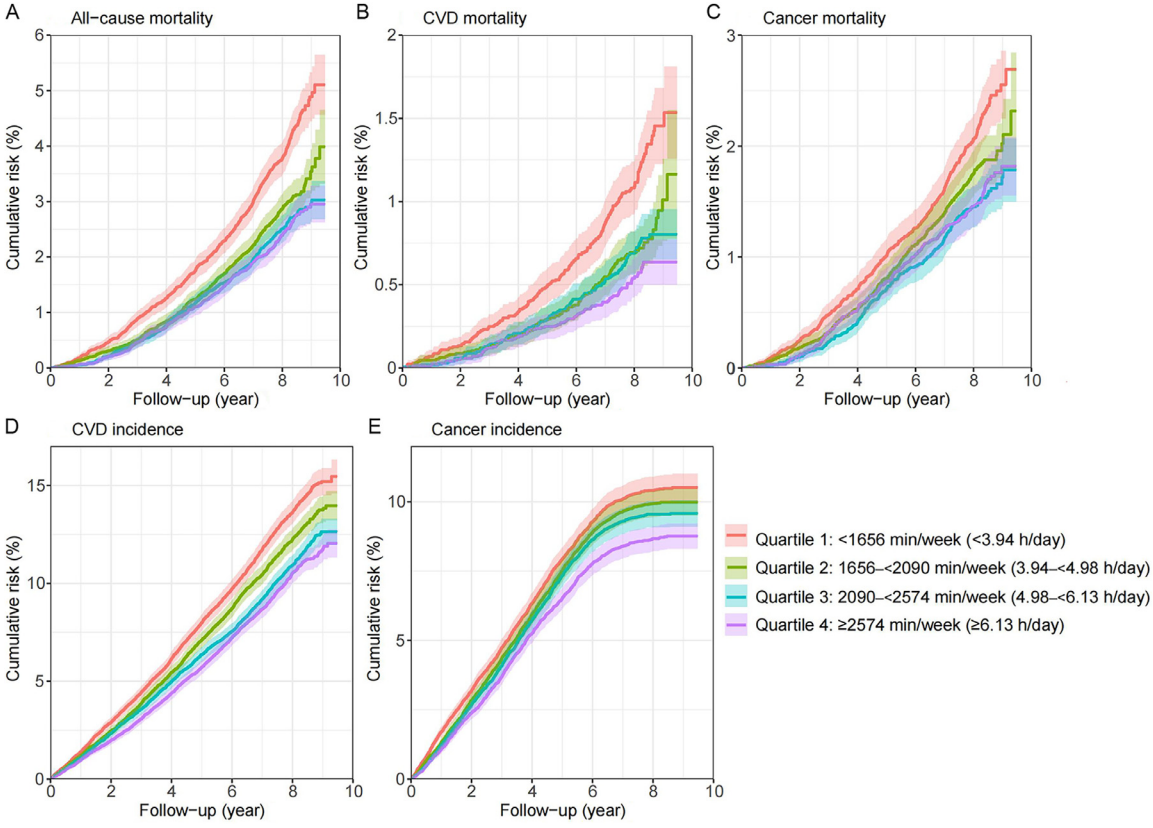

Fig. 2. Cumulative risk of mortality and disease incidence stratified by light-intensity physical activity quartiles. CVD = cardiovascular disease.

no longer statistically significant. Nevertheless, the inverse associations with disease-specific outcomes remained consistent with the primary findings (Supplementary Fig. 7).

4. Discussion

In this large prospective cohort study with accelerometer-measured PA data, we observed a non-linear inverse association between LPA and the risk of all-cause mortality, cause-specific mortality, and CVD incidence. We found that an optimal dose of ~6.0 h/day (2500 min/week) of LPA was associated with a 13%−37% lower risk of mortality and disease incidence. A minimal dose of ~3.5 h/day (1500 min/week) was associated with a 6%−19% lower risk of mortality and disease incidence. Our findings suggest that engaging in ~6.0 h/day and ~3.5 h/day of LPA can respectively maximize and conserve the reductions in mortality and disease incidence. Our findings have significant implications for public health guidelines and will help fill the gap in quantitative recommendations for LPA.

4.1. Comparison to prior studies

Compared to MVPA, LPA is easier to incorporate into daily routines and to maintain over extended periods due to its incidental nature. It carries a lower risk of injury and is suitable for individuals of all ages and fitness levels, especially for older people and those who are unable to engage in MVPA due to chronic diseases or disability. In contrast, MVPA requires considerable time and can be physically challenging for those with established chronic diseases or poor fitness.[5,12,21-23] In our study, sleep, sedentary behavior, LPA, and MVPA accounted for 36.7% (~8.8 h/day), 39.0% (~9.4 h/day), 21.3% (~5.1 h/day), and 3.0% (~0.7 h/day) of daily activity, respectively (Supplementary Table 13). This highlights the substantial contribution of LPA to daily life, despite it often being overshadowed by other activities. Current public health recommendations and PA guidelines primarily focus on the time spent in MVPA, with limited evidence to support the minimal and optimal volumes of LPA.[1,2]

The findings on the dose−response association between LPA and all-cause mortality have been inconclusive across previous studies. Three previous meta-analyses have shown the protective effects of LPA on all-cause mortality.[5,24,25] However, the arbitrary categorization of aggregated summary data in these meta-analyses may result in a loss of information and pose challenges in translating them into definitive PA targets for public health decision-making.[26] In addition, the high between-study heterogeneity made the findings unreliable. Nevertheless, a harmonized meta-analysis including eight studies with 36,383 participants (mean age of 62.6 years) showed a substantial reduction in the risk of all-cause mortality associated with accelerometry-measured LPA.[24] Another pooled analysis of four prospective cohort studies including 11,989 participants with a median follow-up of

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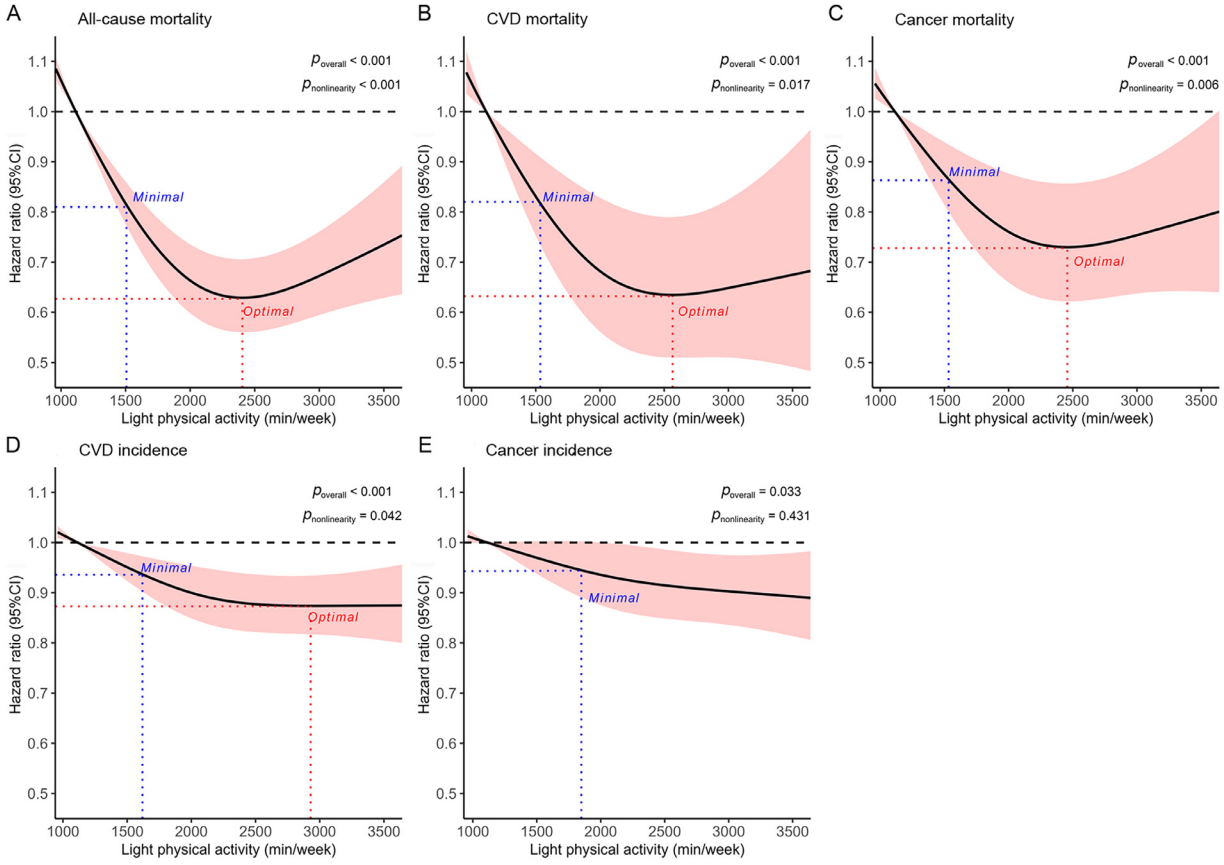

Fig. 3. Dose–response associations of light-intensity physical activity with mortality and disease incidence. Models were adjusted for age, sex, ethnic/racial group, educational attainment, employment status, Townsend Deprivation Index, alcohol intake, smoking status, sleep duration, discretionary screen time, intake of fruits and vegetables, self-reported health, accelerometer wear time, season of accelerometer wear, and moderate-to-vigorous intensity physical activity. To reduce the influence of extreme values, the range was capped between the 2.5th and 97.5th percentile. The reference was set at the 5th percentile of light-intensity physical activity (i.e., 1114 min/week ≈ 2.65 h/day). 95% CI = 95% confidence interval; CVD = cardiovascular disease.

5.2 years found that, compared to 183 min/day of accelerometry-measured LPA, an additional 15 min/day was associated with an 11% lower risk of all-cause mortality, and the optimal risk reduction was observed at 330 min/day (~5.5 h/day).[27] However, the first meta-analysis did not provide the minimal and optimal doses of LPA, whereas the second one provided an optimal dose solely. It is noteworthy that the optimal dose of LPA provided by the previous study (~5.5 h/day) is largely consistent with our result (~6 h/day). A recent study using UK Biobank data corroborates our observation of an inverse dose–response relationship between LPA and all-cause mortality across frailty levels.[28] However, that study did not establish the minimal or optimal dose of such activity, and it utilized a shorter median follow-up period (6.9 years) compared to ours (8.04 years). Our findings substantiated and extended the nonlinear dose-response association between LPA and all-cause

Table 3
Hazard ratios of optimal and minimal levels of light-intensity physical activity for mortality and major disease incidence.

		All-cause mortality	CVD mortality	Cancer mortality	CVD incidence	Cancer incidence
Optimal point (95% CI)	min/week	2404 (2289–2691)	2566 (2253–3640)	2457 (2232–3640)	2929 (2353–3640)	-
	h/day	5.72 (5.45–6.41)	6.11 (5.36–8.67)	5.85 (5.31–8.67)	6.97 (5.60–8.67)	-
Optimal HR (95% CI)		0.63 (0.56–0.71)	0.63 (0.45–0.78)	0.73 (0.61–0.85)	0.87 (0.80–0.93)	-
Minimal point (95% CI)	min/week	1506 (1484–3594)	1535 (1480–3499)	1533 (1494–3578)	1619 (1536–2531)	1848 (1538–3151)
	h/day	3.59 (3.53–8.56)	3.65 (3.52–8.33)	3.65 (3.56–8.52)	3.85 (3.66–6.03)	4.40 (3.66–7.50)
Minimal HR (95% CI)		0.81 (0.78–0.86)	0.82 (0.73–0.89)	0.86 (0.81–0.92)	0.94 (0.90–0.96)	0.94 (0.91–0.98)

Notes: We evaluated both the minimal dose of light-intensity physical activity associated with 50% of the lowest HR and the optimal dose corresponding to the lowest HR (i.e., the nadir of the dose curve). Hazard ratios were adjusted for age, sex, ethnic/racial group, educational attainment, employment status, TDI, self-reported health, smoking status, alcohol intake, intake of fruits and vegetables, sleep duration, discretionary screen time, accelerometer wear time, season of accelerometer wear, and moderate-to-vigorous intensity physical activity.
Abbreviations: 95% CI = 95% confidence interval; CVD = cardiovascular disease; HR = hazard ratio; TDI = Townsend Deprivation Index.

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mortality, indicating that LPA lasting for up to ~3.5 h/day was associated with a substantially lower (19%) all-cause mortality risk, with an additional beneficial effect observed up to an optimal dose of ~6 h/day (37%). Overall, our study findings suggest that encouraging participation in LPA throughout the day is effective for reducing all-cause mortality risk, as it facilitates easy engagement, long-term adherence, and promotion opportunities.

A previous meta-analysis, which included five prospective studies, showed a linear dose–response association between LPA and cancer-specific mortality.[8] However, this meta-analysis did not provide the minimal and optimal doses of LPA. In contrast, we found a significant nonlinear dose–response association. We found that engaging in ~6.0 h/day and ~3.5 h/day of LPA can respectively maximally and minimally reduce the risk of cancer-specific mortality, by 27% and 14% respectively. Our findings highlight the clinical and public health importance of engaging in at least 3.5 h/day of LPA to prevent the development of cancer. It is worth noting that the sample size in the previous meta-analysis was relatively smaller than ours (10,151 vs. 69,492), and the definition of LPA varied significantly across the included studies.

Regarding cancer-specific incidence, only one prior study based on 85,394 individuals from the UK Biobank showed that reallocating 1 h/day from sedentary time to LPA was associated with a 7.0% reduction in the risk of cancers of head and neck, esophageal adenocarcinoma, bladder, breast, kidney, colon, gastric cardia, lung, liver, endometrial, myeloma, myeloid leukemia, and rectal.[29] Another study of 70,747 participants from the UK Biobank established an inverse dose-response association between LPA and the risk of cancer incidence.[30] However, these previous studies above also did not determine the minimal and optimal doses of LPA. In our study, we included more cancer types and consistently found that a higher level of LPA was associated with lower risks of cancer. Additionally, we further determined a linear association between LPA and cancer incidence, with a minimal dose of 4.4 h/day. The optimal dose cannot be identified, which might be due to the fact that before the onset of cancer, individuals with a relatively healthy state can tolerate PA of longer duration, compared to cancer patients, who are often accompanied by fatigue.[31] Our findings suggest that engaging in LPA for at least 4.4 h/day can confer a significant benefit in cancer prevention. The higher the dose of LPA beyond this minimal threshold, the greater the potential reduction in cancer incidence risk.

Consistent with our findings, a meta-analysis of five prospective studies suggested a non-linear dose–response association between LPA and CVD-specific mortality.[7] However, the dose–response curve presented in that meta-analysis appears nearly linear, and that study did not establish the optimal dose. In addition, the findings of that meta-analysis should be interpreted with caution due to inconsistent criteria for defining LPA and the high degree of heterogeneity among included studies. A study involving 24,139 non-exercisers from the UK Biobank reported a modest inverse association between LPA and all-cause mortality, CVD incidence, and CVD mortality, with statistical significance achieved only for CVD mortality at activity levels exceeding 2.2 h/day.[17] However, the generalizability of these findings is limited by the study's relatively small sample size and its specific focus on a non-exercising population. As a result, the applicability of these results in guiding practical public health interventions remains limited. In our study, we used a prospective cohort design with a large sample size ($n = 69,492$) of the general UK population and identified both the optimal level (~6.1–7.0 h/day) and the minimal level (~3.7–3.9 h/day) of LPA for reducing the risk of CVD incidence and mortality. Our findings provide concrete evidence for clinicians and offer critical scientific support for public health policymakers to design activity promotion initiatives and establish health goals.

To further assess the modified effect of other physical activities on the association between LPA and the risk of mortality and morbidity, we performed a subgroup analysis stratified by the duration of MVPA (i.e., <150 min/week and ≥150 min/week), daily sleep duration (<7 h/day, 7–8 h/day, and >8 h/day), and discretionary screen time (<3 h/day and ≥3 h/day). We found that the beneficial effect of LPA on mortality and morbidity was more pronounced among individuals who engaged in less MVPA and slept 7–8 h per day, and those with higher screen time. Existing evidence indicates that replacing sedentary behavior with LPA can significantly reduce all-cause, CVD-, and cancer-specific mortality and incidence.[12,32–35] Although LPA confers antioxidant benefits, this advantage may be offset by the inflammatory response associated with sleep deprivation.[36] Our findings suggest the potential for increasing LPA to offset risks associated with other unhealthy activity status (e.g., low MVPA levels and more screen time) under the foundational requirement of adequate sleep across the 24-h activity cycle. This underscores the importance of holistic behavioral interventions that integrate PA, sleep, and screen time. Further studies are warranted to evaluate the effects of replacing unhealthy activities with LPA on mortality and disease incidence.

Evidence regarding differences in associations of LPA with health outcomes by age, sex, and health status is limited. Similar to a previous large prospective study assessing the association between recommended MVPA and mortality,[37] we found that the effect size between LPA and mortality was stronger among older adults aged ≥60 years (*vs.* younger adults aged <60 years) and among individuals with fair/poor health status (*vs.* excellent and good health status). We also supported previous findings that LPA was associated with all-cause mortality and CVD and cancer outcomes among people with mobility limitations,[38] CVD,[39] type 2 diabetes,[40] hypertension,[41] and cardiometabolic disease.[42] The Physical Activity Guidelines for Americans recommend that the appropriate amount, types, and intensity (including light-intensity) of PA should be tailored according to individuals' capabilities and the severity of chronic conditions.[43] The World Health Organization guidelines also support expanding the scope of actions (including LPA) to people with chronic conditions and disabilities.[1] Therefore, LPA should be particularly promoted for individuals with poor health status or long-standing health

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problem/disability who cannot meet MVPA guidelines. Moreover, these findings suggest that integrating even minimal or optimal amounts of LPA into daily life, which is often more feasible than structured MVPA, can contribute meaningfully to the prevention, management, and reduction of mortality and disease incidence.

Consistent with prior research,[44] our study found that females experienced greater reductions in mortality and disease incidence risk within equivalent quartiles of leisure-time PA. Furthermore, we observed sex-specific differences in the benefits of LPA. This disparity may be attributed to the fact that although males generally demonstrate higher exercise capacity, females possess distinct physiological characteristics, such as higher capillary density and a greater proportion of type I muscle fibers, that may facilitate more relative strength gains and improved vascular responses to PA.[44] These mechanisms could contribute to their heightened sensitivity to PA in terms of mortality risk reduction. Our findings may also help address sex-based disparities in PA benefits by encouraging greater participation in LPAs, especially among women.

4.2. Potential mechanisms

Several potential mechanisms could explain the association between LPA and mortality risk. First, a previous study based on animal models showed that LPA could contribute to an improved metabolism of circulating lipids due to an increase in lipoprotein lipase activity.[45] Second, it is hypothesized that LPA could lead to an enhanced cardio−metabolic profile, improved glycemic control, and a reduction in inflammation levels.[5,46,47] Third, other underlying mechanisms include changes in metabolic hormones, endogenous sex steroids, oxidative stress, and immune function.[48]

4.3. Implications

Our findings help inform the development of public health guidance based on dose−response relationships for LPA. A minimal effective dose of approximately 3.5 h/day provides a practical and achievable target for sedentary individuals, indicating that even modest increases in daily movement, such as standing or slow walking, may yield clinically meaningful risk reductions ranging from 6% to 19%. Furthermore, an optimal dose of approximately 6.0 h/day was associated with greater risk reductions of 13%−37%, highlighting the substantial benefits attainable through higher volumes of LPA. Of note, our results showed no evidence of adverse effects and suggested continued benefits beyond 6 h/day, indicating that healthy adults need not restrict their engagement in light-intensity activities. Individuals already exceeding the optimal dose can be reassured that maintaining or even modestly increasing their current activity level remains advantageous, although marginal gains may diminish. These findings support the incorporation of both minimal and optimal dose targets in future PA guidelines, accompanied by tailored recommendations to promote incremental increases in daily movement.

4.4. Refining activity measurement

The Euclidean Norm Minus One (ENMO) metric is widely employed in large UK populations, such as the UK Biobank.[11] Evidence suggests that monitor-independent movement summary (MIMS) units demonstrate lower between-device coefficients of variation compared to ENMO, which can be more influenced by variations in dynamic range and sampling rate across different devices.[49,50] Differences in dynamic range may lead to under- or overestimation of PA intensity, potentially resulting in misclassification of activity levels and biased estimates of morbidity and mortality risks. Nevertheless, the UK Biobank adopted multiple acceleration metrics without explicitly separating gravitational components, which exhibit strong collinearity ($r > 0.95$), supporting the validity of ENMO for examining associations between PA and health outcomes.[11] Furthermore, ENMO demonstrates high accuracy in classifying PA intensities ($r \geq 0.90$).[51]

Two primary approaches have been used in previous studies to classify PA intensities: traditional cut-point methods (e.g., 30−125 mg for LPA)[40,52] and machine learning models.[12,13,16,17] Traditional cut-point methods frequently misclassified non-MVPA as MVPA, potentially leading to substantial overestimation or underestimation of the observed associations between PA and health outcomes.[12] The machine learning model used in our study can minimize the misclassification bias and limitations of metrics such as ENMO, demonstrating markedly higher precision than cut-point methods (0.75 vs. 0.37), and outperformed previous machine learning approaches applied to free-living data, as evidenced by a higher Cohen's κ value (0.80 vs. 0.68).[12,53] These improvements allow for more reliable estimation of dose-response associations, providing a robust foundation for evidence-based public health recommendations.

4.5. Strengths and limitations

To our knowledge, our study is the first national study to provide the minimal and optimal levels of accelerometer-measured LPA associated with reduced risk of mortality and disease incidence based on a large sample size ($n = 69{,}492$). However, several limitations should be acknowledged. First, we only included middle-aged and older adults aged ≥43 years. Whether the beneficial effects of LPA can be applied to younger populations remains unclear and requires further evaluation. Second, this study was conducted primarily in a Western-based population of middle-aged and older adults. Although we included a large and representative sample suited for detecting associations in populations of European descent, the generalizability of our findings to other racial/ethnic groups, as well as to younger individuals, cannot be assumed. Further research is warranted to establish minimal and optimal thresholds of LPA in younger adults and more diverse racial and ethnic populations. Third, repeated measures of LPA were not available, and thus, the potential changes in this activity over time may have affected the observed associations. However, the misclassification was non-differential, which may have underestimated the association. It has been

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shown that a 7-day accelerometer recording exhibits a relatively stable pattern over time.[54,55] Indeed, many high-quality papers using single-time measurements of PA in the UK Biobank have been published in recent years.[14,56] Fourth, while the use of a composite cancer endpoint enabled a broader evaluation of overall cancer risk, this approach may have masked underlying heterogeneity across specific cancer sites, which could obscure or modify the observed associations. Future studies should therefore evaluate site-specific cancers to determine whether the associations are driven by particular types or represent a generalized effect. Fifth, although the machine learning-based Random Forest approach enhances activity classification accuracy, residual misclassification may remain due to its sensitivity to MET thresholds and population-specific PA patterns. This could lead to potential overestimation of the associations between LPA and risks of mortality and disease incidence. Nevertheless, the model exhibited superior classification precision compared to traditional fixed cut-point methods (0.75 vs. 0.37). When applied to UK Biobank data, the behavioral classification showed strong concordance with expected daily activity profiles, supporting its empirical validity.[12,13,17,57] Sixth, although we adjusted for a wide range of covariates, residual confounding from unmeasured factors cannot be entirely eliminated, which complicates causal interpretation. To address potential confounding, we used comprehensively adjusted models and conducted subgroup analyses to assess how other components of the 24-h activity cycle (sleep duration, screen time, and MVPA) and socioeconomic factors (education and deprivation level) may modify the association between LPA and the outcomes. Variations in effect estimates across subgroups highlight remaining uncertainties in causal inference. Future studies should incorporate more precise and extensive measurements of potential confounders to strengthen causal conclusions. Seventh, to minimize reverse causality, we excluded events occurring within the first 2 or 5 years of follow-up. Consistent with the primary analysis, exclusion of the first 2 years did not alter the results. However, when the exclusion period was extended to 5 years, the nonlinear associations between LPA and cause-specific mortality and incidence were no longer statistically significant. A previous study similarly reported attenuation of nonlinear associations between PA and all-cause mortality with a 5-year exclusion period.[58] It is important to note that the 5-year exclusion may have reduced statistical power owing to the relatively short total follow-up (median 8.04 years) in our study, leading to less precise estimates. Although a 2-year exclusion is widely used in observational research to address reverse causality, future studies with longer follow-up are needed to verify these associations.[14,59,60]

5. Conclusion

Engaging in approximately 3.5 h/day of LPA was conservatively associated with reduced risks of mortality and major disease incidence. Further reductions in risk were observed with higher activity volumes, reaching an optimal dose of approximately 6.0 h/day. These findings suggest that the identified minimal and optimal doses of LPA could provide valuable supplementary recommendations to the existing public health and PA guidelines aimed at reducing disease incidence and mortality risk. The protective effects were particularly evident among vulnerable subgroups, underscoring the importance of developing tailored public-health messaging based on a full 24-h activity cycle, which should explicitly promote LPA as an accessible and effective intervention.

Authors' contributions

BX and MZ contributed to the study design, interpretation of the data analysis, and critical revision of the manuscript and had final responsibility for the decision to submit it for publication; JS drafted the manuscript and critical revision of the manuscript; YQ analyzed the data and contributed to the interpretation of the data and has accessed and verified the data; FL, RL, YY, and MW contributed to the interpretation of the data and critical revision of the manuscript. The corresponding author BX has access to and is responsible for the raw data associated with the study. All the authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Uncited references Q4

[33,34]

Declaration of competing interests

The authors declare that they have no competing interests.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jshs.2025.101099.

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