HOSTED BY

Available online at www.sciencedirect.com

ScienceDirect

Journal of Sport and Health Science 13 (2024) 579–589

Original article

Occupational physical activity, all-cause, cardiovascular disease, and cancer mortality in 349,248 adults: Prospective and longitudinal analyses of the MJ Cohort

Emmanuel Stamatakis [a,*], Matthew N. Ahmadi [a], Tiana-Lee Elphick [a], Bo-Huei Huang [a], Susan Paudel [a], Armando Teixeira-Pinto [b], Li-Jung Chen [c], Borja del Pozo Cruz [d], Yun-Ju Lai [e], Andreas Holtermann [d,f], Po-Wen Ku [g]

[a] *Sydney School of Health Sciences, Faculty of Medicine and Health, Charles Perkins Centre, University of Sydney, Sydney, NSW 2006, Australia*
[b] *Sydney School of Public Health, Faculty of Medicine and Health, University of Sydney, Sydney, NSW 2006, Australia*
[c] *Department of Exercise Health Science, "National" Taiwan University of Sport, Taichung 40404, China*
[d] *Department of Sports Science and Clinical Biomechanics, University of Southern Denmark, Odense 5230, Denmark*
[e] *Division of Endocrinology and Metabolism, Department of Internal Medicine, Puli Branch of Taichung Veterans General Hospital, Nantou 54552, China*
[f] *National Research Centre for the Working Environment, Copenhagen 2100, Denmark*
[g] *Graduate Institute of Sports and Health Management, "National" Chung Hsing University, Taichung 40227, China*

Received 18 October 2023; revised 19 December 2023; accepted 10 January 2024
Available online 8 March 2024

Abstract

Background: Evidence on the health benefits of occupational physical activity (OPA) is inconclusive. We examined the associations of baseline OPA and OPA changes with all-cause, cardiovascular disease (CVD), and cancer mortality and survival times.

Methods: This study included prospective and longitudinal data from the MJ Cohort, comprising adults over 18 years recruited in 1998–2016, 349,248 adults (177,314 women) with baseline OPA, of whom 105,715 (52,503 women) had 2 OPA measures at 6.3 ± 4.2 years (mean ± SD) apart. Exposures were baseline OPA, OPA changes, and baseline leisure-time physical activity.

Results: Over a mean mortality follow-up of 16.2 ± 5.5 years for men and 16.4 ± 5.4 years for women, 11,696 deaths (2033 of CVD and 4631 of cancer causes) in men and 8980 deaths (1475 of CVD and 3689 of cancer causes) in women occurred. Combined moderately heavy/heavy baseline OPA was beneficially associated with all-cause mortality in men (multivariable-adjusted hazard ratio (HR) = 0.93, 95% confidence interval (95%CI): 0.89–0.98 compared to light OPA) and women (HR = 0.86, 95%CI: 0.79–0.93). Over a mean mortality follow-up of 12.5 ± 4.6 years for men and 12.6 ± 4.6 years for women, OPA decreases in men were detrimentally associated (HR = 1.16, 95%CI: 1.01–1.33) with all-cause mortality, while OPA increases in women were beneficially (HR = 0.83, 95%CI: 0.70–0.97) associated with the same outcome. Baseline or changes in OPA showed no associations with CVD or cancer mortality.

Conclusion: Higher baseline OPA was beneficially associated with all-cause mortality risk in both men and women. Our longitudinal OPA analyses partly confirmed the prospective findings, with some discordance between sex groups.

Keywords: Cancer; Cardiovascular disease; Epidemiology; Mortality

1. Introduction

The health and longevity benefits of leisure-time physical activity (LTPA) are well-established.[1,2] The World Health Organization (WHO)[1] and many national authorities[3] have developed guidelines specifying the minimal thresholds of weekly physical activity required for general health. Such guidelines encourage physical activity in any domain, including work. However, the depth and breadth of evidence supporting the health benefits of each physical activity domain is uneven, with LTPA dominating research agendas over the last few decades and occupational physical activity (OPA) receiving much less attention.[4]

Peer review under responsibility of Shanghai University of Sport.
* Corresponding author.
E-mail address: emmanuel.stamatakis@sydney.edu.au (E. Stamatakis).

https://doi.org/10.1016/j.jshs.2024.03.002
Cite this article: Stamatakis E, Ahmadi MN, Elphick T-L, et al. Occupational physical activity, all-cause, cardiovascular disease, and cancer mortality in 349,248 adults: Prospective and longitudinal analyses of the MJ Cohort. *J Sport Health Sci* 2024;13:579–89.

Although early physical activity studies of the 1950s and 1970s[5,6] consistently pointed toward the likely cardiovascular and mortality benefits of physically demanding jobs, current research is far less conclusive. For example, a 2018 meta-analysis found that men in highly active jobs had an 18% higher risk of premature mortality compared with men in physically inactive jobs.[7] An umbrella review of 158 observational studies informing the WHO's 2020 physical activity guidelines[8] found favorable associations with higher levels of OPA across most of the 23 examined health outcomes, including cancer and cardiovascular disease (CVD). Two recent large Nordic studies further perplexed the OPA evidence landscape by reaching almost diametrically opposite conclusions.[9,10] In a cohort of 104,046 Danish adults followed for a median of 10 years, higher levels of baseline OPA showed detrimental associations with all-cause mortality and major CVD events, even among participants with very high LTPA levels.[10] In another cohort study of 437,378 Norwegian adults followed up for 28 years, high levels of baseline OPA were associated with higher longevity and lower CVD and cancer mortality risk in men but not in women.[9]

Besides the incongruent findings summarized above,[7,9,10] no study has examined the effects of OPA changes on prospective health outcomes. Changes in OPA are common[9] and may occur due to a variety of reasons, including deteriorating worker health. Findings from studies of baseline OPA showing harm[7,10] or benefit[8,9] may be more likely to reflect causal relationships if replicated by studies with repeated measures of OPA. Since trials that modify work patterns to change OPA are less feasible (in fact, no such study exists to date), longitudinal studies designed to examine OPA changes over time in relation to long-term outcomes are necessary for advancing this field of research.

Besides the escalating incongruence of the OPA literature outlined above, the vast majority of studies were done in affluent, mostly Nordic, Western countries (e.g., only 2 of the 33 studies included in the 2018 systematic review[7] were from non-Western countries). The distinctively different socio-occupational conditions, economic hierarchies, and regulations in non-Western countries may influence the health effects of OPA by, for example, determining aspects of the work environment, such as more tightly regulated provisions for recovery time and breaks, performance expectations, and norms around working hours. To our knowledge, the only large Chinese study in this field ($n = 142,302$) found manual work to be associated with increased all-cause mortality risk compared to sedentary workers in the whole sample, although associations were attenuated to the null after adjusting for potential confounders.[11] No evidence of an association between OPA and CVD mortality was present in the crude or adjusted models.[11]

The aim of our study was to examine the associations of baseline OPA and OPA changes with all-cause, CVD, and cancer mortality risk in a large cohort of adults in Taiwan, China. We also examined the independent and joint associations of baseline OPA and LTPA levels and the same set of mortality outcomes.

2. Methods

2.1. Study sample

We used data from the MJ Cohort, comprising adults undergoing routine health examination screening at the MJ Health Management Institution across 4 geographically diverse locations in the northern (Taipei), north-west (Taoyuan), central (Taichung), and southern (Kaohsiung) parts of Taiwan, China.[12] Participants have provided informed consent, and our study was approved by the "National" Changhua University of Education Research Ethics Committee, Taiwan, China (NCUEREC-108-072). During each examination, participants completed a self-administered questionnaire, anthropometric measurements, and a physical examination at the time of health check-up. In this study, we included economically active (full-time or part-time employed or self-employed, excluding students, retirees, and unemployed) people aged 18 years or over who were initially measured between 1998 and 2016 (Supplementary Figs. 1–12). We carried out 2 main sets of analyses: (a) baseline-only OPA against mortality to produce estimates comparable with the rest of the literature,[7–10] including the earliest examination with complete data ($n = 349,248$); and (b) OPA changes analyses among participants who attended at least 1 re-examination (follow-up measurement) with complete data ($n = 105,461$). Attendance at re-examinations was not based on any pre-determined criteria. If a participant had multiple re-examinations, we considered the most recent one as the follow-up measurement. To minimize the potential influence of reverse causality, we excluded from each analysis those participants with self-reported CVD or cancer at baseline (baseline OPA analyses), those who reported having developed CVD or cancer prior to the follow-up examination (OPA changes analyses), and those who died within the first 2 years of mortality follow-up.

2.2. Exposures

2.2.1. OPA

OPA was assessed through a closed-ended question comprising 4 options: light (mostly sedentary), moderate (repetitive motions while sitting or standing, e.g., manufacturing), moderately heavy (mostly standing or walking, e.g., construction, agriculture), and heavy (heavy lifting, loading, or moving loads). This measure is broadly comparable to the 4-level scales used in the recent Nordic studies.[9,10] Because of the low percentage of participants in the top OPA category of the original variable and the very low events rates (Supplementary Table 1), we merged the 2 most active OPA groups into "moderately heavy/heavy".

OPA changes over time were classified as decreased/stable/increased. Remaining in the same intensity category was considered "OPA stable", moving upward (e.g., from light to moderate or high intensity) was considered "OPA increased", moving downward (e.g., from moderate to light or from high to moderate) was considered "OPA decreased". The physical activity questionnaires are shown in Supplementary Text 1.

2.2.2. LTPA

Participants self-reported their participation in LTPA using 3 closed-ended questions related to activity type, intensity (light, moderate, medium-vigorous, and high-vigorous categories assigned 2.5, 4.5, 6.5, and 8.5 metabolic equivalents (METs), respectively[13–15]), and duration of time spent on these activities.[16] The questions and data handling have been described in detail elsewhere.[17] In brief, we calculated total LTPA volume (MET-h) by multiplying activity intensity (MET) by duration (h). Based on current physical activity guidelines,[1] participants were categorized into 4 groups: inactive (<1.00 MET-h), low (1.00–7.49 MET-h), moderate (7.50–14.99 MET-h), and high (≥15.00 MET-h). The MJ Cohort LTPA questionnaire has acceptable construct validity.[17,18] The LTPA questionnaire is provided in Supplementary Text 1.

2.2.3. Joint physical activity exposure

Using the OPA and LTPA categorizations above, we also derived a joint OPA–LTPA exposure[10] comprising all 12 mutually exclusive possible combinations of the 2 variables (Supplementary Text 2).

2.3. Sociodemographic and behavioral covariates

Our *a priori* selection of covariates was based on the most recent literature[9,10,13] and their availability in the MJ Cohort.[9] For baseline-only analyses (OPA, LTPA, and joint OPA × LTPA), we adjusted for age (continuous), birth cohort (<1940, 1940–1949, 1950–1959, 1960–1969, 1970–1979, or ≥1980), and the following self-reported variables: baseline education level (junior high school or less, senior high school, and college or higher), occupation type (Supplementary Text 3), daily fruit and vegetable intake (<3 servings, 3–4 servings, or ≥5 servings), combined smoking status (never, former, or current) and pack-years, sleep duration per day (<4 h, 4–<6 h, 6–8 h, and >8 h), alcohol status (former drinker, drinks seldomly, or current drinker), and body mass index (BMI).

2.4. Outcome assessment

Participants included in all analyses were followed up for mortality through the Death file[12] until death or censoring (March 2, 2022) in Taiwan, China. CVD mortality included deaths from coronary heart disease (International Classification of Diseases (ICD)-9: 410–414 and 420–429; ICD-10: I20–I25), stroke (ICD-9: 430–438 and ICD-10: I60–I69), and other circulatory diseases (ICD-9: 390–392, 393–398, 401–405, and 440; ICD-10: I10–I15, I01–I02, I05–I09, I27, I30–I52, I70, and I71). For cancer mortality, we used codes 140–208 of ICD-9 and codes C00–C97 of ICD-10. The accuracy of cause-of-death coding in Taiwan, China has been estimated to be between 81% and 84%.[19]

2.5. Statistical analyses

Considering the strong evidence of sex-specific associations between OPA and mortality,[7–9] we stratified all analyses by biological sex assigned at birth. As BMI may be considered a potential mediator of the association between physical activity and mortality,[9,20] we also carried out a sensitivity analysis excluding it.

2.5.1. Baseline OPA and joint OPA–LTPA analyses

We estimated the associations between OPA (alone and jointly with LTPA) and the 3 mortality outcomes using Cox regression. We examined the proportional hazards assumption by testing interactions with log(time). In CVD and cancer mortality analyses, we accounted for competing risk by using the Fine-Gray sub-distribution method. In Model 1 (crude model), we adjusted for age (year) and birth cohorts.[9] In Model 2, we additionally adjusted for education, occupation type, and marital status. Finally, for Model 3 we added alcohol status, fruit and vegetable intake, sleep duration, smoking status/pack years, and BMI; the baseline OPA models were also adjusted for LTPA.

2.5.2. Changes in OPA analyses

We entered baseline OPA level and change over time as a dual exposure in Cox regression models. Fine-Gray sub-distribution method was used in CVD and cancer mortality analyses. All models were adjusted for all potential confounders mentioned above in the baseline OPA analyses, which they were treated as time-varying covariates.

We reported this study as per the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines (Supplementary STROBE Checklist) (Supplementary Table 2).

2.5.3. Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

3. Results

3.1. Prospective analyses (baseline OPA and LTPA)

Table 1 presents the baseline characteristics of the sample entered in the baseline OPA analyses by OPA level. Supplementary Table 3 presents the sample's characteristics by the original 4-level OPA variable. Supplementary Fig. 1 shows the baseline sample selection process. The mean age of the 171,934 men and 177,314 women entered in these analyses was 38.9 ± 11.5 and 39.2 ± 12.5 years, respectively (mean ± SD). Over a mean mortality follow-up of 16.2 ± 5.5 years for men (2,776,908 person-years) and 16.4 ± 5.4 years for women (2,906,946 person-years), there were 11,696 deaths (2033 of CVD and 4631 of cancer causes) in men and 8980 deaths (1475 of CVD and 3689 of cancer causes) in women. Supplementary Fig. 2 shows the distribution of OPA by occupation type.

3.1.1. Baseline OPA

In the minimally adjusted analyses, the moderate and combined moderately heavy/heavy OPA levels were detrimentally associated with all-cause mortality in men but

Table 1
Characteristics of the sample entered in the baseline analyses by sex and occupational physical activity level.

	Male (n = 171,934)				Female (n = 177,314)			
	All males	Light	Moderate	Moderately heavy/ Heavy[a]	All females	Light	Moderate	Moderately heavy/ Heavy[a]
n (core sample)	171,934	96,812	46,671	28,451	177,314	114,721	50,745	11,848
Age (year)	38.9 ± 11.5	39.0 ± 11.1	37.7 ±11.1	40.4 ± 13.1	39.2 ± 12.5	38.2 ± 12.1	40.8 ± 12.9	42.3 ± 13.1
Follow-up (year)	16.2 ± 5.5	16.0 ± 5.4	16.3 ± 5.5	16.6 ± 5.8	16.4 ± 5.4	16.2 ± 5.4	16.9 ± 5.5	16.2 ± 5.7
Follow-up (person-year)	2,776,908	1,546,270	758,978	471,660	2,906,946	1,857,874	857,325	191,747
All-cause mortality (per 100,000 person-year)	421	387	383	596	309	288	343	355
Cancer death (per 100,000 person-year)	167	148	154	249	127	114	150	152
CVD death (per 100,000 person-year)	73	69	66	98	51	47	56	62
Baseline LTPA (MET- h/week)								
<1.00	35,463.0 ± 20.6	17,126.0 ± 17.7	9915.0 ± 21.2	8422.0 ± 29.6	44,738.0 ± 25.2	27,496.0 ± 24.0	13,791.0 ± 27.2	34,51.0 ± 29.1
1.00−7.49	87,064.0 ± 50.6	50,865.0 ± 52.5	23,771.0 ± 50.9	12,428.0 ± 43.7	97,892.0 ± 55.2	65,725.0 ± 57.3	26,051.0 ± 51.3	61,16.0 ± 51.6
7.50−14.99	20,371.0 ± 11.8	12,366.0 ± 12.8	5384.0 ± 11.5	2621.0 ± 9.2	19,653.0 ± 11.1	12,511.0 ± 10.9	6068.0 ± 12.0	10,74.0 ± 9.1
≥15.00	29,036.0 ± 16.9	16,455.0 ± 17.0	7601.0 ± 16.3	4980.0 ± 17.5	15,031.0 ± 8.5	8989.0 ± 7.8	4835.0 ± 9.5	1207.0 ± 10.2
BMI group (kg/m^2)[b]								
Underweight	5992.0 ± 3.5	2872.0 ± 3.0	1900.0 ± 4.1	1220.0 ± 4.3	24,482.0 ± 13.8	16,867.0 ± 14.7	6260.0 ± 12.3	1355.0 ± 11.4
Normal	83,574.0 ± 48.6	46,437.0 ± 48.0	22,932.0 ± 49.1	14,205.0 ± 49.9	110,924.0 ± 62.6	73,533.0 ± 64.1	30,532.0 ± 60.2	6859.0 ± 57.9
Overweight	52,798.0 ± 30.7	30,674.0 ± 31.7	13,928.0 ± 29.8	8196.0 ± 28.8	26,464.0 ± 14.9	15,431.0 ± 13.5	8760.0 ± 17.3	2273.0 ± 19.2
Obese	29,570.0 ± 17.2	16,829.0 ± 17.4	7911.0 ± 17.0	4830.0 ± 17.0	15,444.0 ± 8.7	8890.0 ± 7.7	5193.0 ± 10.2	1361.0 ± 11.5
Education								
Junior high school or less	20,560.0 ± 12.0	5817.0 ± 6.0	5673.0 ± 12.2	9070.0 ± 31.9	37,873.0 ± 21.4	16,392.0 ± 14.3	16,201.0 ± 31.9	5280.0 ± 44.6
Senior high school	32,805.0 ± 19.1	11,650.0 ± 12.0	11,991.0 ± 25.7	9164.0 ± 32.2	38,325.0 ± 21.6	20,352.0 ± 17.7	14,820.0 ± 29.2	3153.0 ± 26.6
College or higher	118,569.0 ± 69.0	79,345.0 ± 82.0	29,007.0 ± 62.2	10,217.0 ± 35.9	101,116.0 ± 57.0	77,977.0 ± 68.0	19,724.0 ± 38.9	3415.0 ± 28.8
Smoking		NA	NA	NA		NA	NA	NA
Never	88,858.0 ± 51.7	56,036.0 ± 57.9	21,618.0 ± 46.3	11,204.0 ± 39.4	162,338.0 ± 91.6	106,493.0 ± 92.8	45,482.0 ± 89.6	10,363.0 ± 87.5
Former	17,001.0 ± 9.9	9527.0 ± 9.8	4432.0 ± 9.5	3042.0 ± 10.7	3243.0 ± 1.8	1780.0 ± 1.6	1163.0 ± 2.3	300.0 ± 2.5
Current	66,075.0 ± 38.4	31,249.0 ± 32.3	20,621.0 ± 44.2	14,205.0 ± 49.9	11,733.0 ± 6.6	6448.0 ± 5.6	4100.0 ± 8.1	1185.0 ± 10.0
Smoking (pack-year)								
Never	88,858.0 ± 51.7	56,036.0 ± 57.9	21,618.0 ± 46.3	11,204.0 ± 39.4	162,338.0 ± 91.6	106,493.0 ± 92.8	45,482.0 ± 89.6	10,363.0 ± 87.5
Former: <1.875	4232.0 ± 2.5	2517.0 ± 2.6	1098.0 ± 2.4	617.0 ± 2.2	1785.0 ± 1.0	962.0 ± 0.8	645.0 ± 1.3	178.0 ± 1.5
Former: 1.875−<9.000	6367.0 ± 3.7	3643.0 ± 3.8	1665.0 ± 3.6	1059.0 ± 3.7	1165.0 ± 0.7	656.0 ± 0.6	414.0 ± 0.8	95.0 ± 0.8
Former: ≥9.000	6402.0 ± 3.7	3367.0 ± 3.5	1669.0 ± 3.6	1366.0 ± 4.8	293.0 ± 0.2	162.0 ± 0.1	104.0 ± 0.2	27.0 ± 0.2
Current: <1.875	6218.0 ± 3.6	3044.0 ± 3.1	2043.0 ± 4.4	1131.0 ± 4.0	2968.0 ± 1.7	1610.0 ± 1.4	1022.0 ± 2.0	336.0 ± 2.8
Current: 1.875−<9.000	23,822.0 ± 13.9	11,567.0 ± 11.9	7661.0 ± 16.4	4594.0 ± 16.1	6067.0 ± 3.4	3324.0 ± 2.9	2159.0 ± 4.3	584.0 ± 4.9
Current: ≥9.000	36,035.0 ± 21.0	16,638.0 ± 17.2	10,917.0 ± 23.4	8480.0 ± 29.8	2698.0 ± 1.5	1514.0 ± 1.3	919.0 ± 1.8	265.0. ± 2.2
Alcohol								
Former	6416.0 ± 3.7	2939.0 ± 3.0	1875.0 ± 4.0	1602.0 ± 5.6	2301.0 ± 1.3	1135.0 ± 1.0	924.0 ± 1.8	2420 ± 2.0
Seldom[c]	160,236.0 ± 93.2	91,820.0 ± 94.8	43,276.0 ± 92.7	25,140.0 ± 88.4	173,979.0 ± 98.1	113,086.0 ± 98.6	49,390.0 ± 97.3	11,503.0 ± 97.1
Current	5282.0 ± 3.1	2053.0 ± 2.1	1520.0 ± 3.3	1709.0 ± 6.0	1034.0 ± 0.6	500.0 ± 0.4	431.0 ± 0.8	103.0 ± 0.9
Fruit and vegetable intake (servings/day)								
<3	70,613.0 ± 41.1	38,119.0 ± 39.4	19,726.0 ± 42.3	12,768.0 ± 44.9	64,453.0 ± 36.3	41,943.0 ± 36.6	17,752.0 ± 35.0	4758.0 ± 40.2
3−4	84,185.0 ± 49.0	48,945.0 ± 50.6	22,440.0 ± 48.1	12,800.0 ± 45.0	90,452.0 ± 51.0	58,928.0 ± 51.4	25,955.0 ± 51.1	5569.0 ± 47.0
≥5	17,136.0 ± 10.0	9748.0 ± 10.1	4505.0 ± 9.7	2883.0 ± 10.1	22,409.0 ± 12.6	13,850.0 ± 12.1	7038.0 ± 13.9	1521.0 ± 12.8

(*continued on next page*)

Table 1 (Continued)

	Male (n = 171,934)				Female (n = 177,314)			
	All males	Light	Moderate	Moderately heavy/ Heavy[a]	All females	Light	Moderate	Moderately heavy/ Heavy[a]
Sleep (h/day)								
<4	1325.0 ± 0.8	592.0 ± 0.6	386.0 ± 0.8	347.0 ± 1.2	2110.0 ± 1.2	1116.0 ± 1.0	807.0 ± 1.6	187.0 ± 1.6
4−6	33,877.0 ± 19.7	17,921.0 ± 18.5	9803.0 ± 21.0	6153.0 ± 21.6	35,024.0 ± 19.8	20,690.0 ± 18.0	11,337.0 ± 22.3	2997.0 ± 25.3
>6−8	126,010.0 ± 73.3	72,411.0 ± 74.8	33,806.0 ± 72.4	19,793.0 ± 69.6	124,736.0 ± 70.3	83,357.0 ± 72.7	33,737.0 ± 66.5	7642.0 ± 64.5
>8	10,722.0 ± 6.2	5888.0 ± 6.1	2676.0 ± 5.7	2158.0 ± 7.6	15,444.0 ± 8.7	9558.0 ± 8.3	4864.0 ± 9.6	1022.0 ± 8.6
Chronic lung disease	5567.0 ± 3.2	3378.0 ± 3.5	1376.0 ± 2.9	813.0 ± 2.9	4861.0 ± 2.7	3185.0 ± 2.8	1363.0 ± 2.7	313.0 ± 2.6
Birth cohort								
<1940	9626.0 ± 5.6	5186.0 ± 5.4	2160.0 ± 4.6	2280.0 ± 8.0	10,929.0 ± 6.2	6489.0 ± 5.7	3661.0 ± 7.2	779.0 ± 6.6
1940−1949	13,004.0 ± 7.6	6256.0 ± 6.5	3083.0 ± 6.6	3665.0 ± 12.9	17,687.0 ± 10.0	8409.0 ± 7.3	7268.0 ± 14.3	2010.0 ± 17.0
1950−1959	25,107.0 ± 14.6	14,528.0 ± 15.0	6103.0 ± 13.1	44,760. ± 15.7	26,186.0 ± 14.8	15,514.0 ± 13.5	7916.0 ± 15.6	2756.0 ± 23.3
1960−1969	48,873.0 ± 28.4	28,706.0 ± 29.7	13,567.0 ± 29.1	6600.0 ± 23.2	43,318.0 ± 24.4	29,181.0 ± 25.4	12,311.0 ± 24.3	1826.0 ± 15.4
1970−1979	56,938.0 ± 33.1	32,466.0 ± 33.5	16,428.0 ± 35.2	8044.0 ± 28.3	56,965.0 ± 32.1	39,938.0 ± 34.8	14,358.0 ± 28.3	2669.0 ± 22.5
≥1980	18,386.0 ± 10.7	9670.0 ± 10.0	5330.0 ± 11.4	3386.0 ± 11.9	22,229.0 ± 12.5	15,190.0 ± 13.2	5231.0 ± 10.3	1808.0 ± 15.3
Occupation								
Armed forces occupations	3130.0 ± 1.8	1739.0 ± 1.8	805.0 ± 1.7	586.0 ± 2.1	428.0 ± 0.2	333.0 ± 0.3	81.0 ± 0.2	14.0 ± 0.1
Clerical support workers	9035.0 ± 5.3	7175.0 ± 7.4	1271.0 ± 2.7	589.0 ± 2.1	23,203.0 ± 13.1	21,630.0 ± 18.9	1436.0 ± 2.8	137.0 ± 1.2
Craft and related trades workers	25,952.0 ± 15.1	10,581.0 ± 10.9	7425.0 ± 15.9	7946.0 ± 27.9	7146.0 ± 4.0	3831.0 ± 3.3	2200.0 ± 4.3	1115.0 ± 9.4
Elementary laborers	4741.0 ± 2.8	438.0 ± 0.5	1471.0 ± 3.2	2832.0 ± 10.0	2484.0 ± 1.4	577.0 ± 0.5	1098.0 ± 2.2	809.0 ± 6.8
Legislators, senior officials, and managers	10,384.0 ± 6.0	8187.0 ± 8.5	1856.0 ± 4.0	341.0 ± 1.2	4561.0 ± 2.6	3991.0 ± 3.5	503.0 ± 1.0	67.0 ± 0.6
Professionals	13,422.0 ± 7.8	8529.0 ± 8.8	3246.0 ± 7.0	1647.0 ± 5.8	15,047.0 ± 8.5	9785.0 ± 8.5	4155.0 ± 8.2	1107.0 ± 9.3
Service and sales workers	41,761.0 ± 24.3	28,591.0 ± 29.5	8175.0 ± 17.5	4995.0 ± 17.6	26,063.0 ± 14.7	17,980.0 ± 15.7	6224.0 ± 12.3	1859.0 ± 15.7
Skilled agricultural, forestry, and fishery workers	54,742.0 ± 31.8	29,171.0 ± 30.1	19,722.0 ± 42.3	5849.0 ± 20.6	94,243.0 ± 53.2	55,149.0 ± 48.1	33,540.0 ± 66.1	5554.0 ± 46.9
Technicians and associate professionals	4655.0 ± 2.7	612.0 ± 0.6	1253.0 ± 2.7	2790.0 ± 9.8	1966.0 ± 1.1	245.0 ± 0.2	729.0 ± 1.4	992.0 ± 8.4
Other	4112.0 ± 2.4	1789.0 ± 1.8	1447.0 ± 3.1	876.0 ± 3.1	2173.0 ± 1.2	1200.0 ± 1.0	779.0 ± 1.5	194.0 ± 1.6

Note: Data are presented as mean ± SD.

[a] Combination of top 2 OPA categories: moderately heavy and heavy.

[b] Category cut-offs are: underweight (<18.5 kg/m^2); normal (18.5−<24.0 kg/m^2); overweight (24.0−<27.0 kg/m^2); and obese (≥27.0 kg/m^2).

[c] Less than 1 alcoholic drink per week.

Abbreviations: BMI = body mass index; CVD = cardiovascular disease; LTPA = leisure-time physical activity; MET = metabolic equivalent; NA = not available; OPA = occupational physical activity.

beneficially associated in women (Model 1 in Fig. 1). Adjusting for socioeconomic status (Model 2 in Fig. 1A and 1B) inversed the associations of the moderately heavy/heavy OPA category so that it was also shown to be beneficial in men. Further adjustment for BMI, LTPA, and other lifestyle health behaviors (Model 3 in Fig. 1A and 1B) did not materially affect these protective associations with all-cause mortality, although estimates were generally attenuated. Analyses of the 4-group baseline OPA exposure suggested that the original moderately heavy OPA group drove the above effects for men (Supplementary Fig. 3), while the moderately heavy and heavy groups were more balanced contributors in women (Supplementary Fig. 4).

Removing adjustment for BMI had minimal impact. For example, the hazard ratio (HR) for men with moderately heavy/heavy OPA changed from 0.93 (95% confidence interval (95%CI): 0.89−0.98) to 0.92 (95%CI: 0.88−0.97; data not shown). There was evidence for detrimental associations between OPA and CVD/cancer mortality in the minimally adjusted models, which were attenuated following adjustment for the rest of covariates (Supplementary Fig. 5). We found no evidence for multivariable-adjusted associations of OPA with CVD or cancer mortality in women (Supplementary Fig. 6). Additional multivariable-adjusted analyses confirmed that the beneficial OPA and all-cause mortality associations were driven by non-CVD and non-cancer mortality (Supplementary Fig. 7).

3.1.2. *Joint OPA−LTPA analyses*

Supplementary Tables 4 and 5 and Supplementary Fig. 8 show the associations of LTPA with mortality, and they reveal clear associations with non-CVD/cancer mortality only. There were graded beneficial associations with jointly higher OPA and LTPA levels and all-cause mortality in men (Fig. 2A) and women (Fig. 2B), which persisted following multivariable adjustment in most cases. For example, being in the top LTPA category in men was associated with an HR of 0.86 (95%CI: 0.78−0.94) among those with moderate OPA levels and an HR of 0.80 (95%CI: 0.72−0.88) among those in the combined moderately heavy/heavy OPA group. Associations with CVD mortality were inconsistent with any monotone dose-response relationship (Supplementary Figs. 9A and 10A). Similarly, we found little consistent evidence for joint OPA−LTPA associations with cancer in men or women (Supplementary Figs. 9B and 10B). The associations of all-cause mortality and the joint OPA−LTPA variable were driven by non-CVD/non-cancer mortality (Supplementary Fig. 11). We found no evidence for statistical interaction between OPA and LTPA across the 3 mortality outcomes. Repeating the joint OPA−LTPA analyses with the 2 exposures dichotomized to low (OPA: light and moderate; LTPA: <8 MET-h/week) and high (OPA: moderately heavy and heavy; LTPA: ≥8 MET-h/week) (Supplementary Fig. 12) produced broadly similar results to the more detailed joint categorization presented in Fig. 2, while revealing more clearly statistically significant associations in women. For both men and women, high LTPA was beneficially associated with all-cause mortality among participants in both less and more active occupations (e.g., women in the high OPA−high LTPA category had an HR of 0.81 (95%CI: 0.70−0.94), compared to the referent low OPA−low LTPA group) (Supplementary Fig 12).

3.2. *Longitudinal analyses: OPA changes*

Supplementary Fig. 13 shows the longitudinal sample selection process and Supplementary Table 6 presents the characteristics of the sample included in the OPA changes analyses. The mean age of the 53,107 men and 52,354 women entered was 36.5 ± 9.3 years and 37.1 ± 10.9 years, respectively. The 2 OPA measurements occurred 6.2 ± 4.1 years apart in men

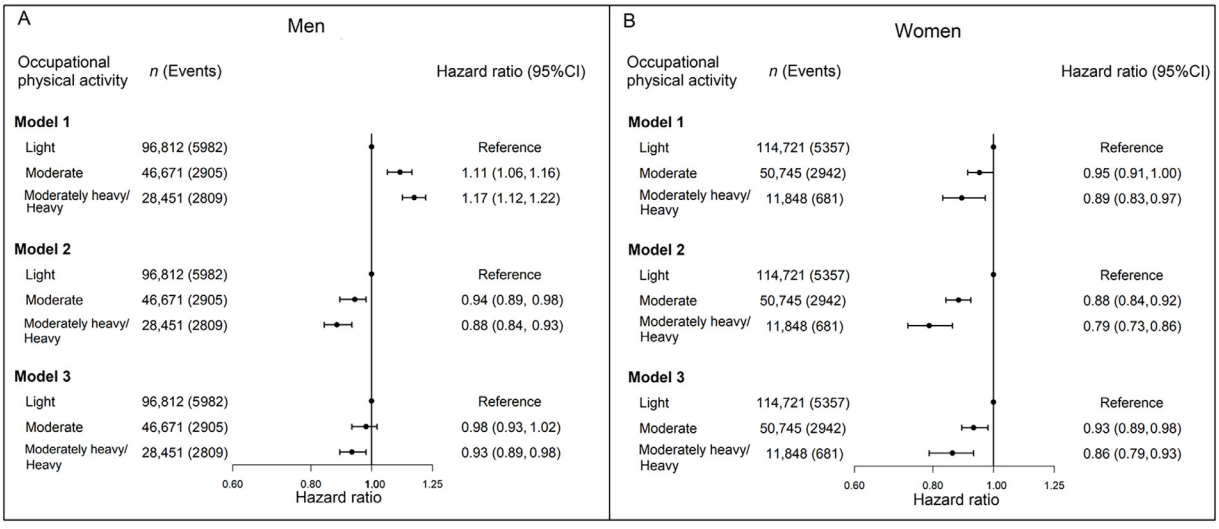

Fig. 1. Associations between baseline OPA and all-cause mortality in (A) men and (B) women. Model 1 is adjusted for age and birth cohort. Model 2 is additionally adjusted for education, occupation type, and marital status. Model 3 is additionally adjusted for sleep duration, alcohol, smoking, LTPA, and BMI. 95%CI = 95% confidence interval; BMI = body mass index; LTPA = leisure-time physical activity; OPA = occupational physical activity.

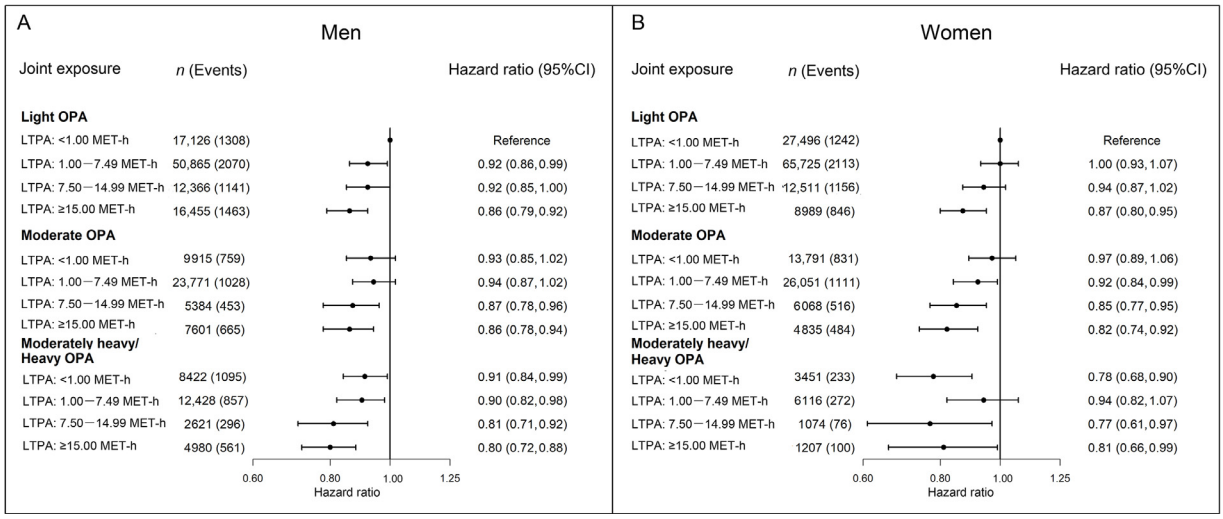

Fig. 2. Joint associations of baseline OPA and LTPA with all-cause mortality in (A) men and (B) women. Models are adjusted for age, birth cohort, education, occupation type, marital status, sleep duration, alcohol, smoking, and BMI. 95%CI = 95% confidence interval; BMI = body mass index; LTPA = leisure-time physical activity; MET = metabolic equivalent; OPA = occupational physical activity.

and 6.4 ± 4.2 years apart in women. Over a mean mortality follow-up of 12.5 ± 4.6 years for men (663,718 person-years) and 12.6 ± 4.6 years for women (662,247 person-years), there were 1600 deaths (306 of CVD and 828 of cancer causes) in men and 1847 deaths (258 of CVD and 774 of cancer causes) in women. The OPA changes sample was younger by approximately 2 years, on average, and more educated (e.g., 76.4% *vs.* 69.0% men had a degree); it was comparable across all other characteristics.

Fig. 3 (men) and Fig. 4 (women) and Supplementary Tables 7 (men) and 8 (women) present the associations of the OPA changes analyses with the 3 mortality outcomes. In the finally adjusted models, OPA decreases over time were associated with higher all-cause mortality risk in men (HR = 1.16, 95%CI: 1.01−1.33), while OPA increases were associated with lower risk in women (HR = 0.83, 95%CI: 0.70−0.97). We noted no consistent evidence of associations of OPA changes with CVD or cancer mortality in men or women. The results of the OPA changes analyses using the original 4-group OPA exposure were consistent with the effects noted above, although the detrimental association with all-cause mortality for men who increased OPA over time was more pronounced (Supplementary Figs. 14 and 15).

3.3. *Age-restricted analyses*

Repeating the Cox models in those aged ≥40 years (n = 69,419 men and n = 65,233 women in baseline OPA analyses; n = 16,682 men and n = 15,843 women in OPA changes analyses) produced a similar pattern of results to that of the

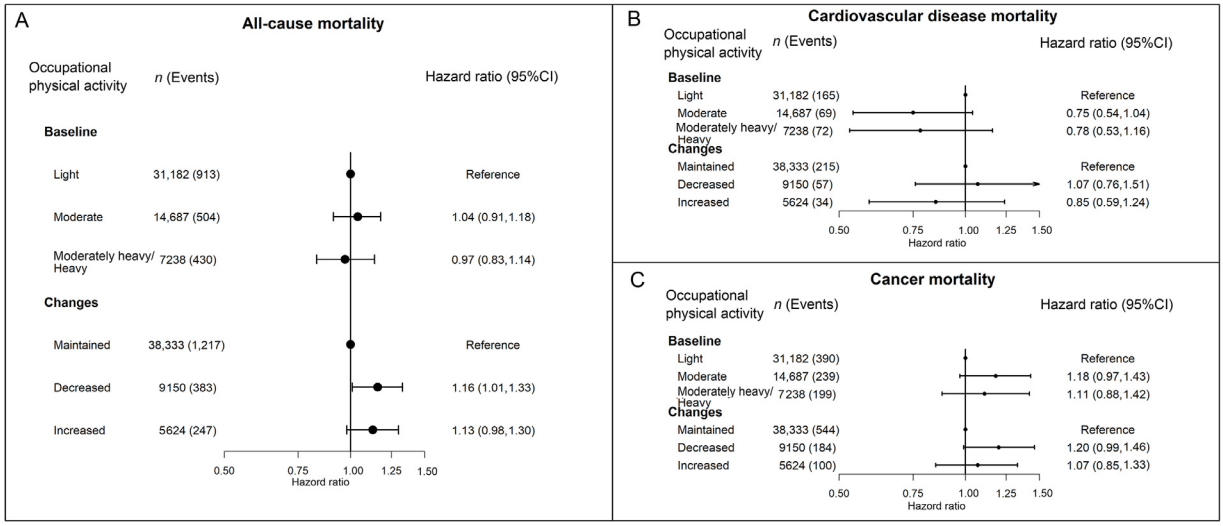

Fig. 3. Associations between OPA changes and risk of (A) all-cause, (B) CVD, and (C) cancer mortality in men. Adjusted for sleep duration, alcohol, smoking, leisure-time physical activity, and BMI. 95%CI = 95% confidence interval; BMI = body mass index; CVD = cardiovascular disease; OPA = occupational physical activity.

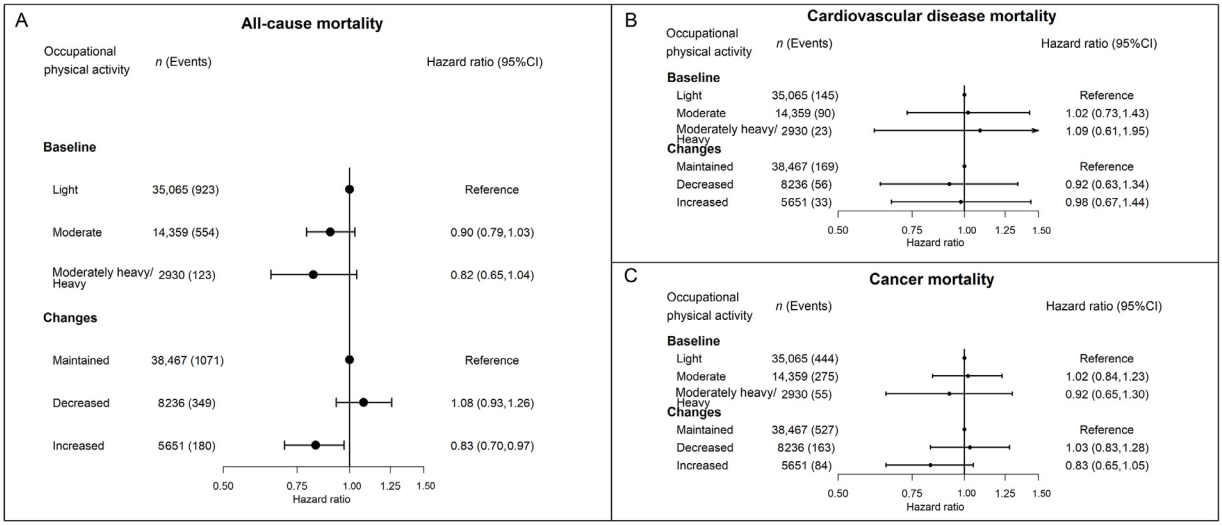

Fig. 4. Associations between OPA changes and risk of (A) all-cause, (B) CVD, and (C) cancer mortality in women. Adjusted for sleep duration, alcohol, smoking, LTPA, and BMI. 95%CI = 95% confidence interval; BMI = body mass index; CVD = cardiovascular disease; LTPA = leisure-time physical activity; OPA = occupational physical activity.

main analyses, albeit any observed associations were more pronounced (Supplementary Figs. 16 and 17). For example, the all-cause mortality HR for men aged ≥40 moderately heavy/heavy baseline OPA was 0.88 (95%CI: 0.84−0.93) *vs.* 0.93 (95%CI: 0.89−0.98) in the whole male sample.

4. Discussion

Despite the occupational domain roots of physical activity epidemiology,[5,6] uncertainty surrounds the health effects of OPA in recent years due to numerous limitations in the current literature and the resulting inconsistencies in findings. This uncertainty is more pronounced in non-Western countries which, to date, have contributed very little to the OPA evidence base.[7,8,11] Our study is the world's first longitudinal investigation into the changes in OPA and mortality and the second large prospective study[11] with all-cause and cause-specific mortality in a non-Western country. We found that heavier baseline OPA levels were associated with lower all-cause mortality in both men and women. The OPA changes analyses produced mixed sex group-specific findings that collectively lend some modest support to the baseline OPA data-based conclusions. These findings are novel and inform global public health practice by expanding the pool of evidence beyond Western counties.

4.1. Baseline OPA and LTPA

The beneficial associations of baseline OPA with all-cause mortality in both men and women partly contradict the results of a recent meta-analysis[7] where men in highly active jobs had 18% (5%−34%) higher mortality than men in light OPA jobs, while women's pooled estimates were of similar magnitude to our beneficial estimates but not statistically significant (10% lower risk, −1% to 20%). Only 2 of the 33 studies included in this systematic review[7] were from non-Western countries, and our results are in broad agreement with the largest of them. In the Golestan Cohort Study (Iran, $n = 50{,}045$),[21] participants in level 2 jobs (standing/occasional walking, i.e., roughly equivalent to "moderately heavy" in the MJ Cohort) had 76% (95%CI: 16%−168%) (women), and those in Level 1 jobs (mostly sitting) had higher (men, 51% (95%CI: 21%−88%); women, 47% (95%CI: 5%−106%)) mortality risk than those in Level 4 jobs (highly active outdoor jobs, roughly equivalent to our collapsed "moderately heavy/heavy" category). A more recent analysis of the China Kadoorie Biobank[11] reported no associations between OPA grouping (mainly sedentary, standing occupation, and manual occupation) and all-cause or CVD mortality in the whole sample ($n = 142{,}302$). However, subgroup analyses revealed differences among socioeconomic status and LTPA groups. They found beneficial associations of OPA and mortality in the least educated but harmful associations in the most educated and those who do LTPA regularly. Collectively, these studies further highlight the complexity of OPA as a health exposure. We found lower (men, 15%; women, 19%) all-cause mortality risk among participants in heavy occupations who reported at least 8 MET-h/week of LTPA at baseline, a finding that is in line with a recent systematic review that concluded regular recreational physical activity is beneficial even among highly active workers.[22]

Our baseline OPA all-cause mortality findings are in partial agreement with one[9] but not the other[10] of the large Nordic cohorts. In the Norwegian study,[9] the HRs (which were only presented as smoothed curves in the appendix) were in fair agreement with our findings in men. In their main analyses of survival times, Dalene and collegues[9] found beneficial associations of baseline OPA with life expectancy and all-cause mortality in men only.

We did not replicate the contrasting OPA (detrimental) and LTPA (beneficial) associations, known as the "physical activity paradox",[23,24] reported in the Danish study[10] and the 2018 meta-analysis (in men only).[7] Our findings confirmed the well-established[1] dose−response associations between LTPA

and all-cause[25] mortality and revealed additive benefits in highly active men and women in both domains. Although the direction of the LTPA associations was favorable in general, our results were less conclusive than meta-analyses of LTPA with CVD or cancer[26] mortality. The weak evidence of associations between both physical activity domains and CVD and cancer mortality in our study is less aligned with recent OPA literature[8,10] and large-scale evidence syntheses.[1] Our results may reflect differences in the distribution of death between Western counties (including Nordic) and South-East Asia. For example, in South-East Asia, CVD-related causes of death represent a much smaller proportion of total mortality than in Northern and Western European countries.[27,28] In the baseline analyses of the MJ Cohort, only 17% of total deaths were due to CVD causes, which is the same as in a recent Chinese OPA study,[11] but considerably than the 27% exhibited by their Norwegian cohort counterparts.[9]

4.2. OPA Changes

The mixed findings of the OPA changes analyses provide some support, albeit modest, to the interpretation of the baseline OPA data. Temporal decreases in a beneficial exposure should at least theoretically be associated with deterioration in risk, and *vice versa*. Both of these findings were present in our study but in different sex groups: OPA decreases in men were associated with higher and OPA increases in women with lower all-cause mortality risk as compared to stable OPA in each sex group. The direction and magnitude of these sex-specific associations support this theoretical paradigm (16% higher risk for men's OPA decreases and 17% lower risk for women's OPA increases). The higher risk associated with men's OPA increases (Fig. 3), a finding that was more pronounced when we analysed the original 4-group OPA exposure (Supplementary Fig. 14), prevents us from confidently confirming the paradigm based on our longitudinal data. Assuming that the 2 aforementioned sex-specific findings are not isolated but part of a consistent pattern, we speculate that they may be partly explained by healthy worker effects (i.e., men whose health deteriorated or who developed chronic disease moved to less active occupations, while women who stayed in good health increased their OPA level by being assigned to more physically demanding roles). We acknowledge that although we excluded those who developed CVD and cancer prior to the second OPA assessment, development of other chronic conditions compromising workers' physical capacity may still be at play.

5. Strengths and weaknesses

We used a large population sample with long follow-up times and repeated OPA measures. Our study is one of the largest analyses of OPA and health[7,8] to directly address at least 2 major gaps identified by the WHO's recent guidelines[4,8]: (a) the need for evidence from non-Western countries, and b) the need to understand domain-specific effects of physical activity. We adjusted for a comprehensive list of potential confounders, including occupation type and sleep. We took extensive measures against reverse causation (i.e., healthier participants employed in active occupations and doing more LTPA) and undiagnosed/occult disease.

Our study also has some limitations. We were able to examine OPA changes in only 30% of the sample entered in the baseline OPA analyses, which might have compromised generalizability and statistical power in the OPA changes analyses,[29] although it is unlikely that this fully explains the less clear longitudinal effects, as the 2 samples differed relatively little. The completeness of the death file data in Taiwan, China is not known.[17,18] Considering that work cultures and work environments are highly variable, it is not clear to what extend our findings are generalizable outside of Taiwan, China. Modelling changes in OPA is not sufficient for causal inference. We did not make full use of all available OPA measurement points. Like in most previous studies,[7–10] the measurement of OPA used in the MJ Cohort was crude, not formally validated against a criterion objective measure, and captured the general nature of OPA but not the detailed characteristics (e.g., frequency, duration, intensity) that would allow for more in-depth assessment. Exposure imprecision might have led to the attenuation of the associations we observed for both OPA and LTPA. Such imprecision may lead to more pronounced exposure misclassification in the OPA changes analyses that consider OPA responses at 2 time points. Estimates were more pronounced in the sensitivity analyses restricted to individuals ≥40 years of age, suggesting that the inclusion of younger participants aged 30−39 years may have diluted associations in the main analyses. Although we were able to control for potential confounders other studies in the field could not consider (e.g., diet, alcohol, work type), we cannot exclude the possibility of unmeasured confounding, such as the absence of information on working conditions or sedentary leisure-time behavior. Differential unmeasured confounding between areas or countries may also have contributed to differences in the OPA and LTPA findings between our study and the recent Danish cohort.[10] Although the MJ Cohort is not representative of the target population[12] (like the large majority of cohort studies), recent empirical work has shown that the poor cohort representativeness does not materially influence the associations between physical activity and mortality.[30]

6. Conclusion

We found that heavier baseline OPA was beneficially associated with non-CVD/non-cancer mortality in a large cohort of male and female workers in Taiwan, China. Our longitudinal results did provide some sex-specific support to the associations of baseline OPA with mortality that we and others[8,9] have reported. Considering that the literature to date[7–10] has exclusively relied on such baseline measurement-only designs, our study provides some partial support to public health guidelines that consider physical activity at work to be health enhancing.[1] Since OPA trials with mortality endpoints are infeasible, our study emphasizes the need for more research investment into population studies designed to better

understand how changes in OPA over time modulate mortality risk. For example, future observational research in this area will benefit from longitudinal studies with more refined measurements of OPA to capture volume, intensity, frequency, posture, and nature (e.g., lifting and/or carrying, walking, handling items while standing) of activities involved in daily work routines, such as the wearable device-based measurements used in other incidental physical activity research.[31–33] Such granular OPA evidence would allow for the refinement of public health guidelines[1] and interventions, which are currently based on a limited body of methodologically weak evidence.[8]

Acknowledgments

The authors thank the participants for their participation in the MJ Health Management Institution prospective cohort study. All of the data used in this research were authorized for use by the MJ Health Resource Centre (Authorization code: MJHRF2019013A) and by the Health and Welfare Data Science Centre. Any interpretations or conclusions described in this paper do not represent the views of the MJ Health Resource Centre. This work was supported by a National Health and Medical Research Council (Australia) Investigator Grant (APP1194510).

Authors' contributions

ES conceived the idea, designed the study, accessed and verified the data and drafted the manuscript; TLE conducted the initial analyses and accessed and verified the data; BHH conducted all analytic revisions and accessed and verified the data; MNA conducted all analytic revisions, accessed and verified the data, and assisted with drafting the manuscript; SP assisted with drafting the manuscript; ATP advised on all statistical aspects and critically revised the manuscript for important intellectual content; LJC, BdPC, YJL, AH, and PWK revised the manuscript for important intellectual content. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Competing interests

The authors declare that they have no competing interests.

Data sharing

Applicants for data access should contact MJ Health Research Foundation (http://www.mjhrf.org/).

Supplementary materials

Supplementary materials associated with this article can be found in the online version at doi:10.1016/j.jshs.2024.03.002.

References

1. 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.
2. Lee IM, Shiroma EJ, Lobelo F, et al. Effect of physical inactivity on major non-communicable diseases worldwide: An analysis of burden of disease and life expectancy. *The Lancet* 2012;**380**:219–29.
3. Piercy KL, Troiano RP, Ballard RM, et al. The physical activity guidelines for Americans. *JAMA* 2018;**320**:2020–8.
4. DiPietro L, Al-Ansari S, Biddle S, et al. Advancing the global physical activity agenda: Recommendations for future research by the 2020 WHO physical activity and sedentary behavior guidelines development group. *Int J Behav Nutr Phys Act* 2020;**17**:143. doi:10.1186/s12966-020-01042-2.
5. Morris JN, Heady JA, Raffle PA, et al. Coronary heart-disease and physical activity of work. *The Lancet* 1953;**262**:1053–7.
6. Paffenbarger RS, Hale WE. Work activity and coronary heart mortality. *N Engl J Med* 1975;**292**:545–50.
7. Coenen P, Huysmans MA, Holtermann A, et al. Do highly physically active workers die early? A systematic review with meta-analysis of data from 19,3696 participants. *Br J Sports Med* 2018;**52**:1320–6.
8. Cillekens B, Lang M, van Mechelen W, et al. How does occupational physical activity influence health? An umbrella review of 23 health outcomes across 158 observational studies. *Br J Sports Med* 2020;**54**:1474–81.
9. Dalene KE, Tarp J, Selmer RM, et al. Occupational physical activity and longevity in working men and women in Norway: A prospective cohort study. *Lancet Public Health* 2021;**6**:e386–95.
10. Holtermann A, Schnohr P, Nordestgaard BG, Marott JL. The physical activity paradox in cardiovascular disease and all-cause mortality: The contemporary Copenhagen General Population Study with 104,046 adults. *Eur Heart J* 2021;**42**:1499–511.
11. Luo M, Gupta N, Holtermann A, et al. Revisiting the "physical activity paradox" in a Chinese context: Occupational physical activity and mortality in 142,302 urban working adults from the China Kadoorie Biobank study. *Lancet Reg Health West Pac* 2022;**23**: 100457. doi:10.1016/j.lanwpc.2022.100457.
12. Wu X, Tsai SP, Tsao CK, et al. Cohort profile: "The Taiwan" MJ Cohort: Half a million Chinese with repeated health surveillance data. *Int J Epidemiol* 2017;**46**:1744−44g. doi:10.1093/ije/dyw282.
13. Chen LJ, Hamer M, Lai YJ, Huang BH, Ku PW, Stamatakis E. Can physical activity eliminate the mortality risk associated with poor sleep? A 15-year follow-up of 341,248 MJ Cohort participants. *J Sport Health Sci* 2022;**11**:596–604.
14. Martinez-Gomez D, Esteban-Cornejo I, Lopez-Garcia E, et al. Physical activity less than the recommended amount may prevent the onset of major biological risk factors for cardiovascular disease: A cohort study of 19,8919 adults. *Br J Sports Med* 2020;**54**:238–44.
15. Martinez-Gomez D, Cabanas-Sanchez V, Yu T, et al. Long-term leisure-time physical activity and risk of all-cause and cardiovascular mortality: Dose−response associations in a prospective cohort study of 210,327 "Taiwanese" adults. *Br J Sports Med* 2022;**56**:919–26.
16. Ainsworth BE, Haskell WL, Herrmann SD, et al. 2011 Compendium of Physical Activities: A second update of codes and MET values. *Med Sci Sports Exerc* 2011;**43**:1575–81.
17. Wen CP, Wai JP, Tsai MK, et al. Minimum amount of physical activity for reduced mortality and extended life expectancy: A prospective cohort study. *The Lancet* 2011;**378**:1244–53.
18. Tu H, Wen CP, Tsai SP, et al. Cancer risk associated with chronic diseases and disease markers: Prospective cohort study. *BMJ* 2018;**360**:k134. doi:10.1136/bmj.k134.
19. Lu TH, Lee MC, Chou MC. Accuracy of cause-of-death coding in Taiwan, China: Types of miscoding and effects on mortality statistics. *Int J Epidemiol* 2000;**29**:336–43.
20. Ekelund U, Kolle E, Steene-Johannessen J, et al. Objectively measured sedentary time and physical activity and associations with body weight

gain: Does body weight determine a decline in moderate and vigorous intensity physical activity? *Int J Obes (Lond)* 2017;**41**:1769–74.
21. Etemadi A, Abnet CC, Kamangar F, et al. Impact of body size and physical activity during adolescence and adult life on overall and cause-specific mortality in a large cohort study from Iran. *Eur J Epidemiol* 2014;**29**:95–109.
22. Prince SA, Rasmussen CL, Biswas A, et al. The effect of leisure time physical activity and sedentary behaviour on the health of workers with different occupational physical activity demands: A systematic review. *Int J Behav Nutr Phys Act* 2021;**18**:100. doi:10.1186/s12966-021-01166-z.
23. Quinn TD, Kline CE, Nagle E, Radonovich LJ, Alansare A, Barone Gibbs B. Cardiovascular responses to physical activity during work and leisure. *Occup Environ Med* 2022;**79**:94–101.
24. Quinn TD, Lane A, Pettee Gabriel K, et al. Thirteen-year associations of occupational and leisure-time physical activity with cardio-respiratory fitness in CARDIA. *Med Sci Sports Exerc* 2023;**55**:2025–34.
25. Arem H, Moore SC, Patel A, et al. Leisure time physical activity and mortality: A detailed pooled analysis of the dose−response relationship. *JAMA Intern Med* 2015;**175**:959–67.
26. Moore SC, Lee IM, Weiderpass E, et al. Association of leisure-time physical activity with risk of 26 types of cancer in 1.44 million adults. *JAMA Intern Med* 2016;**176**:816–25.
27. GBD 2016 Causes of Death Collaborators. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980−2016: A systematic analysis for the Global Burden of Disease Study 2016. *The Lancet* 2017;**390**:1151–210.
28. Institute for Health Metrics and Evaluation. *Global Burden of Disease, Taiwan (Province of China): What causes the most death and disability combined?* Available at: https://www.healthdata.org/taiwan-province-china. [accessed 15.08.2022].
29. Concato J, Peduzzi P, Holford TR, Feinstein AR. Importance of events per independent variable in proportional hazards analysis I. Background, goals, and general strategy. *J Clin Epidemiol* 1995;**48**:1495–501.
30. Stamatakis E, Owen KB, Shepherd L, Drayton B, Hamer M, Bauman AE. Is cohort representativeness passé? Poststratified associations of lifestyle risk factors with mortality in the UK Biobank. *Epidemiology* 2021;**32**:179–88.
31. Ahmadi M, Hamer M, Gill J, et al. Brief bouts of device-measured intermittent lifestyle physical activity, major adverse cardiovascular events, and mortality in non-exercisers. *Lancet Public Health* 2023;**8**:e800−10.
32. Stamatakis E, Ahmadi M, Friedenreich CM, et al. Vigorous intermittent lifestyle physical activity and cancer incidence in non-exercisers: The UK Biobank accelerometry study. *JAMA Oncol* 2023;**9**:1255–9.
33. Stamatakis E, Ahmadi MN, Gill JMR, et al. Association of wearable device-measured vigorous intermittent lifestyle physical activity with mortality. *Nat Med* 2022;**28**:2521–9.