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# Accelerometer-Measured Physical Activity and Mortality in Multiethnic Women Ages 63–99 Years

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## Abstract

**Objectives—**To prospectively examine associations between accelerometer-measured physical activity (PA) and mortality in older women, with an emphasis on light intensity PA.

**Design—**Prospective cohort study with baseline data collection between March 2012 and April 2014.

**Setting—**Women's Health Initiative cohort in the United States.

**Participants—**Community-dwelling women, ages 63–99 (N=6,382).

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**AUTHOR CONTRIBUTIONS:** All authors participated in selected portions or all of the planning, design and execution of the study protocol described in this manuscript. MJL drafted this manuscript in collaboration with all co-authors, each of whom has reviewed, edited, and approved of the final manuscript.

**Measurements—**Minutes per day of usual PA measured by hip worn triaxial accelerometers; physical functioning measured using the SPPB test; mortality follow-up for a mean 3.1 years through September 2016 (450 deaths).

**Results—**When adjusted for accelerometer wear time, age, race-ethnicity, education, smoking, alcohol, self-rated health, and comorbidities, relative risks (95% confidence intervals) for all-cause mortality across PA tertiles were 1.00 (referent), 0.86 (0.69, 1.08), 0.80 (0.62, 1.03) trend $P$=.07, for low light; 1.00, 0.57 (0.45, 0.71), 0.47 (0.35, 0.61) trend $P$<.001, for high light; and, 1.00, 0.63 (0.50, 0.79), 0.42 (0.30, 0.57) trend $P$<.001, for moderate-to-vigorous PA (MVPA). Associations remained significant for high light and MVPA (trend $P$<.001, each) after further adjustment for physical function. Each 30-min/d increment in light (low and high combined) and MVPA was associated, on average, with multivariable relative risk reductions of 12% and 39%, respectively ($P$<.01, each). After further simultaneous adjustment for each intensity, the inverse associations remained significant (light: 0.93 (0.89, 0.97); MVPA: 0.67 (0.58, 0.78)). These relative risks did not differ between subgroups for age or race-ethnicity (interaction, $P$≥ .14, all).

**Conclusions—**When measured by accelerometers, both light intensity and MVPA are associated with reduced mortality in older women. These findings suggest replacing sedentary time with light PA is a public health strategy that could benefit an aging society and warrants further investigation.

### Keywords

Aging; Women's health; Physical activity; Epidemiology; Longevity

## INTRODUCTION

Age-related deterioration in health is associated with reduced physical activity (PA) levels.[1,2] U.S.[3] and international[4] guidelines on PA and public health recommend healthy older adults should do at least 2.5 hours/week of moderate-intensity or 1.25 hours/week of vigorous-intensity aerobic PA for health benefits, a target which few older U.S. adults meet[5] often because they are not capable of engaging in MVPA. Substantially lower all-cause mortality risk is associated with relatively high MVPA levels (3–5 times guideline recommended) assessed by questionnaires.[6,7] The extent to which this extends to older adults is unclear. Typically, only 10%–20% of the variance in device-measured PA is explained by self-reported activity.[8] PA misclassification is large in older adults,[9] especially for light intensity PA which is common in these individuals but currently not recommended for public health. Use of accelerometers to measure PA is novel in prospective studies on older adults and provides the ability to calibrate the effect of PA much better than with self-report, especially for light PA.

Only a few studies have reported on accelerometer measured PA and health outcomes, typically all-cause mortality, specifically in older adults.[10–12] These studies have classified PA using accelerometer cutpoints derived from younger populations, which can result in misclassification in older adults[13] because the energy costs of PA are greater for older people. More data are needed to understand whether associations between PA and mortality are similar for younger old and older old adults, and whether any PA benefit is attenuated by chronic disease burden or functional limitations. In this study, we examined associations

between mortality and accelerometer measured PA using age-relevant intensity cutpoints in multiethnic older women.

## METHODS

### Study Population

Women in the present study were enrolled in the Objective Physical Activity and Cardiovascular Health (OPACH) Study, a prospective investigation of accelerometer measured PA and cardiovascular disease in women ages 63–99 years, ancillary to the Women's Health Initiative (WHI). Details regarding implementation of the WHI[14] and OPACH[15] studies have been published. During 2012–2013, 7,875 women consented to participate in the WHI Long Life Study that included in-home examinations[15] comprised of health questionnaires, anthropometric measurements, and a physical functioning test (Short Physical Performance Battery, SPPB[16]). A subset of 7,048 women further consented to participate in the OPACH study and received an accelerometer, wear instructions, and a sleep log.[15] After the wear interval, accelerometers and logs were mailed to the WHI coordinating center. Almost all women (95.3%) returned their accelerometer, of these 6,489 (96.5%) had usable data for analysis.

### Accelerometer Measurement

Participants were asked to wear a triaxial accelerometer (ActiGraph GT3X+; Pensacola, FL) on their hip for 7 consecutive days during waking and sleeping hours except when bathing or swimming. Acceleration data from the three planes was processed with ActiLife software (version 6) using 15 second epochs and the normal filter, and then integrated in a vector magnitude (VM).[15] The VM counts were averaged and reported as mean total PA, an indicator of total PA volume. The VM counts also were categorized into intensity-specific PA levels as low light intensity (19–225 counts/15 sec), high light intensity (226–518), and MVPA (≥519) using cutpoints derived from the OPACH calibration study[17] (Supplemental Table e1).

Accelerometer wear was identified using information from the sleep logs and a computer-based automated algorithm[18] and non-wear using a standard protocol.[19] Sleep time was excluded using data from sleep logs. To be included in the present analysis, we required at least 4 days with ≥10 hours/day of awake wear-time (convention for compliant wear) resulting in a sample size of 6,382 women.

### Mortality Ascertainment

Mortality surveillance was completed annually through mailed outcomes questionnaires augmented by National Death Index searches, proxy queries, obituaries, and hospital records to identify fatalities.[20] The primary outcome was all-cause mortality through September 30, 2016. Cardiovascular disease (CVD) and cancer mortality were secondary outcomes. Trained physicians reviewed death certificates and other medical records to adjudicate cause of death. International Classification of Disease (10$^{th}$ revision) codes I00-09 and I60-69 identified CVD, and C00-97 identified cancer, as the underlying causes of death.

### Statistical Analysis

Time-to-event was accrued from the first day of accelerometer monitoring to the date of death, loss to follow-up, or September 30, 2016, whichever came first. Hazard ratios were estimated from Cox regression analysis as measures of the relative risk (RR) with 95% confidence intervals (CI) for mortality according to PA levels. Models included progressive adjustment for potential confounders, beginning with accelerometer awake wear-time and age (Model 1), then adding race-ethnicity, education, smoking, alcohol, age at menopause, self-rated general health, and number of comorbidities (Model 2). Physical function, available in 5,479 (86%) of participants, was an assumed mediator of the association between PA and mortality, and was added last (Model 3). While mean accelerometer measured total PA was slightly higher in women with (336.8 min/d) than without (327.2 min/d, $P<.001$) physical function scores, mean age (78.7 vs 78.7 years, $P=.90$), awake wear-time (14.8 vs 14.8 hr/d, $P=.83$), and all-cause death rate (21.9 vs 27.1 per 1000 person years, $P=.74$) were not different. Tertiles of total and intensity-specific PA were modeled, and tests for linear trend were conducted across median values of categorical PA. For continuous PA, relative risks per 30-min/day increment were estimated. Linearity of the association was confirmed using restricted cubic spline regression[21] (Supplemental Figure e1). Tests for interaction were conducted by adding the cross-product term for continuous PA by the subgroup of interest to the Cox regression models. The proportional hazards assumption was examined both graphically plotting survival by PA categories as well as modeling mortality as a function of the interaction between PA and follow-up. No appreciable violations were noted.

Population attributable risk (PAR) of all-cause mortality was estimated for the lowest tertile of total PA and for current smoking, presence of one or more comorbidities and presence of very low physical function (SPPB <5) to quantify the influence that controlling these mortality determinants could theoretically have in the source population of our cohort. PAR was computed as $P_c(1-1/RR_{adj})$, where $P_c$ is the prevalence of a risk factor among decedents and $RR_{adj}$ is the multivariable RR for mortality associated with the risk factor.[22] The potential impact of subclinical morbidity at baseline was examined by discarding the first 6 months of follow-up and by stratifying the PA-mortality association simultaneously on age and comorbidity at baseline. P-values are for two-sided hypotheses tested at alpha .05.

## RESULTS

The cohort at baseline (Table 1) had a mean age of 78.6 years, BMI of 28.1 kg/m$^2$ (29.6% had BMI ≥30), 1.6 comorbid conditions (21.2% had ≥3), and physical function score of 8.2 out of a possible 12. Half were white (49.4%) with the remaining 33.7% and 16.9% being black and Hispanic, respectively. Of the mean 334.1 min/d of total PA, 56.1%, 29.0%, and 14.9% was accounted for by low light, high light and MVPA, respectively. Correlations among the PA measures and with age are shown in Table e2. All baseline characteristics, except age at menopause, were significantly associated with total PA (Table 1) and with all-cause mortality (Table e3).

We documented 450 all-cause deaths (154 CVD, 87 Cancer) during a mean 3.1 year (range, 0.5–4.5) follow-up interval. Crude all-cause death rates (per 1,000 person-years) declined

across incremental tertiles of total PA (lowest vs highest tertile: 38.2 vs 9.4), low light PA (30.8 vs 14.8), high light PA (38.7 vs 10.4) and MVPA (41.9 vs 7.6) (Table 2). Inverse patterns also were observed for CVD and cancer mortality rates. Age and race-ethnic specific all-cause death rates according to total PA are shown in the Figure e2. Except for ages 60–69 where death rates were low irrespective of PA, inverse associations with total PA are present for awake wear-time adjusted deaths rates in women 70–79, 80–89, and ≥90 years. Age and awake wear-time adjusted rates declined across PA tertiles in white and black women, whereas in Hispanic women the numbers of deaths were small and the pattern was less clear. After controlling for awake wear-time, age and the other covariables, strong inverse associations with all-cause mortality were observed for total PA (RR = 1.00, 0.68, 0.49, trend $P<.001$), high light PA (RR = 1.00, 0.57, 0.47, trend $P<.001$), and MVPA (RR = 1.00, 0.63, 0.42, trend $P<.001$) (Table 2). Low light PA was inversely, though non-significantly, associated with mortality (1.00, 0.86, 0.80, trend $P=.07$). Further adjustment for physical function score attenuated these associations, which remained significant for total, high light and MVPA (trend, $P<.001$, each). Results were similar for CVD mortality. For cancer mortality, there was a significant age- and wear-time adjusted inverse association with MVPA (trend, $P=.017$). After multivariable adjustment, while there was suggestion of inverse associations for each PA exposure with cancer mortality, none were statistically significant.

Table 3 shows multivariable relative risks for all-cause mortality associated with a 30-min/d increment in total, light (low and high combined), and MVPA. Each 30-min/d increment in total, light and MVPA was associated, on average, with statistically significant 12%, 12% and 39% lower mortality risks, respectively. When further simultaneously adjusting light and MVPA, significant inverse associations remained (light: RR=0.93; MVPA: RR=0.67). Associations between each PA exposure and mortality generally were consistent across subgroup categories and interaction tests non-significant, with one exception. Associations between MVPA and mortality were stronger in women with lower (SPPB ≤8, RR = 0.56) compared to higher physical function (SPPB >8, RR = 0.77; interaction $P=.009$).

To further understand the influence of PA *intensity* on mortality risk, we examined light and MVPA simultaneously in a multivariable model estimating the relative risks associated for each intensity at the same amount of energy expenditure (5 metabolic equivalent [MET]-hr/week). Energy expenditure was calculated by multiplying the accelerometer measured time (hr/week) by the median measured MET value (1 MET = 3.0 ml $O_2$ uptake/kg/min) observed in our calibration study[17] for light (2.0 METs) and MVPA (3.8 METs). This approach allows determination of whether mortality risks differ by PA intensity when holding constant the amount of PA. For each 5 MET-hr/wk in light PA (equivalent to about 30 min/d of slow walking), the multivariable RR of mortality was 0.69 (95% CI: 0.56, 0.87; β coefficient, −0.359) and for MVPA (equivalent to about 15 min/d of brisk walking) was 0.35 (95% CI: 0.24, 0.52; β coefficient, −1.042). The difference between regression coefficients was significant ($\chi^2_{df\ 1} = 7.0$, $P = .008$) indicating that while both light and MVPA were inversely associated with all-cause mortality, the relative risk reduction associated with MVPA was significantly greater than that for the same amount of light PA.

For public health context, PAR was estimated to quantify the theoretical proportion of deaths in the source population that might be averted, assuming a causal association, if women improved their PA levels beyond that of the lowest tertile (Table 4). PAR also was estimated for current smoking, ≥1 prevalent comorbidity, and very low physical function (SPPB <5). Overall, the PAR was 29.8% for prevalent comorbidity, 23.5% for low total PA, 9.0% for low physical function, and 1.3% for current smoking. In women ≥80 years, corresponding values were 21.2%, 23.3%, 8.7%, and 1.0%, respectively.

Sensitivity analyses showed that after discarding deaths during the first 6 months of follow-up, multivariable associations for total, light, and MVPA with each mortality endpoint were similar to those in the primary analysis (Table e4). To further evaluate the influence of prevalent disease at baseline, we simultaneously stratified these associations on age and number of comorbidities (Table e5). Inverse associations were observed for each PA exposure in women ages <80 and those ≥80 regardless of number of comorbidities, except for light PA in women <80 with ≥3 comorbidities for which sample size was small.

## DISCUSSION

The novel aspect of this large prospective study was use of accelerometers to measure usual daily PA in community-dwelling older women. The results support the hypothesis that higher levels of accelerometer measured PA, even when below the moderate intensity threshold required in current recommendations,[3,4] are associated with reduced all-cause and CVD mortality in women 63–99 years of age. Our findings expand on previous studies showing higher self-reported PA reduces mortality in adults 60 and older,[6,23] specifically in older women,[23] and at less than recommended amounts.[6,23,24] Moreover, our findings challenge the conclusion of recent meta-analyses that MVPA, measured by self-report, is required to offset mortality risk in adults.[6,7] Two principal observations underscore the present results.

First, absolute rates of all-cause and CVD mortality were at least 50% lower when comparing cohort members in the middle to those in the lowest tertile of each PA exposure. This is particularly impressive when considering the relatively small mean differences of 50, 33, and 20 min/d between these tertiles for low light, high light, and MVPA, respectively. Use of accelerometers enhanced accurate quantification of such small differences in usual daily PA, which are not possible by questionnaire assessments. Relatively small improvements in daily PA time, which can be achieved by older adults,[25] could potentially have substantial impact on mortality in later life. Even in the oldest cohort members, ages 80–89 and ≥90 years, absolute rates of all-cause mortality were 44% and 15% lower, respectively, when comparing the middle and lowest total PA tertile. Furthermore, absolute rates of all-cause mortality were 40% lower both in black and white women in the middle compared to lowest total PA group. After controlling for relevant mortality predictors, relative risk reductions in all-cause mortality associated with 30 min/d in total, light and MVPA were evident in women ≥80 years, and in both white and black women. Taken together, the apparent benefit of relatively small amounts of daily PA at less than guideline recommended moderate intensity may reach a broad age and race-ethnic distribution of the aging U.S. population.

Second, we used age-specific accelerometer intensity cutpoints to define light and MVPA. Our accelerometer measures reinforce previous self-reported data[24] in showing older women spend the majority of daily PA time in light PA. Previous findings are inconsistent regarding self-reported light PA and mortality, some showing no association[26] others showing protection.[24] Results of the present study provide clear evidence of reduced risks of all-cause and CVD mortality associated with both light intensity and MVPA measured by accelerometers. Low light PA was inversely associated with all-cause and CVD mortality and, impressively, is defined by accelerometer count levels (19–225 counts/15 sec) just slightly higher than those defining sedentary behavior (<19).[17] Even in women ≥80 years with ≥3 comorbidities at baseline, 30-min/d of light (low and high combined) and MVPA was associated with multivariable risk reductions in all-cause mortality of 10% and 43%, respectively. Because presence of multimorbidity affects more than 80% of adults 85 and older, and is associated with substantial disease burden,[27,28] this is a remarkable observation that suggests relatively small amounts of light PA could confer meaningful mortality benefit in a high risk subset of older women. For those able and interested in doing MVPA, results indicate benefit could be greater.

Accelerometer studies in NHANES have produced mixed results regarding light PA and mortality.[12,29,30] In these investigations, hip worn uniaxial accelerometers were used and light and MVPA were defined as 100-2019 and ≥2020 vertical axis counts/minute, respectively. MVPA was significantly associated with lower multivariable mortality risks when controlling for light PA.[12,29,30] However, only two of these studies observed significantly lower mortality associated with light PA when controlling for MVPA.[12,30] In this regard, Schmid et al.[12] observed a 13% lower mortality risk associated with 30-min/d in light PA in adults ≥65 years. This is consistent with the 8% lower risk for the same duration of light PA, we observed in women ages 63–99 years.

Low total PA in later life has relevance to all-cause mortality reduction similar to that of multimorbidity as suggested by the PARs of about 23% and 29%, respectively, in the older women studied here. If women became active beyond those in the lowest tertile or if presence of multimorbidity were eliminated, all-cause deaths would decrease by about 1 in 4, assuming causal associations. PAR, while a theoretical estimate, does bring into context the force an exposure exerts on population health, which depends both on the amount of exposure and its strength of association with mortality.[31] PAR would be low for characteristics that are not common – e.g., smoking in the present cohort. Because the population prevalence of low PA and its associated mortality risk is high,[32] the potential population impact for delaying mortality in later life through increases in PA is considerable.

The major strength and novelty of our study is accelerometer PA measurement, in a multiethnic cohort of women including a wide range of older ages, functional and general health status. The high proportion (89%) of WHI Long Life Study women that participated in OPACH, high number of accelerometers returned (95%) and high proportion (92%) of women meeting the wear time criteria reduce the likelihood that study results are influenced by self-selection. Use of triaxial accelerometers allowed detection of movement in three planes, whereas studies using uniaxial devices detect movement in a single plane (as in NHANES) which could be less sensitive to movement patterns and intensities of daily life[33]

and older adults.[17] Accelerometer intensity thresholds were determined in a sex- and age-relevant calibration study,[17] which is novel in epidemiologic investigations.[34]

Subclinical disease at baseline could have influenced study findings. The likelihood this is the primary explanation for our results is reduced by the consistency of results after discarding deaths during early follow-up, and when stratified on age (<80 vs ≥80 years), physical functioning, and multimorbidity. PA intensity was defined on an absolute scale. Because of the age-related decline in aerobic capacity,[35] even lighter intensity PA defined on an absolute scale may reflect a higher relative intensity for an older compared to younger adult. Subgroup analyses, particularly in Hispanic women, may have had limited statistical power, which should be considered when interpreting those results. While not extending directly to men, the present results are consistent with those published.[12]

In conclusion, when measured using accelerometers, higher levels of light PA, below the guideline recommended MVPA threshold,[3,4] are associated with reduced all-cause and CVD mortality in a multiethnic cohort of women ages 63–99 years. Mortality benefit of both light and MVPA appears to extend to all subgroups examined – obese, ages ≥80 years, multimorbidity, and low physical function. With continued growth in numbers of older women,[36] the high prevalence of inactivity in this group,[9] and the relatively large amount of daily time in light intensity activities,[24] these findings support greater emphasis on the benefits of light PA in future PA recommendations for our aging population. Further investigation of the health benefits of increasing light PA by reducing time spent sedentary is needed.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Impact Statement

We certify that this work is novel in demonstrating that accelerometer measured usual light intensity physical activity, at less than guideline recommended levels of moderate-to-vigorous physical activity, is associated with statistically significant reductions in all-cause and CVD mortality in a cohort of over 6,000 community-dwelling women ages 63–99 who were followed up for a median of 3.3 years in the Objective Physical Activity and Cardiovascular Health (OPACH) study. The mortality benefit of light intensity physical activity persisted following control for relevant mortality predictors including objectively measured physical functioning (SPPB score) and accelerometer measured moderate-to-vigorous physical activity levels. Furthermore, inverse associations between light intensity physical activity and mortality were consistent in cohort subgroups defined by age, race-ethnicity, BMI, and physical functioning. A novelty of this study is the use of sex- and age-relevant accelerometer cutpoints to define physical activity intensity that were determined in a laboratory calibration study in women of similar age as the OPACH cohort. To our knowledge, this is the first large prospective cohort study to show specifically in older women a protective association between accelerometer measured light intensity physical activity and mortality. Because older adults spend the majority of daily physical activity time in light intensity tasks, and given the expected growth in women ages 80 and older within the U.S. population in coming decades, these results are transformative in suggesting future guidelines should recommend light intensity physical activity, additional to more strenuous activities, for public health in an aging society.

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**Table 1**

Baseline characteristics among all women and according to tertile of total physical activity (N=6,382).

| Characteristic | All | Total Physical Activity (PA) Tertiles | | | P-trend |
|---|---|---|---|---|---|
| | | 1 (low) | 2 | 3 (high) | |
| N | 6,382 | 2,127 | 2,128 | 2,127 | |
| Total PA (min/d) | 334.1 ± 98.9 | 227.6 ± 46.9 | 331.1 ± 24.2 | 443.4 ± 57.2 | <.001 |
| Low light PA (min/d) | 187.5 ± 51.1 | 139.6 ± 31.3 | 189.9 ± 21.1 | 233.1 ± 40.9 | <.001 |
| High light PA (min/d) | 96.9 ± 36.1 | 62.2 ± 20.2 | 95.6 ± 16.9 | 132.8 ± 26.9 | <.001 |
| MVPA (min/d) | 49.7 ± 34.4 | 25.8 ± 16.9 | 45.6 ± 23.6 | 77.6 ± 36.5 | <.001 |
| Follow-up (years) | 3.1 ± 0.7 | 3.0 ± 0.8 | 3.2 ± 0.7 | 3.2 ± 0.7 | .01 |
| **Age** (years) | 78.6 ± 6.7 | 80.7 ± 6.5 | 78.6 ± 6.6 | 76.8 ± 6.4 | <.001 |
| 60–69 | 10.3 | 5.7 | 9.8 | 15.2 | <.001 |
| 70–79 | 40.1 | 32.2 | 41.1 | 46.9 | |
| 80–89 | 45.5 | 55.1 | 45.2 | 36.3 | |
| ≥90 | 4.1 | 7.0 | 3.9 | 1.6 | |
| **Accelerometer wear time** (hr/d) | 14.8 ± 1.4 | 14.2 ± 1.4 | 14.8 ± 1.2 | 15.4 ± 1.2 | <.001 |
| **Age at menopause** (years) | 48.2 ± 6.3 | 48.0 ± 6.5 | 48.2 ± 6.3 | 48.2 ± 6.3 | .51 |
| **BMI** ($kg/m^2$) | 28.1 ± 5.6 | 29.7 ± 6.0 | 28.1 ± 5.3 | 26.4 ± 4.9 | <.001 |
| <18.5 (underweight) | 1.4 | 0.8 | 1.2 | 2.0 | <.001 |
| 18.5–24.9 (normal) | 30.9 | 20.9 | 28.7 | 42.7 | |
| 25.0–29.9 (overweight) | 36.1 | 34.3 | 38.9 | 35.0 | |
| ≥30 (obese) | 29.6 | 40.6 | 29.0 | 19.2 | |
| **Race-ethnicity** | | | | | <.001 |
| White | 49.4 | 58.2 | 47.7 | 42.2 | |
| Black | 33.7 | 30.7 | 35.8 | 34.6 | |

| Characteristic | All | Total Physical Activity (PA) Tertiles | | | P-trend |
|---|---|---|---|---|---|
| | | 1 (low) | 2 | 3 (high) | |
| Hispanic | 16.9 | 11.1 | 16.5 | 23.3 | |
| **Education** | | | | | <.001 |
| High school or less | 20.2 | 20.3 | 19.9 | 20.4 | |
| Some college | 39.2 | 42.5 | 39.1 | 35.9 | |
| College grad or more | 40.7 | 37.2 | 41.0 | 43.7 | |
| **Current smoker** | 2.6 | 3.4 | 2.4 | 1.8 | .001 |
| **Alcohol**, drinks past 3 months | | | | | <.001 |
| None | 34.3 | 38.8 | 33.4 | 30.5 | |
| <1 per week | 40.1 | 41.6 | 41.2 | 37.6 | |
| ≥1 per week | 25.6 | 19.6 | 25.4 | 31.9 | |
| **Self-rated general health** | | | | | <.001 |
| Excellent | 8.9 | 5.1 | 9.1 | 12.7 | |
| Very good | 37.2 | 30.2 | 37.7 | 43.5 | |
| Good | 45.2 | 51.6 | 45.6 | 38.4 | |
| Fair | 8.3 | 12.2 | 7.3 | 5.3 | |
| Poor | 0.4 | 0.9 | 0.2 | 0.1 | |
| **SPPB score (n=5479)** | 8.2 ± 2.5 | 7.2 ± 2.7 | 8.4 ± 2.3 | 8.9 ± 2.2 | <.001 |
| 0–4 | 9.0 | 17.2 | 6.3 | 4.0 | <.001 |
| 5–8 | 41.9 | 48.5 | 43.7 | 34.1 | |
| 9–12 | 49.0 | 34.4 | 50.1 | 61.9 | |
| **Number of comorbidities** | 1.6 ± 1.3 | 1.9 ± 1.4 | 1.6 ± 1.2 | 1.4 ± 1.1 | <.001 |
| 0 | 16.9 | 11.8 | 16.3 | 22.6 | <.001 |
| 1–2 | 58.9 | 55.9 | 59.9 | 60.8 | |
| ≥3 | 21.2 | 29.1 | 20.5 | 13.9 | |

Data are mean ±SD, or %.

Number of comorbidities, range 0–10, include presence or absence of the following: coronary heart disease, stroke, cancer, diabetes, hip fracture, osteoarthritis, depression, COPD, cognitive impairment, frequent falls.

SPPB score, short physical performance battery test of physical functioning (described in reference #16), range 0–12 (missing n=903).

## Table 2

Risk of all-cause, CVD, and cancer mortality by physical activity levels (N = 6,382).

| | Physical Activity Tertile | | | P-Trend |
|---|---|---|---|---|
| | 1 (low) | 2 | 3 (high) | |
| **All-cause deaths** | | | | |
| Total PA, deaths (rate[a]) | 259 (38.2) | 124 (17.8) | 67 (9.4) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.58 (0.47, 0.73) | 0.38 (0.28, 0.51) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.68 (0.54, 0.85) | 0.49 (0.37, 0.66) | <.001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.73 (0.57, 0.93)* | *0.56 (0.41, 0.76)* | *<.001* |
| Low light PA, deaths (rate[a]) | 211 (30.8) | 135 (19.3) | 104 (14.8) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.77 (0.61, 0.95) | 0.69 (0.54, 0.89) | .002 |
| RR (95% CI)[c] | 1.00 (ref) | 0.86 (0.69, 1.08) | 0.80 (0.62, 1.03) | .074 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.91 (0.71, 1.15)* | *0.87 (0.66, 1.14)* | *.28* |
| High light PA, deaths (rate[a]) | 263 (38.7) | 113 (16.2) | 74 (10.4) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.49 (0.39, 0.62) | 0.36 (0.28, 0.48) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.57 (0.45, 0.71) | 0.47 (0.35, 0.61) | <.001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.61 (0.47, 0.78)* | *0.57 (0.42, 0.76)* | *<.001* |
| MVPA, deaths (rate[a]) | 280 (41.9) | 116 (16.6) | 54 (7.6) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.54 (0.43, 0.67) | 0.31 (0.23, 0.42) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.63 (0.50, 0.79) | 0.42 (0.30, 0.57) | <.001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.66 (0.51, 0.84)* | *0.46 (0.33, 0.65)* | *<.001* |
| **CVD deaths** | | | | |
| Total PA, deaths (rate[a]) | 97 (14.3) | 43 (6.2) | 14 (1.9) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.56 (0.39, 0.81) | 0.23 (0.13, 0.41) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.64 (0.44, 0.94) | 0.29 (0.16, 0.53) | <.001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.66 (0.44, 1.01)* | *0.34 (0.18, 0.64)* | *<.001* |
| Low light PA, deaths (rate[a]) | 82 (11.9) | 41 (5.9) | 31 (4.4) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.61 (0.42, 0.89) | 0.55 (0.35, 0.85) | .003 |
| RR (95% CI)[c] | 1.00 (ref) | 0.69 (0.47, 1.02) | 0.64 (0.41, 0.99) | .029 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.78 (0.51, 1.18)* | *0.72 (0.44, 1.18)* | *.16* |
| High light PA, deaths (rate[a]) | 100 (14.7) | 37 (5.3) | 17 (2.4) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.44 (0.30, 0.65) | 0.24 (0.14, 0.39) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.50 (0.34, 0.74) | 0.30 (0.17, 0.51) | <.001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.49 (0.32, 0.76)* | *0.33 (0.18, 0.59)* | *<.001* |

| | Physical Activity Tertile | | | P-Trend |
|---|---|---|---|---|
| | 1 (low) | 2 | 3 (high) | |
| MVPA, deaths (rate[a]) | 99 (14.8) | 39 (5.6) | 16 (2.2) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.57 (0.39, 0.53) | 0.31 (0.18, 0.53) | <.001 |
| RR (95% CI)[c] | 1.00 (ref) | 0.68 (0.45, 0.99) | 0.42 (0.24, 0.75) | .001 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.68 (0.44, 1.05)* | *0.43 (0.23, 0.81)* | *.005* |
| **Total cancer deaths** | | | | |
| Total PA, deaths (rate[a]) | 42 (6.2) | 23 (3.3) | 22 (3.1) | .135 |
| RR (95% CI)[b] | 1.00 (ref) | 0.62 (0.37, 1.05) | 0.68 (0.39, 1.19) | .476 |
| RR (95% CI)[c] | 1.00 (ref) | 0.71 (0.42, 1.19) | 0.85 (0.47, 1.51) | .34 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | 0.68 (0.38, 1.20) | 0.76 (0.41, 1.43) | |
| Low light PA, deaths (rate[a]) | 34 (4.9) | 32 (4.6) | 21 (2.9) | |
| RR (95% CI)[b] | 1.00 (ref) | 1.05 (0.64, 1.72) | 0.76 (0.43, 1.35) | .382 |
| RR (95% CI)[c] | 1.00 (ref) | 1.13 (0.69, 1.87) | 0.82 (0.46, 1.48) | .571 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.98 (0.57, 1.69)* | *0.79 (0.42, 1.46)* | *.47* |
| High light PA, deaths (rate[a]) | 38 (5.6) | 27 (3.9) | 22 (3.1) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.79 (0.48, 1.29) | 0.69 (0.39, 1.21) | .182 |
| RR (95% CI)[c] | 1.00 (ref) | 0.90 (0.54, 1.49) | 0.87 (0.49, 1.54) | .613 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | *0.76 (0.43, 1.34)* | *0.87 (0.47, 1.59)* | *.59* |
| MVPA, deaths (rate[a]) | 43 (6.4) | 30 (4.3) | 14 (1.9) | |
| RR (95% CI)[b] | 1.00 (ref) | 0.83 (0.51, 1.34) | 0.45 (0.23, 0.85) | .017 |
| RR (95% CI)[c] | 1.00 (ref) | 0.95 (0.58, 1.56) | 0.59 (0.30, 1.15) | .155 |
| *RR (95% CI)[d] (N=5479)* | *1.00 (ref)* | 0.92 (0.54, 1.58) | 0.59 (0.29, 1.21) | .18 |

[a] Crude death rate per 1,000 person years.

[b] Adjusted for awake accelerometer wear-time (hr/d) and age (years).

[c] Adjusted for awake accelerometer wear-time (hr/d), age (years), race-ethnicity (white, black, Hispanic), education (high school or less, some college, college or more), current smoking (no, yes), alcohol intake past 3 months (none, <1, ≥1 drinks/week), age at menopause (years), self-rated general health (excellent, very good, good, fair, poor), and number of comorbid conditions (0–10, continuous).

[d] Adjusted for above covariables plus SPPB score (0–12, continuous); N=5479 (384 total deaths, 128 CVD deaths, 75 cancer deaths).

## Table 3

Risk of all-cause mortality associated with a 30-minute/day increment in physical activity in the overall cohort and in cohort subgroups.

| Cohort group | Total PA RR (95% CI)[a] | Light PA RR (95% CI) [a] | MVPA RR (95% CI) [a] |
|---|---|---|---|
| **Overall** (N=6,382; 450 deaths) | 0.88 (0.85, 0.92) | 0.88 (0.85, 0.92) [b] 0.93 (0.89, 0.97) | 0.61 (0.54, 0.71) [b] 0.67 (0.58, 0.78) |
| **Age, yr** | | | |
| **<80** (N=3,211; 90 deaths) | 0.91 (0.85, 0.98) | 0.93 (0.84, 1.01) [b] 0.98 (0.89, 1.08) | 0.68 (0.52, 0.88) [b] 0.69 (0.53, 0.92) |
| **≥80** (N=3,171; 360 deaths) | 0.87 (0.84, 0.91) | 0.88 (0.84, 0.92) [b] 0.92 (0.87, 0.96) | 0.60 (0.51, 0.71) [b] 0.67 (0.56, 0.79) |
| ‡Interaction, p-value | .67 | .74 | .73 |
| **Race-ethnicity** | | | |
| **White** (N=3,150; 320 deaths) | 0.89 (0.85, 0.93) | 0.89 (0.86, 0.94) [b] 0.94 (0.89, 0.99) | 0.60 (0.51, 0.71) [b] 0.64 (0.54, 0.76) |
| **Black** (N=2,151; 96 deaths) | 0.85 (0.79, 0.92) | 0.85 (0.77, 0.92) [b] 0.90 (0.82, 0.99) | 0.53 (0.38, 0.74) [b] 0.63 (0.45, 0.91) |
| **Hispanic** (N=1,081; 34 deaths) | 0.91 (0.81, 1.03) | 0.89 (0.77, 1.04) [b] 0.90 (0.77, 1.05) | 0.88 (0.62, 1.24) [b] 0.96 (0.67, 1.37) |
| ‡Interaction, p-value | .32 | .14 | .38 |
| **BMI, kg/m$^2$** | | | |
| **<30** (N=4,492; 336 deaths) | 0.87 (0.83, 0.91) | 0.87 (0.83, 0.92) [b] 0.91 (0.87, 0.96) | 0.62 (0.54, 0.73) [b] 0.69 (0.59, 0.81) |
| **≥30** (N=1,890; 114 deaths) | 0.88 (0.81, 0.95) | 0.89 (0.81, 0.97) [b] 0.94 (0.86, 1.03) | 0.53 (0.38, 0.75) [b] 0.58 (0.41, 0.82) |
| ‡Interaction, p-value | .86 | .64 | .29 |
| **SPPB score** (*N=5479, 384 deaths)* | | | |
| **≤8** (N=2,792; 279 deaths) | 0.88 (0.84, 0.92) | 0.89 (0.84, 0.94) [b] 0.94 (0.89, 0.99) | 0.56 (0.46, 0.69) [b] 0.61 (0.49, 0.75) |
| **>8** (N=2,687; 105 deaths) | 0.92 (0.85, 0.98) | 0.92 (0.85, 1.01) [b] 0.95 (0.87, 1.03) | 0.77 (0.61, 0.96) [b] 0.79 (0.63, 1.00) |
| ‡Interaction, p-value | .10 | .16 | .009 |

RR, relative risk; CI, confidence interval; BMI, body mass index; SPPB, short physical performance battery.

Light PA is the combined minutes/day in low light PA and high light PA.

[a] Adjusted for awake accelerometer wear-time, age, race-ethnicity, education, smoking, alcohol, age at menopause, number of comorbid conditions, and self-assessed general health status as defined in Table 2 footnote.

[b] Light PA and MVPA are mutually adjusted for one another.

## Table 4

Population attributable risks for all-cause mortality by selected cohort characteristics.

| | Prevalence in cohort (%) | Prevalence in decedents (%) | RR[a] (95% CI) [a] | PAR (%)[b] (95% CI) |
|---|---|---|---|---|
| **All women** (N=6,382; 450 deaths) | | | | |
| Low total PA | 33.3 | 57.6 | 1.69 (1.38, 2.01) | 23.5 (14.3, 31.8) |
| Current smoker | 2.6 | 3.1 | 1.72 (1.01, 2.95) | 1.3 (−0.4, 2.9) |
| ≥1 comorbidity | 83.1 | 92.0 | 1.48 (1.05, 2.09) | 29.8 (3.4, 49.0) |
| Low physical function (SPPB <5) | 9.0 | 23.4 | 1.62 (1.26, 2.09) | 9.0 (3.6, 13.9) |
| **Women ≥80 years** (N=3,171; 360 deaths) | | | | |
| Low total PA | 41.7 | 59.7 | 1.64 (1.31, 2.06) | 23.3 (12.4, 32.9) |
| Current smoker | 1.5 | 2.2 | 1.80 (0.89, 3.65) | 1.0 (−0.6, 2.5) |
| ≥1 comorbidity | 86.9 | 91.7 | 1.30 (0.89, 1.91) | 21.2 (−11.8, 44.4) |
| Low physical function (SPPB <5) | 14.4 | 25.1 | 1.53 (1.17, 2.00) | 8.7 (2.4, 14.6) |

[a]Adjusted for awake accelerometer wear-time, age, race-ethnicity, education, alcohol, age at menopause, self-rated general health and the other characteristics in the table.

[b]Calculated using the following formula[22]: $P_C(1-1/RR_{adj})$, where $P_C$ is the prevalence of a risk factor among decedents and $RR_{adj}$ is the multivariable adjusted[a] relative risk for mortality associated with the specified risk factor.