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Walking Volume and Speed are Inversely Associated with Incidence of Treated Hypertension in Postmenopausal Women

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Abstract

Few studies have evaluated hypertension incidence in relation to walking, which is a common physical activity among adults. We examined the association between walking and hypertension incidence in 83,435 postmenopausal women who at baseline were aged 50–79 years, without known hypertension, heart failure, coronary heart disease, or stroke, and reported the ability to walk at least one block without assistance. Walking volume (metabolic equivalent hours per week; MET-hr/wk) and speed (miles per hour; mph) were assessed by questionnaire. Incident physician-diagnosed hypertension treated with medication was ascertained through annual questionnaires. During a mean 11-year follow-up, 38,230 hypertension cases were identified. After adjustment for covariates including non-walking activities, a significant inverse association with hypertension was observed across categories of baseline walking volume (0 [referent], >0–3.5, 3.6–7.5, >7.5 MET-hr/wk), hazard ratio [HR]: 1.00 [ref], 0.98, 0.95, 0.89, trend $p < 0.001$. Faster walking speeds (<2, 2–3, 3–4, >4 mph) also were associated with lower hypertension risk, HR: 1.00 [ref], 1.07, 0.95, 0.86, 0.79, trend $p < 0.001$. Further adjustment for walking duration (hr/wk) had little impact on the association for walking speed (HR: 1.00 [ref], 1.08, 0.96, 0.86, 0.77, trend $p < 0.001$). Significant inverse associations for walking volume and speed persisted after additional control for baseline blood pressure. Results for time-varying walking were comparable to those for baseline exposures. This study showed that walking at guideline recommended volumes (>7.5 MET-hr/wk) and at faster speeds (>2 mph), is associated with lower hypertension risk in postmenopausal women. Walking should be encouraged as part of hypertension prevention in older adults.

Keywords

Exercise; Epidemiology; Hypertension; Women; Longitudinal; Prevention

INTRODUCTION

Age-related blood pressure (BP) dysregulation and increases in hypertension prevalence are attributed to changes in arterial structure and function occurring with aging.^{1,2} It is estimated that by 2030 there will be 73 million U.S. citizens aged 65 years and older, 55% of whom will be postmenopausal women.³ Compared with their younger premenopausal counterparts, postmenopausal women experience a greater rise in mean BP with increasing age, and a higher prevalence of hypertension.^{4–6} Given the significant health consequences associated

with hypertension,^{7,8} identification of modifiable factors by which older women might lessen their risk of hypertension has important public health implications.

Physical activity (PA) has been inversely associated with hypertension incidence in observational studies.⁹ Sex-specific results have been inconsistent, with a number of studies yielding non-significant findings in women.^{10–13} Available studies have not focused on women 60 and older, in whom hypertension prevalence is high. Evidence for an association between walking and hypertension incidence is sparse. Because walking is the most frequently reported activity among older adults,¹⁴ understanding its potential role in hypertension prevention has relevance to population aging.

The present study prospectively examined the association between walking and hypertension incidence in the Women's Health Initiative (WHI). Our hypothesis was that greater walking volume and speed would each be associated with lower hypertension risk. We further hypothesized that walking speed would be associated with hypertension risk independent of walking duration.

METHODS

Data that support the findings of this study are available from the corresponding author upon reasonable request and with permission from the WHI.

The WHI enrolled 161,808 postmenopausal women, aged 50–79 years, into one or more of three clinical trials (CT; Hormone Therapy [estrogen plus progestin, or estrogen alone]; Dietary Modification; Calcium and Vitamin D Supplementation) or a prospective observational study (OS) at 40 U.S. centers. The WHI was designed to examine determinants of morbidity and mortality in postmenopausal women.^{15–17}

The current analyses included CT and OS women who at baseline: (a) were without a history of hypertension treated with medication, and (b) whose baseline measured BP was systolic <140 mmHg and diastolic <90 mmHg. Although guidelines now define hypertension as systolic and diastolic BP ≥130 and ≥80 mmHg,¹⁸ we used a definition consistent with practice guidelines at the time of baseline measurements (1993–1998).¹⁹ Walking information was missing in 5,943 women; they were excluded. Characteristics of these women were similar to those included, except for slightly lower current menopausal hormone therapy use (38.5% vs 42.9%) and college education (77.2% vs 81.4%) (Table S1). Hypertension rates (per 1000 person-years) were 50.9 and 45.8 in women missing and not missing walking, respectively. Participants were further excluded if at baseline they reported not being able to walk at least one block unassisted, had a history of heart failure, coronary heart disease or stroke, or had <1 year of follow-up, resulting in 83,435 women (32,790 CT; 50,645 OS) for analysis (Figure S1). Institutional Review Board approval and participant informed consent was obtained at WHI centers. This study conformed to STROBE guidelines for human observational studies.

Walking and Physical Activity Assessment

Walking was assessed using self-administered questionnaires at baseline and again periodically during follow-up (CT: years 1, 3, 6, 9, annually thereafter; OS annually year 3 and after). Frequency, duration, and speed of usual walking for at least 10 minutes consecutively was queried (Supplement). Walking volume was computed as the product of walking frequency, duration, and the metabolic equivalent (MET) intensity value corresponding to walking speed response choices (<2 mph: 2 METs; 2–3 mph: 3 METs; 3–4 mph: 4 METs; >4 mph: 5 METs),²⁰ yielding walking MET-hr/wk. Non-walking recreational PA volume was self-reported and summarized as MET-hr/wk (Supplement). Walking and PA assessed by this questionnaire is reproducible (ICC = 0.51–0.77)²¹ and moderately correlated ($r = 0.45$ – 0.52) with criterion measures²² in similarly aged women.

Hypertension Ascertainment

Incidence of physician-diagnosed hypertension treated with medication was identified using postbaseline health questionnaires completed annually (OS) or biannually (CT). The case finding question read: “*Since the date given on the front of this form, has a doctor prescribed pills for high blood pressure or hypertension?*”. Strong agreement exists for WHI hypertension status based on this question and Medicare claims ($\kappa=0.84$; unpublished data). Response reproducibility is high ($\kappa=0.86$) for repeated assessments at enrollment.²³

Covariate Assessment

Information on sociodemographics, medical history, dietary intake, and smoking was obtained using self-administered questionnaires. Waist circumference, weight, and height were measured in clinic; body mass index (BMI; kg/m^2) was calculated.¹⁶ Seated BP was measured after five minutes of rest using auscultatory methods with cuff size based on measured arm circumference.²⁴ Physical function was assessed using the RAND-36 instrument.²⁵ Dietary sodium intake and the Healthy Eating Index (HEI)²⁶ was determined using the WHI food frequency questionnaire.²⁷ Neighborhood socioeconomic status (nSES; 0–100) was derived using variables on education, employment, household income, and socioeconomic indicators based on U.S. census tracts; higher scores reflect more affluence.

Statistical Analysis

Baseline characteristics were summarized according to walking categories using chi-square tests and analysis of variance. Kaplan-Meier plots characterized the unadjusted annualized incidence of hypertension over walking categories; logrank tests determined statistical significance. Cox proportional hazards regression was used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for incident hypertension according to walking exposures.

Person-time for each participant was from the date of baseline examination (OS) or randomization (CT) to date of the questionnaire on which hypertension diagnosis was reported, censoring on loss to follow-up or death, or August 29, 2014, whichever occurred first. Follow-up was stopped in 2014 because a PA intervention trial was initiated in WHI around this time,²⁸ and because revised hypertension guidelines were published in 2017.¹⁸ To avoid potential biases associated with participation in the PA intervention or with changes

in BP monitoring and treatment, we ended follow-up prior to these time points. All statistical models controlled for WHI component (CT or OS) and randomization to trial arms. Model 1 included age and race-ethnicity. Model 2 further controlled for education, alcohol, smoking, dietary sodium and HEI, menopausal hormone therapy, diabetes, hyperlipidemia, cancer, and non-walking PA. We then added covariates separately to Model 2: baseline systolic and diastolic BP (Model 3), BMI (Model 4; repeated using waist), and physical functioning (Model 5). In order to explore whether walking speed contributed to hypertension risk independently from walking duration, we estimated HRs according to walking speed categories controlling for Model 2 covariates and additionally for walking duration (hr/wk). Multicollinearity was assessed using variance inflation factors which were not large (<1.3 in multivariable Model 2) suggesting meaningful collinearity was unlikely. For all analyses, the referent group was women who reported no walking (0 MET-hr/wk, *non-walkers*). Walking volume was defined as tertiles ($>0-3.5$, $3.6-7.5$, >7.5 MET-hr/wk). Walking speed was defined according to questionnaire response choices (<2 , $2-3$, $3-4$, >4 mph). Ordinal scoring was used to evaluate linear trend across walking categories. We also conducted analyses using time-varying exposures for walking volume and speed based on postbaseline reassessments. For walking volume, rather than use different tertile cut-points at each assessment, we evaluated volume as a continuous parameter in the time-varying analysis. Walking speed was based on fixed category definitions at all assessments; the time-varying analysis proceeded accordingly. Covariates were as in Model 2. To evaluate consistency between subgroups, we examined associations across baseline categories of age (50–59, 60–69, 70 years), race-ethnicity (white, black, Hispanic, Asian/Pacific Islander; insufficient data for Native Americans), systolic and diastolic BP (tertiles), BMI (<18.0 , $18.0-24.9$, $25.0-29.9$, ≥ 30 kg/m²), and non-walking PA (0, $>0-9.3$, >9.3 MET-hr/wk). Statistical interactions were tested using cross-product terms controlling for Model 2 covariates. Sensitivity analyses using Model 2 covariates were conducted to evaluate robustness of our primary analyses. In separate models, we excluded (1) hypertension cases identified in follow-up years 1–3, (2) women with limited functional ability, (3) women reporting anti-hypertension use in the absence of known hypertension, (4) women randomized to hormone CT intervention arm because hypertension incidence was higher in these women,²⁹ and (5) women who reported any vigorous intensity non-walking PA. Statistical tests were conducted at two-sided alpha 0.05 using SAS (Windows v9.4; Cary, NC).

RESULTS

The mean age of participants was 61.9 years, the majority of whom were white and educated beyond high school (Table 1). Participants were from middle or higher socioeconomic strata (mean nSES, 76.7). Mean BMI, systolic and diastolic BP were 26.7 kg/m², 118.0 and 72.3 mmHg, respectively. Prevalence of current smoking (7.1%), hyperlipidemia (9.7%), diabetes (1.9%), and cancer (9.3%) were relatively low. Participants had high physical functioning (mean RAND-36 score, 87.2). All characteristics in Table 1 were significantly associated with walking volume, except smoking history and dietary sodium. Similar patterns were seen with walking speed (Table S2). All characteristics, except ADL score, differed significantly with hypertension incidence (Table S3).

During a mean 10.8 year follow-up, 38,230 (45.8%) hypertension cases were documented. The crude incidence rate was 42.4 cases per 1,000 person-years. The annualized cumulative incidence of hypertension was lower ($p<0.001$) across incremental categories of walking volume and speed; differences somewhat greater for walking speed (Figure S2). The hypertension incidence rate (per 1,000 person-years) in non-walkers was 45.8, whereas rates in women in the upper category of walking volume (>7.5 MET-hr/wk) and speed (>4 mph) were 38.0 and 30.1, respectively (Table 2).

Following adjustment for Model 2 covariates and when compared to non-walkers, relative hazards of hypertension were lower across incremental tertiles of walking volume (HR: 1.00 [ref], 0.98, 0.95, 0.89; trend $p<0.001$) (Table 2). An inverse association with hypertension risk was evident for faster walking speeds, although women at the slowest speed had elevated risk (Model 2 HR: 1.00 [ref], 1.07, 0.95, 0.86, 0.79; trend $p<0.001$); comparable when further adjusted for walking duration (HR: 1.00 [ref], 1.08, 0.96, 0.86, 0.77; trend $p<0.001$). Additional adjustment for baseline BP (Model 3), BMI (Model 4), and physical functioning (Model 5) attenuated the associations with walking volume and speed but the inverse trends remained significant. Adjusting for waist circumference instead of BMI did not alter results (not shown).

To further understand how walking volume and speed influence hypertension risk, we estimated associations between speed and hypertension within categories of walking volume (Figure 1). Compared to non-walkers, within each walking volume category there was an inverse gradient in hypertension risk over walking speeds ≥ 2 mph (Trend, $P<.001$). Both women whose volume was consistent with PA recommendations (>7.5 MET-hr/wk) and those whose volume was below this recommendation had significantly lower hypertension risks with increasing walking speeds ≥ 2 mph. We then repeated the analysis within categories of walking duration (hr/wk) and observed a similar pattern of results (Figure S3). Lower hypertension risks were evident at speeds ≥ 2 mph in both women whose walking duration achieved recommendations (≥ 2.5 hr/wk) and those who did not. The elevated hypertension risk associated with walking <2 mph was evident within categories of walking volume and duration.

Analyses using time-varying walking volume showed a 7% lower hypertension risk for each 7.5 MET-hr/wk increment (comparable to recommended PA levels; Model 2 HR=0.93, 95% CI: 0.92–0.94), which was similar to findings for baseline walking volume as a continuous variable (HR=0.95, 95% CI: 0.94–0.96). For time-varying walking speed, compared to non-walkers, HRs (95% CI; Model 2) were 1.03 (1.00–1.06), 0.94 (0.92–0.97), 0.83 (0.80–0.86), 0.63 (0.55–0.72) across walking speed categories <2 , 2–3, 3–4 >4 mph, respectively; comparable to baseline results (Table 2).

Table 3 displays stratified associations for baseline walking volume. To preserve statistical power, HRs are for a 7.5 MET-hr/wk increment. Results were consistent across strata of race-ethnicity, baseline BP and BMI (interaction $p>0.10$, all). The association was somewhat stronger in women aged 50–59 (interaction $p<0.001$) and women reporting no non-walking PA (interaction $p=0.03$). Stratified results were similar for baseline walking speed (Table S4). Robustness of the primary results were evaluated in five sensitivity analyses (specified

in Methods) yielding findings similar to the primary results for both walking exposures (Figure 2).

DISCUSSION

Regular PA is associated with lower hypertension risk.^{30,31} Few studies have focused on women or on walking, which is a prevalent activity in adults.¹⁴ After controlling for sociodemographic, lifestyle and clinical factors, we observed significantly lower hypertension risks of 11% and 21% (Table 2, Model 2) in postmenopausal women reporting the highest walking volume and speed. Walking speed remained significantly associated with lower hypertension risk after adjusting for walking duration (hr/wk). Within each category of walking volume (and of walking duration), an inverse association between hypertension risk and walking speed was evident at speeds ≥ 2 mph. Despite further controlling for baseline BP, BMI or waist, and physical functioning, significant inverse trends persisted for each walking exposure. Associations were not different across race-ethnicity, baseline BP, or BMI subgroups; however, were somewhat stronger in women aged 50–59 and those reporting no recreational PA. Results based on time-varying walking and extensive sensitivity analyses were similar to primary findings for both exposures. Associations for continuous walking volume (per 7.5 MET-hr/wk increment) were somewhat smaller than categorical analysis comparing women in the highest category (>7.5 MET-hr/wk) with non-walkers, however, the continuous model effect size reflects an average relative risk over the entire range of walking volume. Our results indicate that walking at casual speed (e.g., 30-minute mile) to meet or exceed guideline recommended volume (or duration) is associated with lower hypertension risk in later life. Because inactivity prevalence is high in older adults, even a modest relative risk (e.g., HR = 0.89, Table 2, Model 2) could have relevance in reducing the burden of hypertension's consequences, such as CHD and stroke.³²

Two previous studies on hypertension evaluated walking. Hayashi et al. followed-up 6,017 middle-aged men³³ and found a 29% lower hypertension risk associated with walking ≥ 21 compared to ≤ 10 minutes/day. Williams et al. studied 15,945 walkers (79% women, mean age 61) recruited from community walking events,³⁴ and observed a 7% lower hypertension risk for each 12.6 MET-hr/wk increment in walking volume. This is consistent with our finding of a 5% lower hypertension risk for each 7.5 MET-hr/wk increment in baseline walking volume.

We observed a stronger association with hypertension for walking speed than volume. Speeds ≥ 2 mph were inversely associated with hypertension risk within each category of walking volume (Figure 1) and duration (Figure S3). For women walking at recommended volumes of activity (>7.5 MET-hr/wk), risk reductions of 5%, 14%, and 22% were associated with speeds of 2–3, 3–4, and >4 mph, respectively. Importantly, even when walking volume or duration was *less than guideline recommendations* (>0 to 7.5 MET-hr/wk; >0 to 2.5 hr/wk), women walking at speeds ≥ 2 mph had significantly lower hypertension risks. This suggests faster walking speed might confer BP benefit beyond volume or duration of walking. Disentangling these interrelationships, however, is challenging. Speed reflects the intensity of walking activity. Intensity of PA is correlated

with health benefits,³⁵ including BP outcomes.³⁶ Greater intensity through faster walking might provoke more potent BP regulating mechanisms. Walking speed is positively correlated with maximal oxygen uptake.³⁷ Thus, walking speed could be a marker of cardiorespiratory fitness, which is inversely associated with hypertension incidence in women.³⁸ Higher walking speed requires better physical functioning, which is inversely associated with BP in WHI women.³⁹ Controlling for physical function attenuated the association between walking speed and hypertension (Table 2, Model 5).

Interestingly, women whose walking speed was <2 mph had a significantly higher 5–8% risk of hypertension compared to non-walkers (Table 2). CVD incidence also has been elevated at this walking speed in WHI.⁴⁰ Despite extensive adjustments and sensitivity analyses, residual confounding by comorbidity could account for higher risk at the slowest walking speed.

Several mechanisms could explain an association between walking and hypertension. Modulation of total peripheral resistance is important for BP regulation during PA.⁴¹ This is likely mediated by neurohumoral adaptations (decreased sympathetic nervous activity, lower circulating catecholamines and cortisol, improved baroreflex sensitivity, improved renal function, improved sodium-water regulation) and vascular alterations (improved endothelial function, reduced atherosclerotic burden, increased capillary recruitment capacity, angiogenesis) occurring with PA.^{41,42} Enhanced body composition and insulin sensitivity, and lower systemic inflammation also are plausible mechanisms.⁴² Many such physiologic adaptations to regular PA are documented in older adults.^{42,43}

Study strengths include focus on postmenopausal women and the large number of incident hypertension cases. The extensive health and lifestyle information permitted evaluation of several confounding and modifying factors, which enhances confidence in our results. Reverse causation bias is a concern when prospectively assessing associations between hypertension and walking, each could be influenced by pre-clinical cardiovascular manifestations. We cannot completely rule this out as an alternative explanation for the observed inverse associations. However, we excluded participants with evidence of baseline heart failure, CHD, stroke, and severe functional limitations. We observed consistency between our primary results and sensitivity analyses that additionally excluded participants with any functional limitations and hypertension cases occurring during early follow-up (an approach often used in epidemiologic studies).⁴⁴ Follow-up was ended before the 2017 BP guideline change to avoid potential surveillance bias.

Limitations included self-reported walking information. Because walking was assessed separately from other PA types with good reproducibility in WHI age and race-ethnic subgroups,²¹ less misclassification error may have occurred than when summarizing multiple PA domains. Consistency of results for time-varying and baseline exposures is reassuring. Ascertainment of hypertension cases was through self-reported information. WHI participants have reliably reported history of treated hypertension.²³ Information on family history of hypertension was insufficient for analysis. The WHI enrolled volunteers recruited across the U.S. and may not be a representative cross-section of postmenopausal women, a consideration when generalizing our results.

Perspectives

Results from this prospective study indicate walking is associated with lower hypertension incidence in postmenopausal women, independent of non-walking PA. Walking at a casual pace (e.g., 30-minute mile) to achieve the nationally recommended volume (7.5 MET-hr/wk) or duration (2.5 hr/wk) was associated with significantly lower hypertension risk. For women who did not meet recommendations on volume or duration, walking faster was associated with lower hypertension risk. Importantly, associations between walking and hypertension were consistent across categories of baseline BP and measures of adiposity, suggesting that BP benefit through walking is accessible even for women with elevated hypertension risk due to high normal BP or obesity. If our results are confirmed, it seems reasonable to contemplate design of a randomized clinical trial evaluating walking for the primary prevention of hypertension in adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

WHI Investigators: Supplemental File.

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Novelty and Significance

What is new?

- Walking (an understudied exposure) was prospectively evaluated with hypertension risk
- Postmenopausal women (an understudied population) were studied exclusively

What is relevant?

- Elevated BP is highly prevalent in older women
- Lifestyle modifications are cornerstone to hypertension prevention strategies
- Walking is an accessible and enjoyable activity in older adults

Summary

Greater walking volume and speed were associated with lower hypertension risk in postmenopausal women. This carries tremendous implications for enhancing hypertension prevention strategies through targeted promotion of walking, the most common recreational activity in later life when the burden of high BP and its clinical consequences is considerable.

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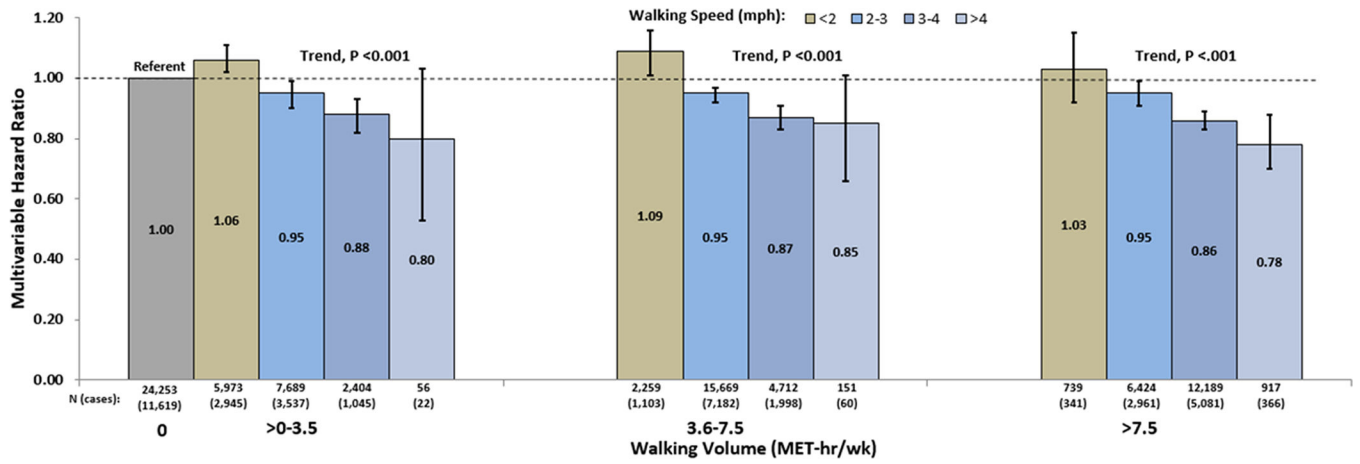


Figure 1. Association between walking speed and hypertension within categories of walking volume.

Covariates as in Model 2, Table 2. Bars are 95% CI. Walking 7.5 MET-hr/wk is the minimal volume required to meet guideline recommendations.³²

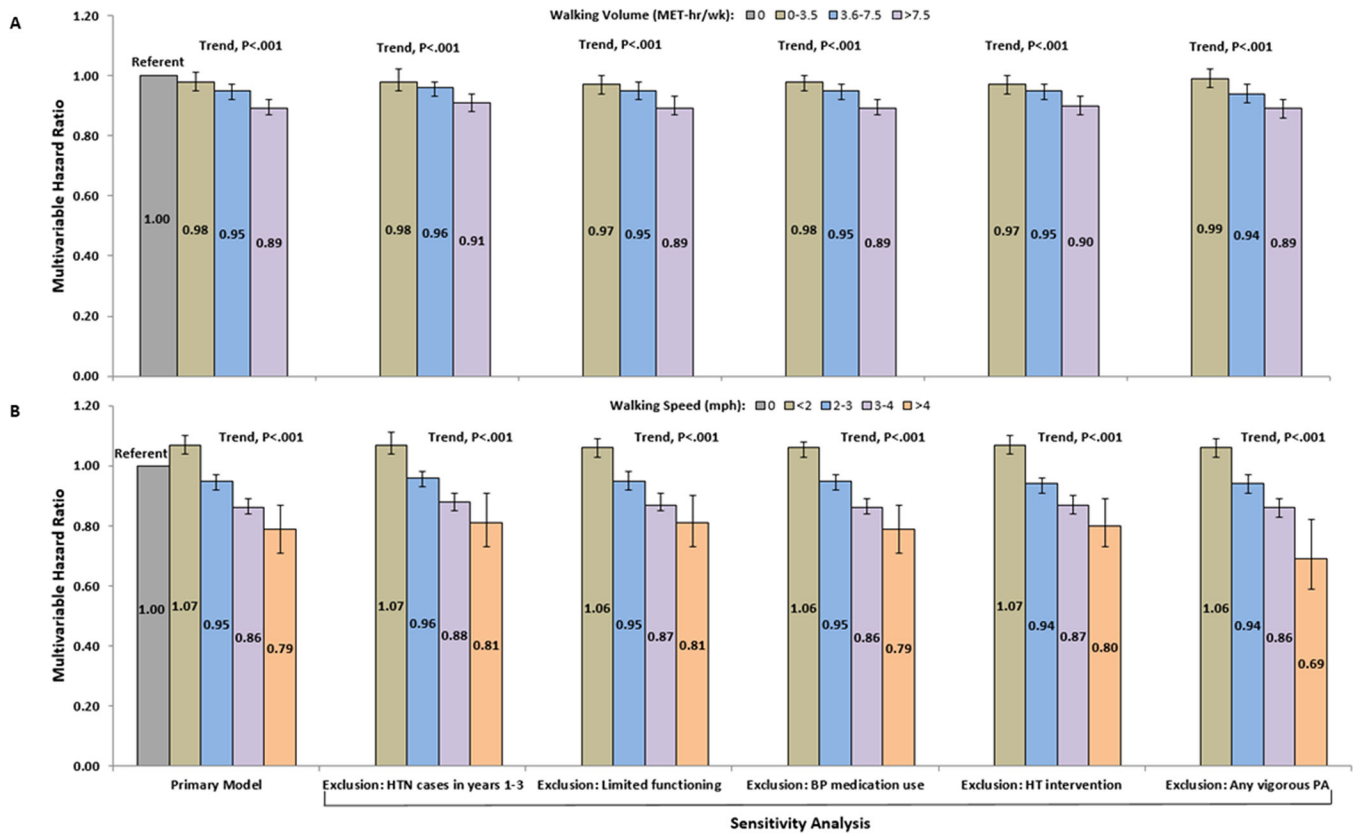


Figure 2. Sensitivity analyses for walking volume (A) and speed (B). Covariates as in Model 2, Table 2. Bars are 95% CI. Functional exclusion was for RAND-36 score <60 or ADL score >4. Medication exclusion was for blood pressure medication without history of diagnosed-treated hypertension.

Table 1.

Baseline characteristics for the overall cohort and according to categories of walking volume (N=83,435).

Characteristic	Overall (n=83,435)	Walking Volume (MET-hr/wk)			P trend	
		0 (n=24,253)	>0-3.5 (n=16,122)	3.6-7.5 (n=22,791)		>7.5 (n=20,269)
Walking volume (MET-hr/wk)	5.2 ± 6.3	0.0 ± 0.0	1.7 ± 0.8	5.2 ± 1.5	14.3 ± 5.9	<0.001
Non-walking PA (MET-hr/wk)	13.8 ± 14.3	6.4 ± 10.4	8.8 ± 9.9	14.1 ± 10.9	26.4 ± 16.2	<0.001
Age (years)	61.9 ± 7.03	61.4 ± 7.0	62.0 ± 7.1	62.4 ± 7.1	61.9 ± 7.0	<0.001
50-59	40.3	43.0	40.1	37.7	40.1	
60-69	43.6	42.3	43.3	44.3	44.4	
70-79	16.2	14.7	16.6	18.1	15.6	
Race-ethnicity						<0.001*
White	86.6	83.69	86.5	88.5	88.3	
African American	5.5	7.58	5.2	4.3	4.7	
Hispanic	4.0	4.63	4.2	3.6	3.3	
Native American	0.4	0.37	0.3	0.3	0.4	
Asian/Pacific Islander	2.5	2.67	2.6	2.4	2.3	
Education						<0.001 [†]
High school or less	18.6	22.6	19.0	16.8	15.6	
College/some college	48.6	49.1	48.7	48.4	48.0	
Post-graduate	32.8	28.3	32.3	34.8	36.3	
nSES score [‡]	76.7 ± 8.0	76.0 ± 8.2	76.4 ± 8.0	77.0 ± 7.8	77.4 ± 7.8	<0.001
Smoking						0.120 [§]
Never	50.6	50.2	51.5	51.0	49.9	
Former	42.3	40.2	40.5	43.0	45.4	
Current	7.1	9.6	8.0	6.0	4.8	
Alcohol use						<0.001 [§]
Never	9.1	10.3	9.6	8.6	8.1	
Former	15.6	17.6	15.5	14.8	14.4	
Current	75.2	72.2	74.9	76.6	77.6	

Characteristic	Walking Volume (MET-hr/wk)			P trend	
	Overall (n=83,435)	0 (n=24,253)	>0-3.5 (n=16,122) 3.6-7.5 (n=22,791) >7.5 (n=20,269)		
Hormone therapy use				<0.001 [§]	
Never	41.8	42.5	43.6	41.2	40.0
Former	15.3	15.4	15.1	15.8	14.8
Current	42.9	42.1	41.2	43.0	45.2
Diabetes	1.9	2.4	2.2	1.6	1.4
Hyperlipidemia	9.7	9.8	9.7	10.1	9.1
Cancer	9.3	8.8	9.3	9.7	9.5
ADL Score ^{//}	4.0 ± 0.2	4.0 ± 0.2	4.0 ± 0.3	4.0 ± 0.2	4.0 ± 0.2
Physical function score [#]	87.2 ± 13.5	84.2 ± 15.1	85.4 ± 14.5	88.1 ± 12.4	91.3 ± 10.2
Systolic BP (mmHg)	118.0 ± 11.5	118.6 ± 11.3	118.6 ± 11.4	118.1 ± 11.5	117.3 ± 11.7
Diastolic BP (mmHg)	72.3 ± 7.6	72.5 ± 7.7	72.5 ± 7.7	72.2 ± 7.6	72.0 ± 7.6
BMI (kg/m ²)	26.7 ± 5.2	27.7 ± 5.7	27.4 ± 5.5	26.3 ± 4.8	25.3 ± 4.4
Waist (cm)	83.2 ± 12.5	85.6 ± 13.3	84.8 ± 12.8	82.4 ± 11.7	79.9 ± 11.0
Healthy Eating Index diet score	67.9 ± 10.6	64.9 ± 11.1	67.1 ± 10.5	69.1 ± 10.0	70.6 ± 9.8
Dietary sodium intake (mg)	2697.1 ± 1206.0	2664.9 ± 1239.2	2736.6 ± 1198.0	2715.6 ± 1163.6	2683.2 ± 1217.8
Study component					
Clinical Trial	39.3	45.6	42.5	37.1	31.9
Observational Study	60.7	54.4	57.5	62.9	68.1
HT intervention	7.9	9.2	8.4	7.5	6.4

Data are mean ± standard deviation or percentage.

Abbreviations: PA, physical activity; nSES, neighborhood socioeconomic status; BMI, body mass index; HTN, hypertension; MET, metabolic equivalent; BP, blood pressure; HT, hormone therapy.

* p-value compares percentage of white vs. non-white participants.

[†] p-value compares percentage of high school vs. some college and/or college graduate.

[‡] Neighborhood socioeconomic status, range 0-100; higher scores indicate more affluent status.

[§] p value compares percentage of never vs. ever.

^{//} Activities of daily living score, range 4-12; higher values reflect greater limitations.

Physical function score, range 0–100; higher values reflect greater physical function.

** p-value compares active vs. placebo and/or not randomized.

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Table 2.

Rates and relative risks of hypertension according to categories of walking volume and speed at baseline (N=83,435).

Exposure	HTN cases (rate [*])	HR (95% CI)					
		Model 1	Model 2	Model 2 [†]	Model 3	Model 4	Model 5
Walking Volume (MET-hr/wk)							
0 (n=24,253)	11,619 (45.8)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
>0-3.5 (n=16,122)	7,549 (44.0)	0.95 (0.93-0.98)	0.98 (0.95-1.01)	---	0.97 (0.94-1.00)	0.98 (0.95-1.01)	0.98 (0.95-1.01)
3.6-7.5 (n=22,791)	10,343 (41.6)	0.90 (0.88-0.93)	0.95 (0.92-0.97)	---	0.96 (0.93-0.99)	0.98 (0.95-1.01)	0.97 (0.95-1.00)
> 7.5 (n=20,269)	8,719 (38.0)	0.83 (0.81-0.86)	0.89 (0.87-0.92)	---	0.91 (0.88-0.93)	0.95 (0.92-0.98)	0.93 (0.91-0.96)
Ptrend	---	<0.001	<0.001	---	<0.001	0.036	0.001
Walking Speed (mph)							
<2 (n=8,971)	4,389 (51.1)	1.08 (1.04-1.11)	1.07 (1.03-1.10)	1.08 (1.04-1.12)	1.06 (1.02-1.09)	1.05 (1.01-1.09)	1.05 (1.01-1.09)
2-3 (n=29,782)	13,680 (42.3)	0.91 (0.88-0.93)	0.95 (0.92-0.97)	0.96 (0.93-0.98)	0.95 (0.92-0.97)	0.97 (0.95-1.00)	0.97 (0.95-1.00)
3-4 (n=19,305)	8,124 (35.9)	0.80 (0.78-0.83)	0.86 (0.84-0.89)	0.86 (0.83-0.89)	0.89 (0.86-0.92)	0.93 (0.90-0.95)	0.90 (0.87-0.93)
>4 (n=1,124)	418 (30.1)	0.71 (0.64-0.78)	0.79 (0.71-0.87)	0.77 (0.69-0.85)	0.81 (0.74-0.90)	0.89 (0.81-0.99)	0.84 (0.76-0.93)
Ptrend	---	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Abbreviations as in Table 1.

^{*} Incidence rate, number of cases/ 1000 person-years.

Model 1: includes study component (WHI OS or CT), randomization to trial arms, age and race-ethnicity.

Model 2: includes Model 1 + education, alcohol, smoking, diet score, dietary sodium, diabetes, hyperlipidemia, cancer, and non-walking PA.

[†] Model 2: includes Model 2 + walking duration (hours/week).

Model 3: includes Model 2 + Systolic and Diastolic BP.

Model 4: includes Model 2 + BMI.

Model 5: includes Model 2 + physical functioning score.

Table 3. Associations between walking volume and hypertension stratified on baseline subgroups.

Characteristic	Cases (rate) ^a	HR ^c (95% CI)	P Interaction
All women (83,435)	38,230 (42.2)	0.95 (0.94, 0.97)	---
Age, years			<0.001
50–59 (n = 32,286)	13,478 (35.3)	0.94 (0.93–0.96)	
60–69 (n = 34,979)	16,934 (45.8)	0.97 (0.96–0.98)	
70 (n = 12,969)	6,328 (53.5)	0.99 (0.96–1.01)	
Race-ethnicity			0.172
White (n = 69,732)	31,755 (41.1)	0.95 (0.94–0.96)	
Black (n = 4,320)	2,392 (64.7)	0.99 (0.96–1.03)	
Hispanic (n = 3,050)	1,313 (47.1)	1.02 (0.97–1.07)	
Asian/Pacific Islander (n = 2,025)	782 (38.6)	1.01 (0.95–1.09)	
Systolic BP (tertiles), mmHg			0.185
112.0 (n = 26,868)	7,431 (21.5)	0.96 (0.94–0.99)	
112.5–124.0 (n = 27,537)	12,914 (43.0)	0.97 (0.95–0.99)	
124.5 (n = 25,755)	16,356 (73.4)	0.97 (0.95–0.98)	
Diastolic BP (tertiles), mmHg			0.713
69.0 (n = 26,865)	9,498 (29.7)	0.97 (0.95–0.99)	
69.5–76.0 (n = 27,788)	12,413 (40.5)	0.96 (0.94–0.98)	
76.5 (n = 25,496)	14,781 (60.9)	0.96 (0.94–0.97)	
BMI , kg/m ²			0.820
<18.5 (n = 1,575)	566 (31.2)	0.95 (0.88–1.03)	
18.5–24.9 (n = 33,760)	13,007 (32.7)	0.99 (0.98–1.01)	
25.0–29.9 (n = 27,957)	13,571 (45.9)	0.97 (0.95–0.98)	
30 (n = 16,942)	9,596 (60.4)	0.99 (0.97–1.01)	
Non-walking PA , MET-hr/wk			0.011
0 (n = 26,414)	12,612 (46.2)	0.94 (0.93–0.96)	
>0–9.3 (n = 27,051)	12,562 (42.9)	0.96 (0.94–0.98)	

Characteristic	Cases (rate)*	HR [†] (95% CI)	P Interaction
> 9.3 (n = 26,769)	11,566 (38.0)	0.98 (0.96–0.99)	

Abbreviations as in Table 1.

* Incidence rate, number of cases / 1000 person-years.

[†] HRs and 95% CIs for a 7.5 MET-hr/week increment in walking volume.

Covariates as in Model 2, Table 2.