Habitual physical activity and central artery stiffening in older adults: the Atherosclerosis Risk in Communities study

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Introduction: Regular physical activity appears to attenuate or even reverse age-related arterial stiffening. Yet, it is not clear if the reduced stiffening associated with habitual physical activity is also observed in community-dwelling older adults.

Methods: Among 3893 older adults in a prospective cohort study, we associated physical activity with measures of central arterial stiffness (via carotid–femoral pulse wave velocity or cfPWV) and pressure pulsatility (via central pulse pressure or cPP). We also examined the association of long-term habitual physical activity, measured as persistence in physical activity levels from mid-life to late-life, with cfPWV and cPP among 1747 participants.

Results: The adjusted mean difference in cfPWV was lower, reflecting less arterial stiffness, for those with moderate ($\beta = -0.30$ m/s) or high ($\beta = -0.38$ m/s) physical activity compared with no physical activity. The adjusted mean difference in cPP was also lower for those with high ($\beta = -2.49$ mmHg) physical activity, relative to no physical activity. Stronger effect estimates were observed among those with persistent physical activity from mid-life to late-life.

Conclusion: Higher physical activity in late-life, and habitual physical activity from mid-life to late-life, is associated with lower central arterial stiffness and pressure pulsatility in a large population-based sample of community-dwelling older adults.

Keywords: aging, arterial compliance, central blood pressure, exercise

Abbreviations: ARIC, Atherosclerosis Risk in Communities; BP, blood pressure; cfPWV, carotid–femoral pulse wave velocity; cPP, central pulse pressure; MET, metabolic equivalent

INTRODUCTION

As illustrated in Thomas Sydenham's axiom that 'man is as old as his arteries,' arterial health, more specifically arterial stiffness, has been regarded as a barometer of biological aging. Most individuals experience stiffening of large conduit arteries with advancing age [1]. Arterial stiffening exerts a number of adverse effects on cardiovascular function and is associated with the development and progression of hypertension, left ventricular hypertrophy, ischemic heart disease, and congestive heart failure [2,3]. The pathogenesis of arterial stiffening includes functional and structural changes in the arterial wall [4,5].

A first-line approach for prevention of cardiovascular disease is lifestyle modification, including regular physical activity [6]. It is well established that habitual exercise, a component of physical activity, is associated with improvements in conventional risk factors for cardiovascular disease in older adults [7]. However, more than 40% of the risk reduction associated with habitual exercise is unexplained by its effects on these traditional risk factors [8]. It is conceivable that some of the beneficial effects of habitual exercise may be through mitigation of age-related vascular dysfunction or arterial stiffening beyond the presence of traditional risk factors. A number of observational and interventional studies indicate that habitual exercise can prevent and reverse arterial stiffening [9–11]. However, the efficacy of exercise in patient populations remains highly controversial as a lack of effects on arterial stiffness has been reported in patients with hypertension, diabetes, and heart failure [12–15]. Additionally, a prior cross-sectional analysis of Atherosclerosis Risk in Communities (ARIC) study data found no association of regular sports or leisure activity with arterial stiffness in community-dwelling middle-aged adults [16]. The reasons for the discrepant results may be because of the following: a narrow range of participants' relatively younger ages, 45–64 years, suggestive of low variability in arterial stiffness measures; the use of arterial distensibility as the measure of arterial stiffening, which does not account for local pulsatile pressure

changes; and lastly, a single measure of physical activity in a sample of mostly sedentary adults. Moreover, prospective studies assessing changes in physical activity and arterial stiffness are very limited [17].

Accordingly, we examined the association between habitual physical activity and arterial stiffness in a community-based biracial cohort of older adults using the most widely established measure of arterial stiffness, carotid–femoral pulse wave velocity (cfPWV), along with repeat measures of physical activity. We hypothesized that higher levels of physical activity and persistence of physical activity over time would be associated with lower arterial stiffness and pressure pulsatility.

METHODS

Participants

The ARIC study is an ongoing, population-based longitudinal study involving four US communities (Forsyth County, North Carolina, USA; Jackson, Mississippi, USA; suburban Minneapolis, Minnesota, USA; and Washington County, Maryland, USA). A total of 6538 participants attended ARIC study visit 5 (2011–2013, age: 66–90 years) and underwent a standardized examination [18]. For the present analyses, we excluded participants with missing information on blood pressure and arterial stiffness, BMI at least $40 \, kg/m^2$, major arrhythmias (Minnesota codes 8-1-3, 8-3-1, and 8-3-2: $\geq 10\%$ atrial and ventricular premature beats, atrial fibrillation, or flutter), peripheral vascular disease (aortic aneurysms, abdominal aorta $\geq 5 \, cm$, history of aortic or peripheral revascularization, presence of an aortic graft, or aortic stenosis), other major cardiovascular disease (history of coronary artery disease, heart failure, or stroke), and moderate or greater aortic regurgitation. Participants who self-identified as neither white nor African American from all centers. and African American from the Minnesota and Maryland field centers, were excluded because of small numbers. After exclusions, the final analytic sample included 3893 participants (median age: 74 years). Institutional review boards approved the study protocol at each field center and participating institutions, and all study participants provided written informed consent.

Measurements

Participants were asked not to consume food or drinks and to refrain from tobacco and vigorous physical activity after midnight prior to the visit or for 8 h prior to the visit. Participants were also asked to bring all prescription and nonprescription medications taken within 2 weeks. Blood samples were obtained following a standardized venipuncture protocol and were assayed in ARIC central laboratories for blood cholesterol, glucose, and insulin concentrations. Diabetes was defined as fasting glucose at least 126 mg/dl, nonfasting glucose at least 200 mg/dl, antidiabetic medication use, or self-report of a physician diagnosis of diabetes.

Brachial blood pressure (SBP, mean BP, and DBP) was measured twice with the participants in the supine position using oscillometric automated sphygmomanometer (VP-1000 Plus; Omron Healthcare, Kyoto, Japan), and the average measurement was used for analyses. cfPWV, an index of arterial stiffness, and carotid artery pressure waveforms, for the subsequent calculation of central pulse pressure (cPP), were obtained in the same testing session using an automatic vascular screening device (VP-1000 Plus; Omron Healthcare) as previously described [19] with excellent reproducibility [20]. Carotid and femoral arterial pressure waveforms were acquired for 30 s by applanation tonometry sensors attached on the left common carotid artery (via neck collar) and left femoral artery (via elastic tape around the hip). In line with prior ARIC publications utilizing cfPWV and cPP data, outlying values at least 3SDs above or below the mean were removed. Carotid blood pressure signal obtained via applanation tonometer was calibrated by equating the carotid mean and DBP to the brachial artery value because the baseline levels of carotid blood pressure are subjected to hold-down force [21]. Carotid blood pressure was derived automatically from the machine. We derived measures of high versus low arterial stiffness/pressure pulsatility from race-combined 25th percentile cut-points of the cfPWV and cPP values. Participants in the upper 25th percentile of the distribution of cfPWV ($\geq 13.26 \, m/s$) and cPP ($\geq 82.1 \, mmHg$) were categorized to have high arterial stiffness/pressure pulsatility. The remaining distribution was categorized to have low arterial stiffness/pressure pulsatility. As a sensitivity analysis, pulse pressure amplification was also derived from the following: [(right brachial SBP − right brachial DBP)/cPP], with a lower pulse pressure amplification indicating greater pressure pulsatility. Measures of low versus high pressure pulsatility were also derived from race-combined 25th percentile cut points of the pulse pressure amplification values. Participants in the lower 25th percentile of the distribution of pulse pressure amplification (≤ 0.84) were categorized to have high pressure pulsatility.

Leisure-time physical activity was assessed through an interviewer-administered modified Baecke Physical Activity Questionnaire during the first (1987–1989) and third (1993–1995) visit examinations of ARIC [22]. In order to eliminate the potential for reverse causality with physical activity and arterial stiffness measured concurrently, persistent physical activity was examined in midlife from the first to third visits. The questionnaire includes asking about the type, duration, and frequency of up to four sport activities they engaged in for the past year. Participants self-reported the number of hours (duration) within a week and number of weeks over a month (frequency) that they performed an activity. Each activity was assigned a metabolic-equivalent (MET) ranging from 1 to 12 METs based on the Compendium of Physical Activities [23]. Assignments were checked by a second reviewer, and discrepancies were resolved by consensus. The total volume of physical activity was estimated using a multiplicative combination of duration, frequency, and intensity (assigned MET value) of all activities to arrive at an overall estimate of average MET-minutes/week of physical activity. Evidence from reliability studies suggests that the Baecke questionnaire has moderate to good reliability (test–retest reliability ranging from 0.74 to 0.88) [22]. The questionnaire has also been shown to have moderate validity (Spearman correlation coefficient = 0.54) against energy expenditure measured with doubly labeled water [24].

Statistical analyses

Distribution-based tertiles (low, moderate, and high) of the overall average MET-min/week were estimated and compared with those reporting no physical activity (0 MET-min/week). Persistence of physical activity (no, low, moderate, or high) was estimated in a subsample of ARIC participants ($n = 1747$ out of $43\,893$ in the analytic sample) who attended visits 1, 3 and 5, and reported the same level of physical activity in mid-life at both visits 1 and 3. We derived measures of high versus low arterial stiffness/pressure pulsatility from race-combined upper 25th percentile cut-points of the cfPWV and cPP values.

Descriptive analysis used chi-square and ANOVA tests to examine differences in demographic and disease characteristics across physical activity categories (no, low, moderate, and high physical activity) at visit 3, and persistence of physical activity categories from visit 1 to visit 3 (persistently no physical activity, persistently low, persistently moderate, and persistently high). Multivariable linear regression was used to estimate the adjusted mean difference in cfPWV (m/s) and cPP (mmHg) across the categories of physical activity (reference: no physical activity). Log-binomial regression was used to estimate the prevalence of high versus low arterial stiffness and pressure pulsatility across physical activity levels (reference: no physical activity). Models were adjusted for continuous age (years), sex, education (<high school, high school, > high school), race-ARIC field center (Jackson-blacks, Forsyth County-blacks, Forsyth County-whites, Minneapolis-whites, Washington County-whites), and smoking status (ever versus never smokers). Mean arterial pressure (MAP) was calculated as $[\{(2 \times DBP) + SBP\}/3]$. Additional analyses examined prevalent hypertension, diabetes, and heart rate as confounders.

RESULTS

Sociodemographic and clinical characteristics of the study population ($n = 3893$) stratified by physical activity levels at ARIC visit 3 are provided in Table 1. Distributional differences across levels of sustained physical activity (persistently no physical activity, persistently low, persistently moderate, persistently high) from ARIC visits 1–3 ($n = 1747$) are also described in Table 1. Participants who did no or low levels of physical activity were more often women and had lower education levels, slightly higher cardiometabolic risk factors (i.e. higher heart rate, BMI, waist circumference, SBP, blood glucose, and triglyceride concentrations), and a higher prevalence of diabetes and hypertension. Similar patterns were observed for the subgroup of participants with persistent levels of physical activity. Unadjusted values of cfPWV and cPP were also slightly higher for those participants with no or low levels of physical activity.

The adjusted mean standardized difference in central arterial stiffness, measured as cfPWV, was significantly lower (reflecting less stiff arteries) for those with moderate [$\beta = -0.30$ m/s; 95% 95% confidence interval (CI) -0.55 to -0.05] or high ($\beta = -0.38$ m/s; 95% CI -0.63 to -0.12) physical activity compared with no physical activity (Table 2). Stronger effect estimates were observed among

TABLE 1. Descriptive sociodemographic and clinical characteristics across physical activity, the Atherosclerosis Risk in Communities study

Characteristics at ARIC visit 3	Physical activity at visit 3 ($N = 3893$)				Persistence of physical activity from visits 1–3 ($N = 1747$)			
	No PA ($n = 1197$)	Low ($n = 899$)	Moderate ($n = 899$)	High ($n = 898$)	No PA ($n = 694$)	Low ($n = 298$)	Moderate ($n = 283$)	High ($n = 472$)
MET-min/week	0	4–612	613–1194	1195–5486	0	13–612	613–1194	1198–5486
Age (years)	74.6 (4.7)	75.2 (4.9)	75.6 (5.1)	75.7 (5.2)	74.5 (4.6)	75.0 (4.8)	75.1 (5.0)	75.6 (5.2)
Female sex, n (%)	728 (61)	605 (67)	542 (60)	431 (48)	435 (63)	205 (69)	171 (60)	271 (57)
Black race, n (%)	349 (29)	173 (19)	158 (18)	121 (14)	264 (38)	53 (18)	41 (15)	43 (9)
Less than HS education, n (%)	238 (20)	96 (11)	79 (9)	48 (5)	160 (23)	25 (8)	13 (5)	11 (2)
Ever smokers, n (%)	633 (56)	460 (53)	489 (56)	521 (60)	375 (57)	150 (53)	143 (52)	281 (62)
Heart rates (beat/min)	63 (10)	62 (10)	62 (10)	61 (9)	63 (10)	61 (9)	61 (9)	60 (10)
BMI (kg/m^2)	28.5 (4.6)	27.9 (4.5)	27.4 (4.2)	27.1 (4.2)	28.7 (4.8)	27.6 (4.3)	27.2 (4.1)	26.7 (4.0)
Waist circumference (cm)	100 (12)	99 (12)	98 (12)	98 (12)	100 (13)	99 (12)	98 (12)	98 (11)
SBP (mmHg)	130 (17)	130 (18)	130 (17)	130 (17)	131 (18)	131 (17)	131 (17)	129 (16)
DBP (mmHg)	67 (10)	66 (10)	66 (10)	67 (10)	69 (10)	66 (10)	67 (10)	67 (10)
HDL-C (mg/dl)	51 (13)	54 (14)	53 (13)	54 (13)	51 (13)	54 (14)	54 (14)	54 (13)
Insulin (μU/ml)	14.2 (14.9)	12.9 (9.3)	12.6 (9.6)	11.9 (9.0)	15.0 (18.2)	13.2 (9.3)	12.2 (8.3)	11.1 (8.0)
Glucose (mg/dl)	113 (28)	113 (30)	111 (25)	110 (22)	113 (29)	112 (33)	109 (19)	109 (20)
Triglycerides (mg/dl)	127 (60)	125 (55)	123 (58)	118 (56)	125 (58)	126 (55)	121 (55)	116 (51)
TG-HDL ratio	2.7 (1.8)	2.6 (1.6)	2.6 (1.6)	2.5 (1.6)	2.7 (1.6)	2.6 (1.6)	2.5 (1.5)	2.4 (1.5)
HOMA-IR	4.1 (5.5)	3.8 (3.4)	3.6 (3.3)	3.3 (2.9)	4.4 (6,7)	3.8 (3.3)	3.4 (2.7)	3.1 (2.7)
Diabetes, n (%)	340 (29)	218 (24)	191 (22)	180 (20)	209 (30)	61 (21)	51 (18)	77 (17)
Hypertension, n (%)	911 (77)	633 (71)	630 (71)	591 (67)	544 (79)	201 (68)	202 (72)	300 (64)
cfPWV, m/s	11.9 (3.1)	11.6 (2.8)	11.5 (2.9)	11.5 (3.0)	12.0 (3.2)	11.6 (2.9)	11.7 (3.0)	11.1 (2.8)
cPP (mmHg)	71 (18)	72 (17)	71 (17)	69 (17)	72 (19)	73 (16)	71 (17)	69 (17)
High cfPWV, n (%)	330 (28)	223 (25)	225 (25)	199 (22)	204 (29)	75 (25)	72 (25)	86 (18)
High cPP, n (%)	324 (27)	249 (28)	224 (25)	173 (19)	198 (29)	80 (27)	74 (26)	79 (17)
Low PA, n (%)	309 (26)	216 (24)	207 (23)	166 (18)	177 (26)	56 (19)	68 (24)	87 (18)

Data are means ± SD unless stated otherwise. ARIC, Atherosclerosis Risk in Communities study; BP, blood pressure; cfPWV, carotid–femoral pulse wave velocity; cPP, central pulse pressure; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostatic model assessment of insulin resistance; MET, metabolic equivalent; PA, pulse pressure amplification; TG, triglycerides.

TABLE 2. Adjusted mean difference (95% confidence interval) in arterial stiffness and pulsatility at visit 5 across physical activity levels at visit 3 and persistence of physical activity from visit 1–3, the Atherosclerosis Risk in Communities study

Physical activity	Visit 3, PA, $n = 3893$	Persistence of PA from visit 1 to visit 3, $n = 1747$
Carotid–femoral pulse wave velocity (m/s)		
No PA	Reference	Reference
Low	−0.18 (−0.42 to 0.07)	−0.18 (−0.58 to 0.22)
Moderate	−0.30 (−0.55 to −0.05)*	−0.21 (−0.62 to 0.21)
High	−0.38 (−0.63 to −0.12)*	−0.73 (−1.10 to −0.36)*
Central pulse pressure (mmHg)		
No PA	Reference	Reference
Low	−0.07 (−1.48 to 1.34)	0.19 (−2.05 to 2.43)
Moderate	−0.89 (−2.30 to 0.52)	−1.13 (−3.44 to 1.17)
High	−2.49 (−3.92 to −1.05)*	−4.19 (−6.28 to −2.11)

Models adjusted for age, gender/sex, education, race-center, smoking status, and mean arterial pressure. PA, physical activity.
*$P < 0.05$ versus reference.

the subsample with persistently high levels of physical activity from mid-to-late life (Table 2). Results were robust to further adjustment for prevalent hypertension and heart rate but attenuated after adjustment for prevalent diabetes (data not shown).

The adjusted mean difference in central pressure pulsatility, measured as cPP, was significantly lower only for those with high ($\beta = -2.49$ mmHg; 95% CI −3.92 to −1.05] physical activity (Table 2), relative to no physical activity. Among those with persistent levels of physical activity, those with persistently high physical activity ($\beta = -4.19$ mmHg; 95% CI −6.28 to −2.11] also had a significantly lower cPP compared with those with persistently no physical activity. Results were robust to further adjustment for prevalent diabetes and hypertension, and heart rate (data not shown).

High levels of physical activity were associated with a lower adjusted prevalence of high cfPWV versus low cfPWV (Table 3). Although only statistically supported for high levels of physical activity, a graded relationship showing a lower prevalence of high cfPWV was observed across low

TABLE 3. Adjusted prevalence ratio (95% confidence interval) of high arterial stiffness and pulsatility at visit 5 across physical activity levels at visit 3 and persistence of physical activity from visits 1 to 3, the Atherosclerosis Risk in Communities study

Physical activity	Visit 3, PA, $n = 3893$	Persistence of PA from visit 1 to visit 3, $n = 1747$
High versus low carotid–femoral pulse wave velocity		
No PA	Reference	Reference
Low	0.94 (0.81–1.09)	0.97 (0.77–1.22)
Moderate	0.92 (0.80–1.06)	0.95 (0.75–1.20)
High	0.83 (0.71–0.97)*	0.72 (0.56–0.91)*
High versus low central pulse pressure		
No PA	Reference	Reference
Low	1.00 (0.87–1.15)	0.91 (0.73–1.13)
Moderate	0.93 (0.80–1.08)	0.91 (0.72–1.15)
High	0.79 (0.67–0.93)*	0.66 (0.51–0.85)*
Low versus high pulse pressure amplification		
No PA	Reference	Reference
Low	0.93 (0.80–1.09)	0.80 (0.61–1.06)
Moderate	0.88 (0.75–1.03)	1.04 (0.80–1.34)
High	0.75 (0.63–0.90)*	0.85 (0.66–1.09)

Models adjusted for age, gender/sex, education, race-center, smoking status, and mean arterial pressure. PA, physical activity.
*$P < 0.05$ versus reference.

(prevalence ratio: 0.94; 95% CI 0.81 to 1.09), moderate (prevalence ratio: 0.92; 95% CI 0.80 to 1.06), and high (prevalence ratio: 0.83; 95% CI 0.71 to 0.97) levels of physical activity, relative to those with no physical activity. Similar patterns were observed when restricted to participants with persistent levels of physical activity from mid-to-late life (Table 3). However, the results were attenuated and no longer statistically supported after adjustment for prevalent diabetes, hypertension, and heart rate (data not shown). For the outcome of pressure pulsatility, only high physical activity was also associated with a lower prevalence of high cPP (prevalence ratio: 0.79; 95% CI 0.67−0.93) and low PA (prevalence ratio: 0.75; 95% CI 0.63−0.90), respectively (Table 3). Results were robust to further adjustment for prevalent diabetes and hypertension, and heart rate (data not shown).

DISCUSSION

The present study is one of the largest studies to evaluate the relationship between habitual physical activity and central arterial stiffness in older adults aged 75 years and older. Both central arterial stiffness and pressure pulsatility were significantly lower in community-dwelling older adults who habitually engaged in physical activity. More specifically, we observed that a high level of physical activity was associated with lower cfPWV and central pulse pressure in the cross-temporal analyses. These results remained significant even after further adjustment for diabetes and hypertension. We observed stronger associations when restricting to participants with persistent levels of physical activity from mid-to-late life. Taken together, these results are consistent with the notion that habitual physical activity is associated with lower arterial stiffness in older adults.

The present study is not the first to investigate the association between regular physical activity and arterial stiffness using the well characterized ARIC study cohort. In contrast to the present study, the previous ARIC study [16] did not support the role of habitual physical activity in modulating age-related arterial stiffening. In light of these disparate results, we should note two important differences between these studies. First, the ages of the participants were 45−64 years in the previous study [16]. Due, at least in part, to the 'law of initial baseline' (the baseline effect where

greater reductions in arterial stiffness can be expected in participants with elevated arterial stiffness at baseline as more space is available to favor larger changes), the influence of lifestyle modifications, including exercise training, appears to manifest more clearly in older adults with elevated baseline (pretraining) levels of arterial stiffness [10,11]. Second, in the previous study, arterial distensibility was measured using arterial diameter change at the carotid artery and pulse pressure measured at the brachial artery [16]. Because of the measurement location specificity and a wide variance between the central and peripheral blood pressure readings, this approach to measuring arterial stiffness has important caveats [25]. In the present study, we used cfPWV that has become the reference standard for the measurement of arterial stiffness [26]. Our present results are consistent with a recent community-based study from Europe showing that moderate-to-vigorous physical activity was associated with a slower age-related progression of central arterial stiffness [17].

Exercise intervention studies have reported that exercise training programs composed of aerobic (endurance) exercise are effective in reversing arterial stiffening associated with advancing age in apparently healthy adults [10]. However, existing intervention studies do not address persistent habitual activity over the long-term, and the efficacy of regular exercise in patient populations remains highly controversial as a lack of effects on arterial stiffness has been reported in patients with hypertension, diabetes, and heart failure [12–15]. Many of the participants in the present study had hypertension and diabetes and other chronic diseases. Yet an inverse association between regular physical activity and arterial stiffness was evident. More importantly, even low volume of physical activity that these patient populations could tolerate were associated with reduced arterial stiffening, compared with no physical activity. Therefore, older patients should be encouraged to perform physical activity to potentially reduce arterial stiffening and therefore, hopefully reduce cardiovascular disease risk.

Physiological mechanisms underlying the influences of regular exercise on arterial stiffness are not fully understood. The elastin–collagen composition of the arterial wall changes over a period of years, yet destiffening of central arteries can be accomplished with several months of exercise training interventions [10]. Accordingly, it is unlikely that this may be a physiological mechanism underlying reductions in arterial stiffness. An exercise-stimulated reduction in vasoconstrictor tone exerted by the vascular smooth muscle cells is thought to be a primary mechanism. Indeed, a study using systemic inhibition of α-adrenergic receptors and nitric oxide synthase on arterial compliance before and after 3 months of aerobic exercise training in middle-aged and older adults indicate that the removal of tonic restraint exerted by sympathetic vasoconstrictor tone abolished the training-induced reductions in arterial stiffness [27]. Further work is needed to understand the physiological mechanisms relating physical activity to reduced arterial stiffening.

A major strength of the present study is that the inclusion of repeat measures of physical activity adds to the existing literature by expanding our understanding of the impact of habitual physical activity, measured as persistence of physical activity levels over two measurements on average 6 years apart, on arterial stiffening. Another strength is the use of data from the well characterized, biracial population-based ARIC cohort study. In all the field centers that the data were collected, the measurements were carefully standardized, the same device was used at all the centers, and the written procedures were strictly followed. This has resulted in smaller variabilities associated with the measurements compared with other published data [28]. Notably, the mean values of cfPWV obtained in the present US population are very similar to those collected in European populations with similar age and blood pressure categories [28].

Major limitations of the present study should also be considered. First, the reported associations between physical activity and arterial stiffness are largely based on one cross-sectional measure of arterial stiffening in older adults ($n = 3893$), which, therefore, does not allow for discerning causality from consequence. However, the magnitude of the strength of the association tended to be greater whenever the associations were examined in a smaller subsample of participants ($n = 1747$) who self-reported the same level of physical activity across 6 years in mid-life. Second, longitudinal changes in physical activity were assessed as persistence of physical activity in mid-life, which reflects a time span when comorbidities and lifestyle changes associated with retirement can have profound impacts on levels of physical activity. Third, corresponding changes in arterial stiffness were not available; data collection is currently underway in the ARIC cohort to examine age-related changes in arterial stiffness prospectively over 5 years. Lastly, we estimated physical activity using the interviewer-administered self-reported Baecke physical activity questionnaire, which is subject to report and recall errors.

In conclusion, this large, biracial, population-based cohort study demonstrated that regular physical activity was associated with lower levels of central arterial stiffness and pressure pulsatility in community-dwelling older adults. We observed a mild attenuation of these associations after adjustment for cardiovascular disease risk factors, including diabetes and hypertension. These results are consistent with the public health messages that even moderate levels of regular physical activity are associated with the attenuation of vascular stiffening that occurs with advancing age.

Conflicts of interest

There are no conflicts of interest.

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Reviewer's Summary Evaluation

Reviewer 2

A number of relevant publications on this subject clearly demonstrated, in cross-sectional analyses, a dose-dependent inverse association between levels of physical activity and arterial stiffness. At variance, interventional studies performed with a randomized controlled design, provided inconsistent conclusions on the effects of physical activity in reducing arterial stiffening, especially when results were adjusted for concomitant BP changes. Therefore, much of this apparently strong association could be mediated by reverse causality or confounding effect. This represent, to my perspective, a relevant limitation to the present study.