

ORIGINAL ARTICLE

Effects on carotid–femoral pulse wave velocity 24 h post exercise in young healthy adults

Sophy J Perdomo¹, Anne M Moody², Stephanie M McCoy¹, Emma Barinas-Mitchell³, John M Jakicic¹ and Bethany Barone Gibbs¹

Arterial stiffness, often measured by carotid–femoral pulse wave velocity (cfPWV), is a subclinical marker of cardiovascular disease that is known to be reduced by exercise training. Exercise is also known to have acute vascular effects, yet it is unclear whether exercise 24 h before cfPWV testing influences this outcome. Thirty healthy, young adults completed a supervised, 30-min bout of moderate-to-vigorous intensity treadmill running. cfPWV, systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were measured both before (after 48 h of abstaining from exercise) and 24 h after (with no additional exercise) the exercise session. From pre-exercise to 24 h post exercise, cfPWV decreased from 6.05 ± 0.82 to 5.84 ± 0.87 m s⁻¹ ($P = 0.02$), SBP from 119.7 ± 13.8 to 116.8 ± 11.4 mm Hg ($P = 0.03$) and DBP from 65.1 ± 5.7 to 63.2 ± 5.4 mm Hg ($P = 0.02$), with no significant changes in HR. cfPWV was positively correlated with SBP pre-exercise ($r = 0.54$, $P < 0.01$) and post exercise ($r = 0.53$, $P < 0.01$). Changes in blood pressure explained 4–5% of the variability in cfPWV change; adjustments slightly attenuated the 24-h effects of exercise on cfPWV. Some evidence of gender differences was observed with higher cfPWV in males across assessments ($P < 0.05$) and statistically significant reductions in cfPWV in males (-0.36 ± 0.54 m s⁻¹ ($P = 0.02$)) but not in females (-0.07 ± 0.31 m s⁻¹ ($P = 0.41$)). In conclusion, cfPWV decreased 24 h after an exercise bout, suggesting that exercise completed in the past 24 h should be considered before cfPWV testing.

Hypertension Research (2016) 39, 435–439; doi:10.1038/hr.2015.161; published online 14 January 2016

Keywords: Exercise; pulse wave analysis; vascular stiffness

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death worldwide, and it is projected to remain the leading cause of death by 2030.^{1,2} It is well established that aerobic exercise reduces CVD risk² by reducing CVD risk factors, including blood pressure and arterial stiffness.^{3–5} In addition to chronic training adaptations, exercise is known to have acute benefits on blood pressure immediately and up to 24 h post exercise.^{6,7} However, whether exercise has a similar effect on arterial stiffness 24 h after an exercise bout is not well studied.

Carotid–femoral pulse wave velocity (cfPWV) is the non-invasive, gold-standard method for assessing arterial stiffness.⁸ Vascular testing protocols, including cfPWV, typically instruct subjects to refrain from activities that could introduce measurement error (for example, smoking, caffeine and eating) before testing.^{4,8} Some vascular testing protocols (for example, arterial compliance and flow-mediated dilation) also instruct subjects to refrain from exercise preceding the assessment because of the potential transient effects of exercise on vascular function.^{9,10} Although it is known that exercise acutely increases arterial compliance,¹¹ several cfPWV consensus statements do not recommend that subjects refrain from exercise before

testing.^{8,12,13} Few studies have evaluated ‘next-day’ effects of exercise on arterial stiffness with varied results; however, most studies have been small and have used heterogeneous methods.^{14–17} Further, these studies have not examined the effects by gender, although gender is known to have an effect on arterial stiffness.¹⁸ Determining whether transient changes in cfPWV are present 24 h after exercise is important for reducing potential variability during vascular testing procedures. Thus, the purpose of this study was to examine the effects of a 30-min bout of treadmill running at a moderate-to-vigorous intensity on cfPWV after 24 h. We hypothesized that a single exercise session would decrease cfPWV. Secondary objectives were to evaluate whether this effect differed by gender or was related to concomitant changes in resting blood pressure or resting HR.

METHODS

Participants

Thirty healthy men ($N = 15$) and women ($N = 15$), 18–35 years old, volunteered to participate in the study (Table 1). Recruitment was completed through advertisements, with most of the sample comprising students and staff of the University of Pittsburgh. Written informed consent was obtained for all

¹Department of Health and Physical Activity, University of Pittsburgh, Pittsburgh, PA, USA; ²Department of Health and Exercise Science, Appalachian State University, Boone, NC, USA and ³Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA, USA
Correspondence: SJ Perdomo, Department of Health and Physical Activity, University of Pittsburgh, 32 Oak Hill Ct, Pittsburgh, PA 15219, USA.
E-mail: Sjp80@pitt.edu

Previous presentation: This study was presented as an abstract at the American College of Sports Medicine Annual Meeting in Orlando, FL 27–31 May, 2014.

Received 3 August 2015; revised 11 November 2015; accepted 8 December 2015; published online 14 January 2016

subjects before experimental procedures. Approval for this study was granted by the Institutional Review Board at the University of Pittsburgh.

Exclusion criteria were pre-established and included any medical condition or medication affecting energy metabolism or cardiovascular response, history of cardiac events or conditions, current pregnancy, obesity (body mass index (kg m^{-2}) $\geq 30 \text{ kg m}^{-2}$), high-risk classification according to the American College of Sports Medicine criteria¹⁹ or a self-reported inability to run for 30 min.

Experimental procedures

Each participant visited the laboratory on three separate occasions. During the first workload estimation visit, a treadmill speed needed to elicit the desired heart rate (HR) for a moderate-to-vigorous intensity bout (70–75% of age-predicted maximum HR) was determined. Participants began with a 5-min warm-up at a speed of 3.0 miles per hour (mph) at 0% grade, after which the speed was increased to 5.0. If the target HR was not reached by the third minute, the speed was increased by 0.5 mph every 3 min until the target HR was achieved.

The second exercise session visit occurred a minimum of 2 days after the first visit, as participants were instructed to refrain from exercise for 48 h. Participants were also instructed to fast for 12 h before the second visit and verbal confirmation of protocol compliance was obtained. During this visit, cPWV was measured, after which the participant performed the exercise session. The exercise session included a 5-min warm-up at 3.0 mph, 30 min of moderate-to-vigorous intensity treadmill running and a 5-min cool down at 3.0 mph. To achieve the targeted moderate-to-vigorous intensity, the 30-min exercise bout was initiated at the predetermined workload from the first visit. Thereafter, the HR was monitored with a Polar monitor and the treadmill speed was adjusted by ± 0.2 mph if the last minute of each 5-min interval was either above or below the 70–75% age-predicted maximum HR. Using this method, participants achieved average HRs during the exercise sessions of $73.0 \pm 4.9\%$ of age-predicted maximum HR.

Before the third visit, which occurred 24 h later, participants were instructed to refrain from any additional exercise and fast for 12 h. This was confirmed before cPWV testing.

Assessments

Anthropometrics. Height and weight were measured with the participant wearing lightweight clothing and no shoes. Weight was measured to the nearest 0.1 kg on a Tanita WB 110A digital scale, and height was measured to the nearest 0.1 cm using a stadiometer. Body mass index was computed from weight and height.

Table 1 Characteristics of participants ($n=30$)

Characteristic	Overall	Male	Female
Age (years)	23.8 \pm 2.5	23.4 \pm 1.8	24.3 \pm 3.0
Weight (kg)	68.9 \pm 9.9	73.8 \pm 6.6 ^a	63.9 \pm 10.3 ^a
Height (cm)	170.3 \pm 8.4	175.8 \pm 6.4 ^a	164.7 \pm 6.3 ^a
BMI (kg m^{-2})	23.7 \pm 2.2	23.9 \pm 1.7	23.4 \pm 2.6

Abbreviation: BMI, body mass index.

Data are presented as mean \pm s.d.

^aSignificant gender differences $P < 0.01$.

Table 2 Pre-exercise, 24-h post-exercise and 24-h change in outcomes

Measure	Pre-exercise	24-h post-exercise	Change	P-value
Pulse wave velocity (m s^{-1})	6.05 \pm 0.82	5.84 \pm 0.87	–0.22 \pm 0.46	0.02
Systolic blood pressure (mm Hg)	119.7 \pm 13.8	116.8 \pm 11.4	–2.90 \pm 6.8	0.03
Diastolic blood pressure (mm Hg)	65.1 \pm 5.7	63.2 \pm 5.4	–1.88 \pm 4.3	0.02
Heart rate (bpm)	62.1 \pm 10.0	61.6 \pm 10.9	–0.55 \pm 6.7	0.65

Abbreviations: BP, blood pressure; bpm, beats per minute.

Data are presented as mean \pm s.d.

Pulse wave velocity. cPWV was measured using the Complior Analyse (Alam Medical; Vincennes, France) on the right side of the body by a trained technician between 0700 and 1000 hours. Subjects laid supine for a 10-min rest period before cPWV measurement.⁸ Using a tape measure, aortic distance was estimated by subtracting the distance from the carotid artery site to the sternal notch from the distance of the sternal notch to the femoral artery site.^{5,12,20} Piezoelectric sensors were held in place to obtain 10 valid waveforms. This process was repeated until three runs of 10 waveforms were captured. Average cPWV (m s^{-1}) was calculated as aortic distance divided by the average time differential between the foot of the waveform at the carotid and femoral sites within each run, and then averaged across the three runs. Inter-rater reliability of technicians in our laboratory ranged from 85 to 96% in 2014.

Resting blood pressure and HR. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and HR measurements were obtained after a 10-min supine rest immediately before and following cPWV testing with an automated Dinamap V100 (GE Healthcare, Pittsburgh, PA, USA). Pre- and post-cPWV measurements were averaged for analysis.

Statistical analysis

All variables were evaluated for normality. Our sample size ($n=30$) was calculated to provide 90% power to detect an effect size of 0.5 for the change in cPWV, assuming a correlation of $r=0.5$ from pre- to post-exercise within individuals and a type I error rate of 0.05. Descriptive statistics were summarized overall and by gender with means (s.d.'s) and compared across genders by two-sided independent *t*-tests. Effect sizes were calculated using Cohen's *d* formula.²¹ Equality of variance across groups was confirmed with Levene's test. Changes in cPWV, SBP, DBP and HR were evaluated using two-sided paired *t*-tests. Pearson's correlations between changes in cPWV and changes in SBP, DBP and HR were examined to evaluate potential mechanisms through which exercise might influence cPWV. Linear regression was used to examine whether changes in cPWV were independent of changes in SBP, DBP and HR in separate models.^{11,22–24} The coefficient of determination (R^2) was calculated to describe common variance between the change in cPWV and changes in SBP, DBP and HR. A 2×2 factorial analysis of variance was used to evaluate a gender \times exercise interaction, and equality of covariance matrices was confirmed by Box's test. Error bars were calculated as s.e.m.

RESULTS

Twenty-four-hour post-exercise changes in outcomes

The sample had a mean age of 24 ± 3 years and a mean body mass index of $23.7 \pm 2.2 \text{ kg m}^{-2}$ (Table 1). These were similar in males and females. Pre- and post-exercise outcomes are displayed in Table 2. Overall, cPWV, SBP and DBP significantly decreased 24 h post exercise from baseline ($P < 0.05$). The mean cPWV decreased from 6.05 ± 0.82 to $5.84 \pm 0.87 \text{ m s}^{-1}$ ($P = 0.02$), with an effect size of 0.26. Because one subject had a large decrease in cPWV (-1.90 m s^{-1}), we repeated analyses using a non-parametric related samples Wilcoxon-signed rank test and found similar results ($P = 0.01$). Results were also similar when we repeated the paired *t*-test after excluding this subject ($-0.16 \pm 0.33 \text{ m s}^{-1}$, $P = 0.02$). Individual changes in cPWV 24 h post exercise are displayed by gender in Figure 1. The mean SBP decreased from 119.7 ± 13.8 to $116.8 \pm 11.4 \text{ mm Hg}$ ($P = 0.03$), with an effect

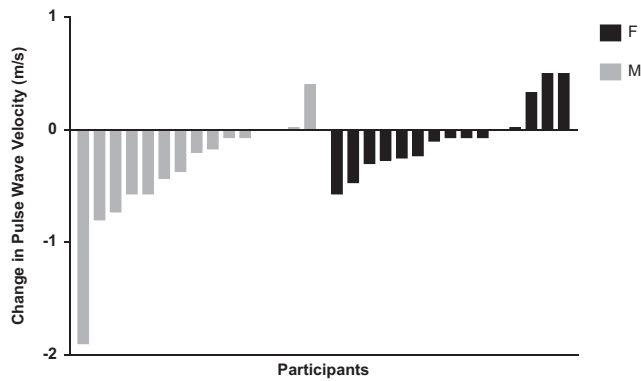


Figure 1 Changes in carotid-femoral pulse wave velocity 24 h post exercise. Each bar represents the change in carotid-femoral pulse wave velocity (cfPWV) from pre- to 24 h post-exercise in each participant. Black bars represent females and gray bars represent males.

size of 0.21; DBP decreased from 65.1 ± 5.7 to 63.2 ± 5.4 mm Hg ($P=0.02$), with an effect size of 0.33. HR did not change ($P=0.65$).

Gender differences

Pre- and post-exercise outcomes by gender are displayed in Table 3. Figures 1 and 2 illustrate gender differences in cfPWV. The mean cfPWV in men was 12% higher pre-exercise and 8% higher post-exercise when compared with their female counterparts (main effect of gender $F=4.30$; $P=0.047$). The mean post-exercise cfPWV was reduced by 0.36 ($P=0.02$; 5.6%) in males and 0.07 ($P=0.41$; 1.2%) in females. The gender \times exercise interaction failed to reach statistical significance ($F=3.38$; $P=0.08$). SBP was also higher in males; however, changes in SBP as well as DBP and HR were similar across genders.

Correlations

cfPWV was correlated with SBP pre-exercise ($r=0.544$, $P<0.01$) and post-exercise ($r=0.529$, $P<0.01$), and the magnitude of the correlation was higher in females. Furthermore, the change in SBP was correlated with change in cfPWV only in females ($r=0.525$, $P<0.05$). cfPWV was not significantly correlated with pre-exercise, post-exercise or change in DBP. Although pre-exercise and post-exercise HR were not significantly correlated with cfPWV, change in HR was correlated with change in cfPWV only in males (0.541 , $P<0.01$; Table 4). Changes in cfPWV were adjusted for changes in SBP, DBP and HR in separate models. Changes in SBP, DBP and HR explained 5.2%, 4.1% and 9.8% of the variability in cfPWV change, respectively. The 24-h effects of exercise on cfPWV were attenuated to -0.17 ($P=0.06$) after adjustment for changes in SBP, to -0.18 ($P=0.06$), after adjustment for changes in DBP, and to -0.20 ($P=0.02$), after adjustment for changes in HR (Table 4).

DISCUSSION

The main finding of the present study is that cfPWV had a small, but significant, decrease 24 h post exercise in a group of healthy, young adults. This was accompanied by significant decreases in resting SBP and DBP. Changes in blood pressure explained a small proportion of the variability in changes in cfPWV. Males had a higher cfPWV both pre- and post exercise and had a significantly decreased post-exercise cfPWV, whereas females did not.

Few studies have evaluated the effect of exercise on arterial compliance 24 h post exercise, and these have yielded mixed

Table 3 Pre-exercise, 24-h post exercise and 24-h change in outcomes by gender

Measure	Pre-exercise	24-h post-exercise	Change	P-value
<i>Pulse wave velocity ($m s^{-1}$)</i>				
Male	6.42 ± 0.83^a	6.06 ± 0.99	-0.36 ± 0.54	0.02
Female	5.69 ± 0.64^a	5.62 ± 0.70	-0.07 ± 0.31	0.41
<i>Systolic blood pressure (mm Hg)</i>				
Male	127.4 ± 12.4^a	123.9 ± 7.7^a	-3.5 ± 7.9	0.11
Female	112.1 ± 10.8^a	109.7 ± 10.0^a	-2.3 ± 5.8	0.14
<i>Diastolic blood pressure (mm Hg)</i>				
Male	65.0 ± 6.2	62.8 ± 5.5	-2.2 ± 4.1	0.06
Female	65.2 ± 5.3	63.6 ± 5.4	-1.6 ± 4.6	0.21
<i>Heart rate (bpm)</i>				
Male	63.0 ± 9.6	63.2 ± 10.1	0.23 ± 8.1	0.91
Female	61.3 ± 10.7	60.0 ± 11.8	-1.3 ± 5.1	0.33

Abbreviations: BP, blood pressure; bpm, beats per minute.

Data are presented as mean \pm s.d.

^aSignificant gender differences $P<0.05$.

results.^{14–17} Our results are similar to a study by Michaelides *et al.*,¹⁵ where cfPWV was reduced 24 h after a 30-min bout of walking in 60 older adults with a mean age of 61.4 ± 1.0 years.¹⁵ Three other studies found no effect of exercise on arterial compliance.^{14,16,17} Kingwell *et al.* found no change in cfPWV among 12 young adult males (24 ± 6 years) 24 h after a 30-min bout of cycling at 65% of maximal oxygen consumption (VO_2 max).¹⁷ Aizawa *et al.*¹⁴ found no difference in carotid and brachial arterial distensibility using high-resolution ultrasound in nine hypertensive older individuals (68.2 ± 5.4 years) 24 h after a maximal treadmill exercise test.¹⁴ Nickle *et al.*¹⁶ found no difference in large and small arterial compliance using Diastolic Pulse Contour Analysis in 32 older individuals (71 ± 7 years) 24 h after a 30-min bout of cycling at 50% VO_2 max.¹⁶ Although not statistically significant, Nickle *et al.*¹⁶ demonstrated that both large and small arterial elasticity increased (improved) 24 h following exercise. The different results could be attributed to small sample sizes, different methodologies used to measure arterial stiffness, different experimental exercise protocols and differences in participant characteristics.

Other studies have evaluated more proximal (for example, 30 min after) and longer-term (for example, ≥ 1 month) effects of exercise on arterial stiffness, typically showing a benefit. Arterial stiffness has been observed to decrease immediately after,²⁵ and between 2 and 60 min after aerobic exercise.^{16,17,26–29} Aerobic training studies of longer duration (6 days to 8 months) have also reduced arterial stiffness.^{30–36}

Several physiological mechanisms could contribute to the 24-h reduction in cfPWV observed in the current study. Exercise increases the requirements of the muscle for oxygen and nutrients, therefore increasing blood flow and shear stress, both of which raise the production of nitric oxide.¹¹ Nitric oxide is a potent vasodilator, reducing vascular smooth muscle cell tone,^{11,29} which transfers stress from collagen fibers to elastin.^{17,37} This shift increases arterial compliance and decreases cardiac load due to elastin's crucial role in determining vascular distensibility.²⁹ Furthermore, changes in the

expression of other vasodilators, vasoconstrictors, inflammatory mediators, reactive oxygen species or antioxidants may contribute.¹¹ Consistent with these mechanisms, SPB and DBP are reduced at 30 min as well as 24 h following exercise,⁷ the latter of which was measured and observed in the current study. Other studies have found that changes in blood pressure do not appear to be fully responsible for changes in cfPWV.²⁷ This was also consistent with our study, which found that controlling for changes in SBP and DBP only slightly attenuated the effects of exercise on cfPWV and explained 4–5% of the variability in changes in cfPWV.

Repeated bouts of exercise over time are also believed to benefit cfPWV through structural vascular adaptations.¹⁷ Chronic increases in the blood flow and shear stress during exercise induce a larger vessel diameter,³⁹ better endothelial function,² increased antioxidant enzyme activity, improved physiological function and enhanced systemic resistance to oxidative stress.⁴⁰ These changes are thought to benefit arterial compliance, consequently reducing risk for CVD. Although we would not expect these chronic benefits to contribute to the 24-h decreases in cfPWV observed in this study, these adaptations are likely additive to the transient effects of exercise because we observed a 24-h effect among young, healthy volunteers who self-reported an ability to run for 30 min. Thus, chronic and acute transient effects may contribute to the benefit of exercise on arterial stiffness.

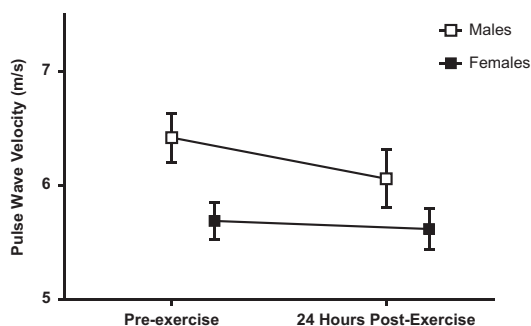


Figure 2 Gender differences in carotid–femoral pulse wave velocity pre-exercise and 24 h post exercise. The mean carotid–femoral pulse wave velocity (cfPWV; \pm s.e.m.) for men was higher when compared with their female counterparts (main effect of gender $F=4.30$; $P=0.047$). There was a significant decrease in cfPWV from pre- to post exercise (main effect of exercise $F=7.28$; $P=0.012$). Males had a larger reduction in post-exercise cfPWV compared with females, the gender \times exercise interaction, however, failed to reach statistical significance ($F=3.38$; $P=0.077$).

The overall gender differences found in our study are consistent with Mitchell *et al.* who found that males had a higher cfPWV compared with their female counterparts.⁴¹ These gender differences could be due to differences in estrogen receptor- α on smooth muscle cells,⁴² differences in hormone and endothelin-1 production or differences in resting blood pressure between males and females.²⁶ Despite gender differences in cfPWV, the effect of exercise on cfPWV was not statistically different by gender (gender \times exercise interaction, $F=3.38$; $P=0.08$). However, this trend might have reached statistical significance with a larger sample size and, within gender, the decrease in cfPWV was only statistically significant in males. One potential reason for the greater reduction in cfPWV among males is that, although intensity was controlled, males ran at a significantly higher speed during their exercise session compared with females (5.4 vs. 4.9 mph, $P=0.036$). Another potential explanation could be that males in our study had higher pre-exercise cfPWV. Lower cfPWV in females could have resulted in a ‘floor’ effect, where there was little room for further reductions, and could have contributed to the more pronounced 24-h decrease in cfPWV in males compared with females. Whether these gender differences would persist in a study population with stiffer or more comparable arteries is an area of further research.

One implication of our results is that exercise performed up to 24 h before cfPWV testing in a research or clinical setting should be considered. Although small, the systematic decrease we observed could introduce error. For example, a testing protocol could require patients to refrain from exercise at least 24 h before cfPWV testing. Viewed another way, our findings in healthy, young adults suggest that regular bouts of exercise (for example, on most days of the week) could be important to realize the maximum acute and chronic benefits on arterial stiffness and cardiovascular health in this population.

The present study has a number of strengths. Arterial stiffness was measured by cfPWV, which is the gold standard,⁸ exercise sessions were supervised to standardize intensity and there was a sufficient sample size to detect small changes in 24-h cfPWV. Some limitations should be considered. Our study used a single-arm pre-post design, and thus our results are hypothesis-generating and should be interpreted cautiously. Of note, because some participants may have been unfamiliar with the cfPWV procedure, ‘white coat’ effects resulting in an increased sympathetic tone could have been stronger in the pre-exercise compared with the post-exercise assessments and may have contributed to the observed differences.^{8,43,44} In addition,

Table 4 Correlations and changes in cfPWV adjusted for changes in blood pressure and heart rate

Measure	Pre-exercise (r)	24-h post-exercise (r)	Change (r)	Adjusted change in cfPWV ($m s^{-1}$) ^a	R ^{2a}
Systolic blood pressure (mm Hg)	0.544**	0.529**	0.227	0.172	5.2%
Male	0.199	0.216	0.078	—	—
Female	0.680*	0.837**	0.525*	—	—
Diastolic blood pressure (mm Hg)	0.329	0.316	0.203	0.176	4.1%
Male	0.427	0.462	0.213	—	—
Female	0.296	0.190	0.178	—	—
Heart rate (bpm)	0.276	0.248	0.313	0.204*	9.8%
Male	0.386	0.170	0.541*	—	—
Female	0.137	0.292	-0.068	—	—

Abbreviations: bpm, beats per minute; cfPWV, carotid–femoral pulse wave velocity; DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure.

* $P<0.05$.

** $P<0.01$.

^aResults from separate linear regression models with change in cfPWV as the dependent variable and change in SBP, DBP or HR as the independent variable.

our results cannot be generalized to populations other than young, healthy adults.

In conclusion, arterial stiffness decreased 24 h following a moderate-to-vigorous bout of exercise in young, healthy adults. These findings are important to consider for standardization of cPWV testing protocols and, potentially, realizing a maximal benefit of exercise on cardiovascular health through regular rather than sporadic exercise bouts. Future studies should investigate the effects of exercise on cPWV in other populations (for example, older, unfit or obese, with varying intensities or durations of exercise) and evaluate the combined contribution of acute and chronic effects of exercise on cPWV.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

We thank Megan Frame and Jessica Harvey for their work on this project.

- Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med* 2006; **3**: e442.
- Seals DR, DeSouza CA, Donato AJ, Tanaka H. Habitual exercise and arterial aging. *J Appl Physiol* 2008; **105**: 1323–1332.
- Cardoso CG Jr, Gomides RS, Queiroz ACC, Pinto LG, da Silveira Lobo F, Tinucci T, Mion D Jr, de Moraes Forjaz CL. Acute and chronic effects of aerobic and resistance exercise on ambulatory blood pressure. *Clinics* 2010; **65**: 317–325.
- Madden K, Lockhart C, Cuff D, Potter T, Meneilly G. Aerobic training-induced improvements in arterial stiffness are not sustained in older adults with multiple cardiovascular risk factors. *J Hum Hypertens* 2012; **27**: 335–339.
- Townsend RR, Wilkinson IB, Schiffrin EL, Avolio AP, Chirinos JA, Cockcroft JR, Heffernan KS, Lakatta EG, McEniery CM, Mitchell GF, Najjar SS, Nichols WW, Urbina EM, Weber T. Recommendations for improving and standardizing vascular research on arterial stiffness: a scientific statement from the American Heart Association. *Hypertension* 2015; **66**: 698–722.
- Sharman JE, La Gerche A, Coombes JS. Exercise and cardiovascular risk in patients with hypertension. *Am J Hypertens* 2015; **28**: 147–158.
- Taylor-Tolbert NS, Dengel DR, Brown MD, McCole SD, Pratley RE, Ferrell RE, Hagberg JM. Ambulatory blood pressure after acute exercise in older men with essential hypertension. *Am J Hypertens* 2000; **13**: 44–51.
- Van Bortel LM, Laurent S, Boutouyrie P, Chowienczyk P, Cruickshank JK, De Backer T, Filipovsky J, Huybrechts S, Mattace-Raso FU, Protogerou AD, Schillaci G, Segers P, Vermeersch S, Weber T. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J Hypertens* 2012; **30**: 445–448.
- Hu M, Yan H, Ranadive SM, Agiovlasis S, Fahs CA, Atiq M, Atique N, Fernhall B. Arterial stiffness response to exercise in persons with and without Down syndrome. *Res Dev Disabil* 2013; **34**: 3139–3147.
- Thijssen DH, Black MA, Pyke KE, Padilla J, Atkinson G, Harris RA, Parker B, Widlansky ME, Tschakovsky ME, Green DJ. Assessment of flow-mediated dilation in humans: a methodological and physiological guideline. *Am J Physiol Heart Circ Physiol* 2011; **300**: H2–H12.
- Miura H. Arterial function during various acute exercises. *J Sports Med Phys Fit* 2012; **1**: 605–610.
- Mattace-Raso F, Hofman A, Verwoert GC, Wittemana JC, Wilkinson I, Cockcroft J, McEniery C, Yasmin Laurent S, Boutouyrie P, Bozec E, Hansen TW, Torp-Pedersen C, Ibsen H, Jeppesen J, Vermeersch SJ, Rietzschel E, De Buyzere M, Gillebert TC, Van Bortel L, Segers P, Vlachopoulos C, Aznaouridis C, Stefanadis C, Benetos A, Labat C, Lacolley P, Stehouwer C, Nijpels G, Dekker JM, Stehouwer C, Ferreira I, Twisk JW, Czerlichow S, Galan P, Hercberg S, Pannier B, Guérin A, London G, Cruickshank JK, Anderson SG, Paini A, Agabiti Rosei E, Muiesan ML, Salvetti M, Filipovsky J, Seidlerova J, Dolejsova M. Determinants of pulse wave velocity in healthy people and in the presence of cardiovascular risk factors: 'establishing normal and reference values'. *Eur Heart J* 2010; **31**: 2338–2350.
- Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, Pannier B, Vlachopoulos C, Wilkinson I, Struijker-Boudier H. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J* 2006; **27**: 2588–2605.
- Aizawa K, Petrella RJ. Acute and chronic impact of dynamic exercise on arterial stiffness in older hypertensives. *Open Cardiovasc Med J* 2008; **2**: 3–8.
- Michaelides A, Soulis D, Antoniadou C, Antonopoulos AS, Miliou A, Ioakeimidis N, Chatzistamatiou E, Bakogiannis C, Marinou K, Liakos C, Stefanadis C. Exercise duration as a determinant of vascular function and antioxidant balance in patients with coronary artery disease. *Heart* 2011; **97**: 832–837.
- Nickel KJ, Acree LS, Gardner AW. Effects of a single bout of exercise on arterial compliance in older adults. *Angiology* 2011; **62**: 33–37.
- Kingwell BA, Berry KL, Cameron JD, Jennings GL, Dart AM. Arterial compliance increases after moderate-intensity cycling. *Am J Phys Heart Circ Phys* 1997; **273**: H2186–H2191.
- Hayward CS, Kelly RP. Gender-related differences in the central arterial pressure waveform. *J Am Coll Cardiol* 1997; **30**: 1863–1871.
- Pescatello LS, Arena R, Riebe D, Thompson PD (eds). *ACSM's Guidelines for Exercise Testing and Prescription*, 9th edn. Lippincott Williams & Wilkins: Philadelphia, PA, USA, 2013, 19–30.
- Weber T, Ammer M, Rammer M, Adji A, O'Rourke MF, Wassertheurer S, Rosenkranz S, Eber B. Noninvasive determination of carotid-femoral pulse wave velocity depends critically on assessment of travel distance: a comparison with invasive measurement. *J Hypertens* 2009; **27**: 1624–1630.
- Cohen J. *Statistical Power Analysis for the Behavioral Sciences*, 2nd edn. Academic press: New York, NY, USA, 1988, 20–27.
- Peng F, Pan H, Wang B, Lin J, Niu W. The impact of angiotensin receptor blockers on arterial stiffness: a meta-analysis. *Hypertens Res* 2015; **38**: 613–620.
- Seidlerová J, Filipovsky J, Mayer O, Wohlfahrt P, Cífková R. Positive effects of antihypertensive treatment on aortic stiffness in the general population. *Hypertens Res* 2014; **37**: 64–68.
- Wilkinson IB, MacCallum H, Flint L, Cockcroft JR, Newby DE, Webb DJ. The influence of heart rate on augmentation index and central arterial pressure in humans. *J Physiol* 2000; **525**: 263–270.
- Wang H, Zhang T, Zhu W, Wu H, Yan S. Acute effects of continuous and interval low-intensity exercise on arterial stiffness in healthy young men. *Eur J Appl Phys* 2014; **114**: 1385–1392.
- Doonan RJ, Mutter A, Egiziano G, Gomez Y-H, Daskalopoulou SS. Differences in arterial stiffness at rest and after acute exercise between young men and women. *Hypertens Res* 2012; **36**: 226–231.
- Naka KK, Tweddell AC, Parthimos D, Henderson A, Goodfellow J, Frenneaux MP. Arterial distensibility: acute changes following dynamic exercise in normal subjects. *Am J Phys Heart Circ Phys* 2003; **284**: H970–H978.
- Seo J-B, Chung W-Y, Kim S-H, Kim M-A, Zo J-H. Immediate impact of exercise on arterial stiffness in humans. *World J Cardiovasc Dis* 2013; **3**: 40–45.
- Sung J, Yang JH, Cho SJ, Hong SH, Huh EH, Park SW. The effects of short-duration exercise on arterial stiffness in patients with stable coronary artery disease. *J Korean Med Sci* 2009; **24**: 795–799.
- Collier S, Kanaley J, Carhart R, Frechette V, Tobin MM, Hall AK, Luckenbaugh AN, Fernhall B. Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow and blood pressure in pre-and stage-1 hypertensives. *J Hum Hypertens* 2008; **22**: 678–686.
- Currie DK, Thomas SG, Goodman JM. Effects of short-term endurance exercise training on vascular function in young males. *Eur J Appl Phys* 2009; **107**: 211–218.
- Donley DA, Fournier SB, Reger BL, DeVallance E, Bonner DE, Olfert IM, Frisbee JC, Chantler PD. Aerobic exercise training reduces arterial stiffness in metabolic syndrome. *J Appl Physiol* 2014; **116**: 1396–1404.
- Fantin F, Rossi A, Morgante S, Soave D, Bissoli L, Cazzadori M, Vivian ME, Valsecchi M, Zamboni M. Supervised walking groups to increase physical activity in elderly women with and without hypertension: effect on pulse wave velocity. *Hypertens Res* 2012; **35**: 988–993.
- Madden KM, Lockhart C, Cuff D, Potter TF, Meneilly GS. Short-term aerobic exercise reduces arterial stiffness in older adults with type 2 diabetes, hypertension, and hypercholesterolemia. *Diabetes Care* 2009; **32**: 1531–1535.
- Yokoyama H, Emoto M, Fujiwara S, Motoyama K, Morioka T, Koyama H, Shoji T, Inaba M, Nishizawa Y. Short-term aerobic exercise improves arterial stiffness in type 2 diabetes. *Diabetes Res Clin Pract* 2004; **65**: 85–93.
- Kawamoto R, Kohara K, Katoh T, Kusunoki T, Ohtsuka N, Abe M, Kumagi T, Miki T. Effect of weight loss on central systolic blood pressure in elderly community-dwelling persons. *Hypertens Res* 2014; **37**: 933–938.
- Belz GG. Elastic properties and Windkessel function of the human aorta. *Cardiovasc Drugs Ther* 1995; **9**: 73–83.
- Mota MR, Pardono E, Lima LC, Arsa G, Bottaro M, Campbell CS, Simões HG. Effects of treadmill running and resistance exercises on lowering blood pressure during the daily work of hypertensive subjects. *J Strength Cond Res* 2009; **23**: 2331–2338.
- Ferreira I, Twisk J, Stehouwer C, van Mechelen W, Kemper H. Longitudinal changes in VO2max: associations with carotid IMT and arterial stiffness. *Med Sci Sports Exerc* 2003; **35**: 1670–1678.
- Radak Z, Chung HY, Goto S. Systemic adaptation to oxidative challenge induced by regular exercise. *Free Radic Biol Med* 2008; **44**: 153–159.
- Mitchell GF, Parise H, Benjamin EJ, Larson MG, Keyes MJ, Vita JA, Vasani RS, Levy D. Changes in arterial stiffness and wave reflection with advancing age in healthy men and women the Framingham Heart Study. *Hypertension* 2004; **43**: 1239–1245.
- Pepine CJ, Nichols WW, Pauly DF. Estrogen and different aspects of vascular disease in women and men. *Circ Res* 2006; **99**: 459–461.
- Van Bortel LM, Duprez D, Starmans-Kool MJ, Safar ME, Giannattasio C, Cockcroft J, Kaiser DR, Thuillez C. Clinical applications of arterial stiffness. Task Force III: recommendations for user procedures. *Am J Hypertens* 2002; **15**: 445–452.
- Boutouyrie P, Lacolley P, Girerd X, Beck L, Safar M, Laurent S. Sympathetic activation decreases medium-sized arterial compliance in humans. *Am J Physiol Heart Circ Physiol* 1994; **267**: H1368–H1376.