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ORIGINAL ARTICLE

A systematic review on the effect of acute aerobic exercise on arterial stiffness reveals a differential response in the upper and lower arterial segments

Andrew F Mutter[1], Alexandra B Cooke[1], Olivier Saleh[2], Yessica-Haydee Gomez[2] and Stella S Daskalopoulou[1,2]

The objective of this systematic review was to provide insight into the controversy that still abounds as to the impact of acute aerobic exercise on immediate changes in arterial stiffness. Electronic databases were searched to identify articles assessing the effects of acute aerobic exercise on parameters of arterial stiffness. Eligible studies included arterial stiffness measurements before and after acute aerobic exercise in healthy human subjects. Forty-three studies were included. The effect of acute aerobic exercise on arterial stiffness was found to be dependent on the anatomical segment assessed, and on the timing of the measurement post-exercise. Arterial stiffness of the *central* and *upper body peripheral arterial segments* was found to be increased relative to resting values immediately post-exercise (0–5 min), whereas, thereafter (>5 min), decreased to a level at or below resting values. In the *lower limbs*, proximal to the primary working muscles, arterial stiffness decreased immediately post-exercise (0–5 min), which persisted into the recovery period post-exercise (>5 min). This systematic review reveals a differential response to acute exercise in the lower and upper/central arterial segments in healthy adult subjects. We further showed that the effect of acute aerobic exercise on arterial stiffness is dependent on the timing of the measurements post-exercise. Therefore, when assessing the overall impact of exercise on arterial stiffness, it is important to consider the arterial segment being analyzed and measurement time point, as failure to contextualize the measurement can lead to conflicting results and misleading clinical inferences.
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INTRODUCTION

Physical activity and exercise have a crucial role in the maintenance of healthy living. With ~30% of adults being insufficiently active, reduced physical activity is a contributing factor to >3.2 million deaths globally each year.[1] Regular exercise has notably been shown to reduce the incidence of cardiovascular (CV) complications, such as obesity, diabetes, hypertension and atherosclerosis, in both men and women.[2]

Arterial stiffness is widely used as a marker to assess the severity and progression of CV diseases.[3] Many techniques have been developed to measure arterial stiffness, including echo tracking, Doppler ultrasound and applanation tonometry.[4] The latter has the ability to quantify the velocity of generated pulse waves between arterial segments (pulse wave velocity, or PWV) and the timing of the reflected pulse wave by peripheral sites, augmenting the forward travelling wave at the aorta (augmentation index or AIx).[3] It has been previously shown that increased PWV and AIx are directly and independently associated with an increased risk for CV complications and events.[5,6] More recently, the carotid–ankle vascular index (CAVI) has emerged as a blood pressure-independent measure of arterial stiffness in the aorta, femoral artery and tibial artery.[7,8]

Under conditions of acute physical stress, such as aerobic exercise, segments of the arterial tree are differentially affected and respond/recover in a time-dependent manner following physical stress cessation. The overall impact of resistance-type exercises on the modulation of arterial stiffness has been well characterized,[9] as have the effects of chronic physical activity.[10] However, controversy still abounds as to the impact of acute aerobic exercise on immediate changes in arterial stiffness.

To our knowledge, this is the first systematic review that aims to synthesize evidence pertaining to acute changes in arterial stiffness shortly following aerobic exercise.

METHODS

Data sources, search strategy and study selection

The present systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Supplementary Table 1).[11] Two researchers independently conducted the

[1]Division of Experimental Medicine, Department of Medicine, Faculty of Medicine, McGill University, Montreal, QC, Canada and [2]Division of Internal Medicine, Department of Medicine, Faculty of Medicine, McGill University Health Centre, Montreal, QC, Canada
Correspondence: Dr SS Daskalopoulou, Department of Medicine, FRSQ Chercheur-Boursier Clinicien, McGill University, Montreal, Quebec, Canada H3G 1A4.
E-mail: stella.daskalopoulou@mcgill.ca
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literature search using the MEDline, EMBASE, Cochrane Library, Sport Discus and Web of Science electronic databases. Relevant articles were extracted using standardized search terms. The strategy implemented for the MEDline search is illustrated in Figure 1, and was modified in accordance with the indexing systems of each aforementioned database.

Additionally, manual searches of reference lists and 'related article' links to the articles selected for review were screened for eligibility. Records were limited to full-text articles involving human subjects published between January 1985 and January 2016, written in the English language and without restrictions in study design. Included in this review are all relevant clinical studies assessing the effect of acute aerobic exercise on parameters of arterial stiffness, in which measurements of arterial stiffness were performed before and after acute aerobic exercise in healthy human subjects. In the screening process, articles were carefully examined to ensure that participants were free of any CV disease, or CV risk factors, and were not taking cardioprotective medications. When studies incorporated weight-bearing activities during aerobic exercise, or involved cold or heat exposure, only the control condition was considered (if applicable) to avoid any confounding stress to the arterial system. Furthermore, for comparability reasons, aerobic exercise that exceeded 5 h was not included. The study selection procedure is illustrated in Figure 2.

1. (arterial stiffness or pulse wave velocity or PWV or aortic stiffness or wave reflection or pulse wave analysis or augmentation index or augmentation pressure or arterial wall distensibility or arterial compliance or elastance or hemodynamic* or haemodynamic* or crPWV or cfPWV or baPWV or PWA or Aix or central blood pressure or stiffness index).mp.
2. Hemodynamics/ or Aorta, Abdominal/ or Aorta, Thoracic/ or capillary resistance/ or elasticity/ or pulse transit time/ or blood flow velocity/ or vascular resistance/ or vascular capacitance/ or pulsatile flow/ or brachial artery/ or carotid arteries/ or femoral artery/ or radial artery/
3. (exer* or physical activ* or treadmill* or biking or run* or bicycl* or cycl* or jog* or fitness test* or aerobic exercise* or ergomet* or physical stress).mp.
4. exercise/ or running/ or physical exertion/ or physical endurance/ or jogging/
5. 1 and 2
6. 3 and 4
7. 5 and 6
8. limit 7 to (English language and Humans and yr="1985 -Current" and Full text and Medline)

Figure 1 MEDline search strategy.

Information was extracted independently by two researchers from each included study on: (1) characteristics of study participants (age, sex, body mass index (BMI), and baseline physical activity and health when available); (2) study type, inclusion criteria and population size; (3) imposed physical activity modality and (4) outcome measures of arterial stiffness. When values were not reported in the text, or included only in the form of graphs, authors were contacted and asked to provide missing data or for clarifications. When a response could not be obtained, values were estimated with precision from the graphs.

Risk of bias in the reporting of this review was considered. Article selection bias was accounted for through the use of two independent reviewers, and a third when consensus was not met. Strict inclusion/exclusion criteria were enforced on each article considered, as aforementioned. Although several tools may be applied, no clear 'gold-standard' quality assessment method in the review of observational studies exist and thus misclassification of study quality may occur. One must subjectively assess scale criteria for a given study, and there are underlying problems associated with this strategy.[12] Subject selection bias was, however, inherent within the included studies, as the research groups enrolled only healthy individuals, free from CV disease or risk factors. However, this was by design as we aimed to compile the evidence pertaining to the normal physiological arterial response to acute exercise.

RESULTS

A total of 43 studies were identified, confirmed by all authors, and included in this review.[13–54] Table 1 contains subject characteristics of the selected studies. All included studies were observational in nature and included only healthy individuals. Altogether, these studies evaluated 1089 adults (811 men and 253 women). Specifically, 27 studies enrolled only men,[13–38] 1 study enrolled only women,[39] 14 enrolled both men and women[40–54] and sex distribution was not

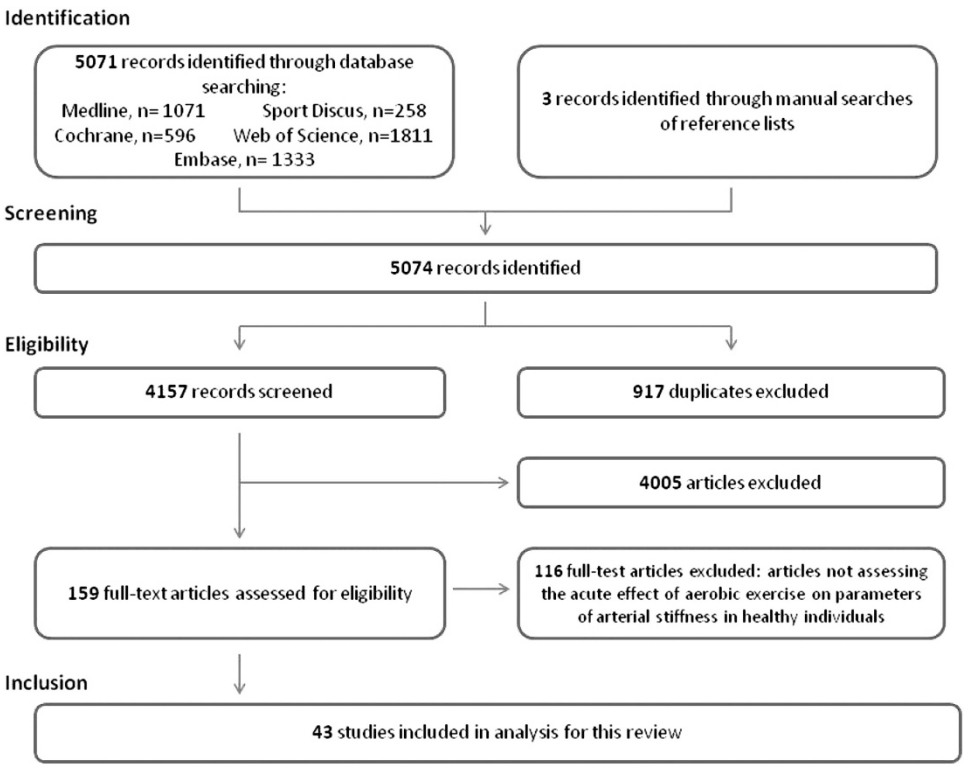

Figure 2 Implemented search procedure. A full color version of this figure is available at the *Hypertension Research* journal online.

Table 1 Subject characteristics of included studies

Study	*Subjects*	n	*Sex (men/women)*	*Age (years)*	*BMI (kg m^{-1})*
Akawaza *et al.*[39]	Healthy older women	9	0/9	61 ± 2[&]	H: 154 ± 1 cm[&] W: 52 ± 2 kg[&]
Attinà *et al.*[28]	Healthy middle-aged men (aged 27–66 years)	15	15/0	45 ± 3[&]	25.5 ± 0.7[&]
Babcock *et al.*[49]	Healthy young adults	55	12/43	22 ± 5	24.5 ± 2.7
Boutcher *et al.*[33]	Healthy young men (family history of HTN)	20	20/0	22.2 ± 0.5[&]	21.9 ± 0.7[&]
	Healthy young men (no family history of HTN)	20	20/0	21.3 ± 0.6[&]	22.2 ± 0.5[&]
Burr *et al.*[50]	Healthy young adults	13	9/4	25 ± 6	H: 175 ± 8 cm W: 74.6 ± 16 kg
Campbell *et al.*[34]	Healthy young men	10	10/0	31 ± 5	24 ± 3
Casey *et al.*[13]	Healthy young men (age 20–29 years)	14	14/0	24 ± 1	24.2 ± 0.5
	Healthy middle-aged men (age 40–59 years)	16	16/0	48 ± 2[&]	26.1 ± 0.7[&]
Chandrakumar *et al.*[35]	Healthy young men	15	15/0	20.2 ± 0.2[&]	21.8 ± 0.4[&]
Collier *et al.*[36]	Healthy young men	10	10/0	24.9 ± 0.9[&]	H = 174.8 ± 1.5 cm[&] W: 76.8 ± 2.4 kg[&]
Dischl *et al.*[14]	Healthy endurance-trained men	11	11/0	33 ± 5	22.2 ± 1.8
Doonan *et al.*[15]	Healthy young men	53	53/0	23 ± 5.4	22.3 ± 2.2
Doonan *et al.*[45]	Healthy young adults	122	67/55	Men: 24.4 ± 6.2 Women: 23.7 ± 4.8	Men: 22.8 ± 2.7 Women: 21.7 ± 2.1
Gkaliagkousi *et al.*[51]	Healthy middle-aged adults	15	9/6	39.3 ± 5.6	23.3 ± 2.8
Hanssen *et al.*[29]	Healthy young men	21	21/0	19–31 years	23 ± 1
Heffernan *et al.*[24]	Healthy young African-American men	12	12/0	22 ± 1[&]	27.3 ± 1.2[&]
	Healthy young Caucasian men	12	12/0	22 ± 1[&]	28.7 ± 1.0[&]
Heffernan *et al.*[24]	Healthy young resistance-trained men	15	15/0	21.9 ± 0.6[&]	H = 177.9 ± 1.6 cm[&] W = 92.9 ± 3.3 kg[&]
	Age-matched non-resistance-trained men (controls)	15	15/0	22.7 ± 0.9[&]	H = 179.1 ± 1.4 cm[&] W = 81.6 ± 3.4 kg[&]
Heffernan *et al.*[16]	Healthy young men	13	13/0	25 ± 0.7[&]	H = 174.7 ± 1.3 cm[&] W = 74.5 ± 2.2 kg[&]
Hu *et al.*[46]	Healthy young adults	15	10/5	26.2 ± 0.6[&]	23.9 ± 0.9[&]
Hull *et al.*[54]	Healthy young adults	25	18/7	29.3 ± 5.8	23.1 ± 1.8
Kingwell *et al.*[17]	Healthy young men	12	12/0	24 ± 6	22.9 ± 0.9[&]
Lane *et al.*[52]	Healthy young adults	62	31/31	24.7 ± 0.4[&]	25.6 ± 4.2[&]
Liu *et al.*[32]	Healthy young college basketball athletes	10	10/0	23.9 ± 1.2	22.8 ± 2.6
	Age-matched healthy sedentary adults	9	9/0	24.2 ± 3.0	21.6 ± 2.5
Lydakis *et al.*[40]	Healthy young adults	15	7/8	26.6 ± 3.6[&]	24.3 ± 3.1[&]
McClean *et al.*[18]	Healthy young men	8	8/0	22.9 ± 2.8	25.1 ± 3.0
Milatz *et al.*[30]	Healthy young men	32	32/0	33.7 ± 8	24 ± 2.5
Munir *et al.*[48]	Healthy young adults	25	NR	19–33 years	NR
Naka *et al.*[41]	Healthy young adults	50	46/4	30.7 ± 5.8	24.5 ± 3.5
Nickel *et al.*[42]	Healthy older adults	32	14/18	71 ± 7	24.7 ± 2.8
Nieman *et al.*[47]	Healthy endurance-trained young adults	16	8/8	Men: 39.3 ± 2.3[&] Women: 35.8 ± 2.8[&]	Men: H = 180 ± 3.0 cm[&] W = 80.3 ± 4.5 kg[&] Women: H: 163 ± 3.0 cm[&] W = 57.6 ± 2.9 kg[&]
Nottin *et al.*[43]	Healthy young adults	12	10/2	22.0 ± 3.1	H = 177.1 ± 9.7 cm W = 71.2 ± 11.4 kg
Payne *et al.*[37]	Healthy young men	30	30/0	22.3 ± 2.4	22.9 ± 2.6
Rakobowchuk *et al.*[19]	Healthy young men	9	9/0	20.1 ± 1.2	H = 181 ± 7 cm W = 77.5 ± 6.8 kg
Ranadive *et al.*[44]	Healthy young adults	15	9/6	25 ± 5	22.9 ± 3.4
Ribeiro *et al.*[25]	Healthy young men	14	14/0	31.0 ± 1.0[&]	26.6 ± 0.9[&]
Sharman *et al.*[20]	Healthy young men	12	12/0	31 ± 1[&]	24.3 ± .9[&]
Sharman *et al.*[38]	Healthy young men	12	12/0	29 ± 1[&]	23.8 ± 0.9[&]
Sugawara *et al.*[21]	Healthy young men	18	18/0	24 ± 1[&]	H = 171.5 ± 1.3 cm W = 65.7 ± 2.1 kg[&]
Sugawara *et al.*[22]	Healthy young men	9	9/0	25 ± 1[&]	23.1 ± 0.5[&]
Tordi *et al.*[23]	Healthy young men	11	11/0	22.5 ± 0.7[&]	H = 177.7 ± 1.1 cm[&] W = 70.5 ± 2.4 kg[&]
Wang *et al.*[26]	Healthy young men	15	15/0	21.2 ± 0.4[&]	22.7 ± 0.5[&]

Table 1 (Continued)

Study	*Subjects*	n	*Sex (men/women)*	*Age (years)*	*BMI (kg m^{-1})*
Yan *et al.*[53]	Healthy young AA and CA adults	100	AA 28/24 CA 25/23	AA men: 25 ± 1[&] CA men: 25 ± 1[&] AA women: 24 ± 1[&] CA women: 26 ± 1[&]	AA men: 26.7 ± 1.1[&] CA men: 25.7 ± 1.1[&] AA women: 28.9 ± 1.1[&] CA women: 23.1 ± 1.2[&]
Zheng *et al.*[27]	Healthy young men	16	16/0	22.0 ± 0.3[&]	23.0 ± 0.5[&]
Zhou *et al.*[31]	Healthy young men	19	19/0	24.7 ± 0.3[&]	21.9 ± 0.3[&]

Abbreviations: AA, African American; BMI, body mass index; CA, Caucasian American; H, height; HTN, hypertension; NR, not reported; W, weight.
Values are presented as mean plus/minus s.d., or as mean plus/minus s.e. (indicated by an ampersand).

reported for 1 study.[48] All studies enrolled participants between the ages of 20 and 35 years, except Nickel *et al.*,[42] Gkaliagkousi *et al.*,[51] Nieman *et al.*,[47] Akazawa *et al.*[39] and Attinà *et al.*[28] who enrolled only older healthy adults. Mean participant BMI in all studies was below 30 kg m^{-1}. Baseline physical activity of participants was not reported for the majority of included studies; however, when reported, this information is recorded in Tables 2a, 2b and 3 (column Inclusion criteria).

Pertinent results for the selected papers are contained in Tables 2 and 3 and Supplementary Table 2. All included studies used a form of cycling, running or leg extensor exercise as their primary aerobic physical stressor with the exception of Ranadive *et al.*[44] who incorporated graded arm-cycling protocols as the acute physical stressor. As noted in Tables 2a, 2b and 3, exercise intensity and duration varied among studies (column Exposure), ranging from 10 min of low-intensity walking[25] or 30 s of high-intensity exercise[32,49] to a 2 h marathon-paced run.[47] Differential effects of acute aerobic exercise, depending on the anatomical segment analyzed and the time of measurements post-exercise, were noted. Summarized findings were separated into time intervals post-exercise, those measured in the first 5 min (Tables 2a and 2b), and those measured >5 min post-exercise (Table 3).

Arterial stiffness measurements immediately post-exercise (0–5 min)

A total of 24 studies characterized changes in arterial stiffness in the first 5 min post-exercise.[13–15,18–23,25–27,29–32,34,37,38,40,41,45–49,51,54] Findings pertaining to changes in arterial stiffness during this time period are somewhat discordant. Some groups have demonstrated nonsignificant changes in arterial stiffness parameters in the first 5 min following acute aerobic exercise,[13,14,18–23,29,30,40,41,45–48,51,54] whereas significant but diverging results have also been reported.[14,15,19,21–23,26,27,31,32,34,37,40,41,46–49,54]

Included studies assessing *central and upper body peripheral* arterial segments in the first 5 min post-exercise demonstrated either significantly increased,[14,15,19,32,37,40,41,46–49,54] nonsignificantly increased,[13,19,23,30,34,47,48,51] or nonsignificantly decreased[13,14,18,40,46,47,54] arterial stiffness parameters (Table 2a). Specifically, Rakobowchuk *et al.*[19] demonstrated significantly increased heart-femoral PWV 2 min post-exercise, which remained elevated for 20 min, whereas Naka *et al.*[41] found brachial–radial PWV to be 35% higher 3 min post-exercise. Lydakis *et al.*[40] also found a decrease in Ti (the transit time of the pulse wave between the heart and reflection sites in the periphery) immediately following exercise to exhaustion. Similarly, our group (Doonan *et al.*[45]) demonstrated that carotid–femoral PWV (cfPWV) was significantly elevated at 2 and 5 min following exhaustive treadmill exercise.[15] Even after 10 min of lower intensity cycling exercise (60% maximum heart rate), Hull *et al.*[54] demonstrated significantly elevated cfPWV within the first 5 min post-exercise cessation. The majority of studies have reported a decrease in AIx post-exercise;[13,14,29,34,46] however, the AIx75 (AIx corrected for a heart rate of 75 beats per minute) was shown to be elevated in the first 5 min following exercise under multiple conditions.[14,15,46,47] Further, Tordi *et al.*[23] observed elevated carotid–radial PWV 2 min following both intermittent and constant aerobic exercise; however, this did not reach statistical significance. Hu *et al.*[46] have, however, recently demonstrated a significant increase in carotid β-stiffness index 3 min post-exercise in young adults, which was also noted by Liu *et al.*[32] after a series of four 30 s bouts of cycling exercise in basketball athletes. Similar trends in the cfPWV and AIx75 in response to exercise were observed by Doonan *et al.*,[45] even though authors chose to focus on investigating sex differences in response to acute aerobic exercise rather than comparing post-exercise values with resting baseline values. Although nonsignificant, Gkaliagkousi *et al.*[51] and Nieman *et al.*[47] also reported increased cfPWV immediately after high-intensity treadmill running. Similarly, Milatz *et al.*[30] demonstrated a 10% increase in aortic PWV compared with baseline after 60 min of cycling exercise, and Campbell *et al.*[34] observed elevated cfPWV after cycling exercise to exhaustion.

Conversely, all studies investigating arterial stiffness in *lower limb arterial segments*, at or near the primary exercising muscle groups, reported significantly[19,21–23,26,27,31,34] or nonsignificantly[23,41] *decreased* arterial stiffness in the first 5 min following exercise (Table 2b). Specifically, Sugawara *et al.*[21] found significant decreases in femoral–ankle PWV, and femoral–tibial PWV[22] in the exercising limb 2 min after light cycling, whereas nonsignificant differences were found in the non-exercising limbs. Campbell *et al.*[34] also demonstrated a significant decrease in femoral–tibial PWV immediately after maximal cycling exercise.[34] Femoral–dorsalis pedis PWV was shown to be significantly decreased 2 min following cycling exercises of varying intensities,[19] whereas Tordi *et al.*[23] also demonstrated decreases to carotid–dorsalis pedis PWV (a composite measure of central and peripheral arterial stiffness) as early as 4 min post-cycling interval exercise. Furthermore, three studies measuring CAVI, a vascular measure that incorporates the working lower limb vessels in addition to upper limb vessels (carotid–ankle), consistently observed a significantly lower CAVI after exercise termination.[26,27,31]

Arterial stiffness measurements >5 min post-exercise

As time from exercise cessation increases, there is a consistent trend towards a decrease in parameters of arterial stiffness towards or below resting values, independent of the arterial segment being analyzed (Table 3).[14–17,19,23,24,34,36,41,42,44,45,47,48] However, a small number of studies that only measured arterial stiffness starting 10–60 min post-exercise (i.e., not from time 0–10 min) noted no changes to various parameters of arterial stiffness following exercise.[13,16,24,28,33,35,39,45,47,50,51] Specifically, Naka *et al.*[41] demonstrated a 6% decrease *vs.* resting values

Table 2a Central and upper body peripheral arterial stiffness post-exercise (0–5 min)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
Babcock *et al.*[49]	Healthy young adults (18–40 years)	Non-smoker, inactive to recreationally active, no history of CV, respiratory or renal disease	β-Stiffness, elastic modulus, pulsatility index, PWV-β, wave intensity PWV, pulsatility index and resistance index at baseline and **immediately post-exercise** (carotid Doppler ultrasound)	High-intensity cycling (Wingate anaerobic test) for *30 s* (resistance 7% of body mass for non-athletes, 7.5% for athletes), followed by cool down of 2 min of light resistance cycling. Warm-up consisted of 5 min brisk treadmill walking	Overall increase in all parameters post-exercise β-Stiffness index Pre-exercise: 3.6 ± 0.9 Post-exercise: 4.0 ± 1.2 Elastic modulus (kPa) Pre-exercise: 43.5 ± 11.6 Post-exercise: 57.3 ± 17.5 PWV-β (m s^{-1}) Pre-exercise: 4.08 ± 0.5 Post-exercise: 4.4 ± 0.6 Wave intensity PWV (m s^{-1}) Pre-exercise: 3.1 ± 1.3 Post-exercise: 4.0 ± 1.9 Pulsatility index Pre-exercise: 1.9 ± 0.4 Post-exercise: 2.6 ± 0.5 Resistance index Pre-exercise: 0.8 ± 0.1 Post-exercise: 0.9 ± 0.0	Post *vs.* pre $P=0.010$ $P<0.001$ $P<0.001$ $P=0.008$ $P<0.001$ $P<0.001$
Casey *et al.*[13]	Healthy younger (20–29 years) and older (40–59 years) men	Non-smoker, sedentary or recreationally active, normotensive, non-obese, taking no medications and with no history of CV disease	AIx, AP and PPA before and **2, 4, 6, 8 and 10 min post-exercise** (applanation tonometry)	Cycling consecutively at steady state of 65% of age-predicted maximal HR (220—age) for 5 min	No differences in any measured variables during 10 min recovery period in both younger and older subjects (see Supplementary 2)	Post *vs.* pre NS for all
Campbell *et al.*[34]	Healthy young men	Non-smokers, normotensive, taking no medications and with no history or symptoms of cardiovascular, pulmonary, metabolic, neurological disease and no hypercholesterolemia	AIx, aortic PWV and femoral-to-tibial PWV before and at three time points post exercise; **0–5, 6–10 and 11–15 min** (applanation tonometry)	Upright cycle ergometer exercise until exhaustion. Start at 60 W (60 r.p.m. pedal frequency, at 3 min increased pedal frequency to 70 b.p.m., and then increased by 30 W every 3 min until 180 W. This was follower by 1 min stages of 20 W increments until volitional exhaustion	**AIx (%)** Pre-exercise: −9 ± 4 0–5 min post-exercise: −27 ± 2 **Aortic PWV** (cm s^{-1}) Pre-exercise: 5.4 ± 0.2 0–5 min post-exercise: 5.9 ± 0.3 *Values estimated from line graphs	Post *vs.* pre $P<0.05$ NS
Dischl *et al.*[14]	Healthy endurance-trained men	No inconspicuous medical history, and not taking any medication. Weekly aerobic activity of at least 2 h during the previous 12 weeks	Radial and central Aix, Aid and AId before and **5, 15, 25, 35 and 45 min post-exercise** (applanation tonometry)	15 min warm-up, followed by maximal intensity treadmill running test (Wasserman graded)	See Supplementary Table 3a	See Supplementary Table 3a
				15 min warm-up, followed by m run at pace of 4.5 min km^{-1}	See Supplementary Table 3b	See Supplementary Table 3b

Table 2a (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
Doonan *et al*.[15]	Healthy young men	Non-smoker, non-obese, normotensive, taking no medications affecting arterial stiffness, no history of CV, renal or inflammatory disease, or conditions affecting capacity to exercise	cfPWV, AIx75, before and **2, 5, 10 and 15 min post-exercise** (applanation tonometry)	Incremental treadmill exercise to volitional exhaustion using Bruce Protocol	cfPWV and AIx75 were significantly elevated immediately post-exercise cfPWV ($m\,s^{-1}$) Pre-exercise: 6.0 ± 0.7 2 min Post-exercise: 9.0 ± 2.0 5 min Post-exercise: 6.6 ± 1.3 AIx75 (%) Pre-exercise: −11.4 ± 10.0 5 min post-exercise: 12.9 ± 7.5	Post *vs*. pre $P<0.05$ $P<0.05$ $P<0.05$
Doonan *et al*.[45]	Healthy young adults	Non-smokers, non-obese, normotensive, taking no medications affecting arterial stiffness or contraceptive pills, and no history of CV disease, renal or inflammatory disease	cfPWV, AIx and AIx75 before and **2, 5, 10 and 15 min post-exercise** (applanation tonometry)	Incremental treadmill exercise to volitional exhaustion using Bruce Protocol	See Supplementary Table 4 No sex differences were found post-exercise for cfPWV or AIx75 cfPWV ($m\,s^{-1}$) Post–pre exercise change (men): 3.0 ± 1.7 Post–pre exercise change (women): 2.4 ± 1.1 AIx75 (%) Post–pre exercise change (men): 22.9 ± 11.4 Post–pre exercise change (women): 17.2 ± 10.1	 NS after adjustment (M *vs*. W) NS after adjustment (M *vs*. W)
Gkaliagkousi *et al*.[51]	Healthy middle-aged adults (aged 18–65 years)	Healthy, with no history of CVD or significant comorbidity. No antihypertensive or lipid-lowering medication, aspirin, or anti-inflammatory/anti-platelet medication	cfPWV pre-exercise, **immediately (1 min) post-exercise** (applanation tonometry)	Incremental treadmill exercise until exhaustion (Bruce Protocol)	cfPWV ($m\,s^{-1}$) Pre-exercise: 6.9 ± 0.25 1 min post-exercise: 7.6 ± 0.3 *Values estimated from line graphs	Post *vs*. pre NS
Hanssen *et al*.[29]	Healthy young men	Non-smoking, normotensive, BMI < 30 $kg\,m^{-1}$, no history of CV, pulmonary or metabolic disease	AIx and AIx75 before, and **5, 20, 35 and 50 min post-exercise**	HIIT: treadmill exercise (1% inclination) consisting of 10 min warm-up at 70% HR_{max}, followed by 4 × 4 min interval training at 90–95% HR_{max}, with 3 min recovery at 70% HR_{max} between intervals MCT: constant treadmill exercise (1% inclination) at 80% HR_{max} (varied duration to match O_2 uptake during HIIT) Subjects performed both protocols (crossover design) at least 72 h apart	HIIT AIx (%) Pre-exercise: −2 ± 8 5 min post-exercise: −1.3 ± 9 AIx75 (%) Pre-exercise: −10.8 ± 9 5 min post-exercise: 8.3 ± 9 MCT AIx (%) Pre-exercise: −2.6 ± 8 5 min post-exercise: −3.7 ± 8 AIx75 (%) Pre-exercise: −11.9 ± 8 5 min post-exercise: −4.4 ± 8	Post *vs*. pre associations not reported

Table 2a (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
Hu *et al.*[46]	Healthy young adults	Non-smoking, sedentary or moderately active, normotensive, with no history of CVD, respiratory, or metabolic disease, and no use of HR or BP altering, or anti-inflammatory medications	AP, AIx, AIx75 and β-stiffness index before and **3 min post-exercise** (applanation tonometry)	Incremental graded cycle ergometer test to exhaustion (started at 50 W and then increased of 30 W every 2 min until termination)	AP (mmHg) Pre-exercise: 2.8 ± 1.2 Post-exercise: − 0.8 ± 1.1 AIx (%) Pre-exercise: 7.9 ± 3.7 Post-exercise: − 0.8 ± 3.0 AIx75 (%) Pre-exercise: −3.2 ± 4.0 Post-exercise: 9.0 ± 2.8 β-Stiffness index Pre-exercise: 5.6 ± 1.1 Post-exercise: 8.9 ± 0.8 *Presented as mean ± s.e.	Post *vs.* pre NS NS *P*<0.01 *P*<0.001
Hull *et al.*[54]	Healthy young adults	Non-smokers, normotensive, sedentary or only recreationally active	AP, AIx75 and cfPWV before and **immediately post-exercise** (applanation tonometry)	10 min of stationary upright cycling exercise at 60% of their age-predicted maximum heart rate (cadence 50–60 r.p.m.)	AIx75 (%) Pre-exercise: −2.5 ± 11.6 Post-exercise: − 3.2 ± 11.6 AP (mmHg) Pre-exercise: 1.6 ± 3.0 Post-exercise: 1.0 ± 3.3 cfPWV (m s^{-1}) Pre-exercise: 5.7 ± 0.8 Post-exercise: 5.8 ± 0.7	Post *vs.* pre NS NS *P*<0.05
Liu *et al.*[32]	Healthy young basketball atheletes and age-matched sedentary young adults	Free of hypertensive, CV and metabolic diseases, and not taking any CV medications	β-Stiffness and pressure–strain elastic modulus (E_P) at baseline, and **immediately after** each exercise bout (carotid Doppler ultrasound)	Four 30 s bouts of leg cycling exercise, each 8 min apart (250 W workload with a cadence of 60–70 r.p.m.)	The elastic modulus (E_P) was significantly higher than baseline after each cycling bout in basketball athletes; however, no significant changes were noted in their sedentary counterparts (values not provided) β-Stiffness was only significantly higher than baseline after the third and fourth cycling bout in basketball athletes. No other significant changes were observed (values not provided)	
Lydakis *et al.*[40]	Healthy adults	Non-smokers, normotensive, no CV disease or medications	AIx and T_i before and **immediately post-exercise** (applanation tonometry)	Single-leg knee extensor exercise to exhaustion (10 W increase every 2 min for men, 5 W increase every 2 min for women)	AIx (%) Pre-exercise: 2 ± 1 Post-exercise: 1 ± 4 Ti (ms) Pre-exercise: 150 ± 2 Post-exercise: 134 ± 2 *Presented as mean ± s.e.	Post *vs.* pre NS *P*<0.001

Table 2a (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
McClean *et al.*[18]	Healthy young men	Non-smokers, no history of CV disease, taking no medications, antioxidants or lipid-lowering supplements	brPWV measured before and **immediately (<2 min) post-exercise** (sensor-based device)	Exercise on cycle ergometer at 60% of age-predicted HRmax for 60 min	brPWV ($m\,s^{-1}$) Controls: Pre-exercise: 6.48 ± 1.90 Post-exercise: 6.56 ± 1.60 Exercise group: Pre-exercise: 7.11 ± 1.80 Post-exercise: 6.47 ± 1.90	Post *vs.* pre NS NS
Milatz *et al.*[30]	Healthy young men	Non-smokers, not taking antihypertensive medications and free of acute or chronic diseases	aPWV measured pre-exercise and **1, 15, 30, 45 and 60 min post-exercise** (oscillometric method)	60 min of endurance cycling exercise on a cycle ergometer at 45% VO_{2max}	aPWV was 10% above baseline at 1 min post-exercise (NS) aPWV ($m\,s^{-1}$) Pre-exercise: 6.0 ± 0.7 1 min post-exercise: 6.6 ± 0.9 *Values estimated from bar graphs	Post *vs.* pre NS
Munir *et al.*[48]	Healthy young adults	Non-smokers, recreationally active, no history of CV disease and not taking any regular medications	Radial and digital pressure waveforms, PWV, brachial and femoral artery diameter and blood flows at rest and **1–3, 15, 30 and 60 min post-exercise** (applanation tonometry and servocontrolled finger pressure cuff)	Incremental exercise on bicycle ergometer. Start at 25 W, increase by 25 W every 2 min until a) exhaustion, or b) a maximum of 12 min total	cfPWV ($m\,s^{-1}$) Pre-exercise: 7.1 ± 0.2 Post-exercise (1–3 min): 7.4 ± 0.2 Central AI (%) Pre-exercise: 6.2 ± 2.0 Post-exercise (1–3 min): 3.3 ± 1.8 Peripheral systolic AI (%) Finger: Pre-exercise: 51 ± 3.8 Post-exercise (1–3 min):19 ± 4.0 Peripheral diastolic AI (%) Radial: Pre-exercise: 37 ± 1.8 Post-exercise (1–3 min): 11 ± 1.5 Finger: Pre-exercise: 37 ± 1.7 Post-exercise (1–3 min): 8.5 ± 1.4 *Presented as mean ± s.e.	Post *vs.* pre NS NS $P<0.001$ $P<0.001$ $P<0.001$
Naka *et al.*[41]	Healthy adults	Non-smokers, non-obese, sedentary, no CV complications and taking no medications	brPWV before and at **every min post-exercise for 60 min** (oscillometry)	Maximal symptom-limited treadmill exercise using Bruce Protocol (until RER < 1.1)	brPWV ($m\,s^{-1}$) Pre-exercise: 7.3 ± 0.9 brPWV was 35% above baseline at 3 min post (values not provided)	Post *vs.* pre $P<0.05$

Table 2a (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
Nieman *et al*.[46]	Healthy young adults	Non-smoking, active normotensive individuals with a history of regular participation in competitive running races capable of running for 2 h (marathon pace). No history heart disease, cancer, diabetes, arthritis, stroke or peripheral artery disease	AIx and cfPWV **mmediately and 30, 60, 120, 180, 240 and 300 post-exercise** (applanation tonometry)	Treadmill running for 2 h at 75% VO_{2max}	Men cfPWV NS differences immediately post-exercise *vs.* rest condition (data not shown) AIx75 Significantly increased immediately post-exercise *vs.* rest condition (data not reported) Women cfPWV NS differences immediately post-exercise *vs.* rest condition (data not shown) AIx75 NS differences immediately post-exercise *vs.* rest condition (data not shown)	 NS $P<0.05$ NS NS
Payne *et al*.[37]	Healthy young men	No history of cardiovascular or other significant illnesses, and not taking any regular medications	Radial AIx and carotid AIx before and **immediately and 10 min post-exercise** (applanation tonometry)	15 min cycling exercise at 65–70% of maximum heart rate	Radial AIx (%) Pre-exercise: 57 ± 14.6 Immediately post-exercise: 66 ± 13.2 Carotid AIx (%) Pre-exercise: 18.9 ± 12.2 Immediately post-exercise: 36.0 ± 14.8	Post *vs.* pre $P<0.01$ $P<0.001$
Rakobowchuk *et al*.[19]	Healthy young men	Non-smokers, normotensive, non-obese, taking no medications, no CV complications, recreationally active	Femoral artery distensibility at rest and **immediately post-exercise** (ultrasound) fdpPWV at rest and from **2–60 min continuously post-exercise** using *applanation tonometry* at femoral, and infrared blood volume detection device at dorsalis pedis artery	Three sessions: (1) Familiarization (2) Single sprint interval (1 Wingate anaerobic test) (3) Multiple sprint intervals (4 Wingate anaerobic tests)	No statistical difference in femoral artery distensibility before and after all exercise conditions Trend towards increased stiffness index at 2 min ($P=0.06$) (values not provided) Heart-femoral PWV increased significantly (2 min) and returned to baseline levels at 20 min into recovery for both exercise sessions (values not provided)	NS $P<0.001$
Ribeiro *et al*.[25]	Healthy young men	Non-smokers, non-athletes, with no history of hypertension, renal dysfunction, any CV or metabolic disease and not taking any medications	AP and AIx75 pre-exercise and **immediately post-exercise** (applanation tonometry)	10 min treadmill walking (speed of 5 km h^{-1})	AP (mmHg) Pre-exercise: 0.6 ± 1.3 Post-exercise: -0.1 ± 1.2 AIx75 (%) Pre-exercise: -5.2 ± 2.8 Post-exercise: -5.5 ± 2.1 *Presented as mean ± s.e.	Post *vs.* pre NS NS

Table 2a (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	*P-value*
Sharman *et al.*[20]	Healthy men	Non-smokers, no history of CV diseases, sedentary or recreationally active	PPA before, during and **2, 4, 6, 8 and 10 min post-exercise** (applanation tonometry)	Cycling on ergometer at 50 r.p.m., beginning at 50%, then increasing to 60, 70 and 80% of HRmax (4 min at each intensity)	No significant change in PPA during recovery (2–10 min) compared to baseline PPA Pre-exercise: 1.57 ± 0.05 2 min post: 1.76 ± 0.05 4 min post: 1.73 ± 0.05 *Presented as mean ± s.e.	Post *vs.* pre NS NS
Sharman *et al.*[38]	Healthy young men	Non-smokers, normotensive, not taking medications, no history of hypertension, CVD, diabetes or hypercholesterolemia	AIx (%) pre-exercise and **2 and 10 min post-exercise** in a seated position (applanation tonometry)	Cycling on a stationary bicycle ergometer at 50 r.p.m. and 60% of their maximal heart rate for 10 min	AIx (%) Pre-exercise: 6 ± 3 2 min post-exercise: 0 ± 3	Post *vs.* pre associations not reported
Tordi *et al.*[23]	Healthy young men	Non-smokers, taking no medication, no CV, pulmonary or metabolic complications.	crPWV and cdpPWV before and **for 30 min (2, 4, 6, 8, 10, 12,14, 16, 20, 24 and 28 min)** after IE and CE (applanation tonometry)	IE: 30 min total (6 consecutive periods of: 4 min at 65% HRmax+1 min at 85% HRmax)	crPWV nonsignificantly elevated post-IE crPWV ($m\,s^{-1}$) Pre-exercise: 8.1 ± .5 2 min post-exercise: 8.8 ± 1.5 4 min post-exercise: 8.8 ± 1.4 *Presented as mean ± s.e.	Post *vs.* pre NS NS
				CE: 30 min total. Constant exercise at level equal to HR average of IE	crPWV nonsignificantly elevated post-CE crPWV ($m\,s^{-1}$) Pre-exercise: 8.7 ± 1.1 2 min post-exercise: 9.6 ± 1.7 4 min post-exercise: 8.9 ± 1.1 *Presented as mean ± s.e.	Post *vs. p*re NS NS

Abbreviations: AI/AIx, augmentation index; AIx75, augmentation index adjusted to heart rate of 75 b.p.m.; AP, augmentation pressure; BMI, body mass index; b.p.m., beats per minute; brPWV, brachial–radial PWV; CE, continuous exercise; cfPWV, carotid–femoral PWV; cdpPWV, carotid–dorsalis pedis PWV; CV cardiovascular; CVD, cardiovascular disease; fdpPWV, femoral–dorsalis pedis PWV; HIIT, high-intensity interval training; HR, heart rate; HRR, heart rate reserve; HRmax, maximum heart rate; IE, interval exercise; maPWV, midthigh–ankle PWV; NS, nonsignificant; PPA, pulse pressure amplification; PWV, pulse wave velocity; RER, respiratory exchange ratio; r.p.m., revolution per minute; Ti, transit time of the reflected wave; VO_{2max}, maximal oxygen

Table 2b Lower body peripheral arterial stiffness post-exercise (0–5 min)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
Campbell *et al.*[34]	Healthy young men	Non-smokers, normotensive, taking no medications, and with no history or symptoms of cardiovascular, pulmonary, metabolic, neurological disease and no hypercholesterolemia	AIx, aortic PWV and femoral-to-tibial PWV before and at 3 time points post-exercise; **0–5, 6–10 and 11–15 min** (applanation tonometry)	Upright cycle ergometer exercise until exhaustion. Start at 60 W (60 r.p.m. pedal frequency, at 3 min increased pedal frequency to 70 b.p.m., and then increased by 30 W every 3 min until 180 W. This was followed by 1 min stages of 20 W increments until volitional exhaustion	**Femoral-to-tibial PWV** Pre-exercise: 7.2 ± 0.3 0–5 min post-exercise: 6.3 ± 0.3 *Values estimated from line graphs	Post *vs.* pre $P<0.05$
Naka *et al.*[41]	Healthy adults	Non-smokers, non-obese, sedentary, taking no medications and no CV complications	brPWV, and maPWV before and at **every min post-exercise for 60 min** (oscillometry)	Maximal symptom-limited treadmill exercise using Bruce Protocol (until RER < 1.1)	maPWV (m s^{-1}) Pre-exercise: 8.0 ± 1 maPWV did not differ from baseline at 3 min (values not provided)	NS
Rakobowchuk *et al.*[19]	Healthy young men	Non-smokers, recreationally active, taking no medications and no CV complications	Femoral artery distensibility (by ultrasound) at rest and **immediately post-exercise** fdpPWV at rest and from **2–60 min continuously post-exercise** using applanation tonometry at femoral, and infrared blood volume detection device at dorsalis pedis artery	Three sessions: (1) Familiarization (2) Single sprint interval (1 Wingate anaerobic test) (3) Multiple sprint interval (4 Wingate anaerobic tests)	fdpPWV decreased significantly 2 min post-exercise and returned to baseline at 44 min into recovery for all exercise conditions (values not provided)	$P<0.001$
Sugawara *et al.*[21]	Healthy young males	Non-smokers, sedentary or recreationally active and taking no regular medications	faPWV pre-exercise and **2 min post-exercise** (applanation tonometry)	Single-leg (left) cycling in supine position for 5 min at 20 or 30 W	faPWV (m s^{-1}) Left side (active) Pre-exercise: 9.1 ± 0.3 Post-exercise:8.4 ± 0.3 Right side (inactive) Pre-exercise: 9.0 ± 0.2 Post-exercise: 9.2 ± 0.2 Left leg *vs.* right Leg post-exercise *Presented as mean ± s.e.	Post *vs.* pre $P<0.05$ NS $P<0.05$
Sugawara *et al.*[22]	Healthy young men	Non-smokers, normotensive, non-obese, sedentary or recreationally active, taking no medications and free from overt chronic diseases	fptPWV in both legs (exercising and non-exercising limbs) at rest and **2 min post-exercise** (applanation tonometry)	5 min of single-leg (left) cycling at 30 W on ergometer	fptPWV (mm s^{-1}) Exercising limb: Pre-exercise: 958 ± 23 Post-exercise: 876 ± 25 Non-exercising limb: Pre-exercise: 937 ± 23 Post-exercise: 936 ± 19	Post *vs.* pre $P<0.05$ NS
Tordi *et al.*[23]	Healthy young men	Non-smokers, taking no medication and no CV, pulmonary or metabolic complications	crPWV and cdpPWV before and for 2–30 min **(2, 4, 6, 8, 10, 12, 14, 16, 20, 24 and 28 min)** after IE and CE (applanation tonometry)	IE: 30 min total (6 consecutive periods of: 4 min at 65% HRmax+1 min at 85% HRmax)	After IE, cdpPWV values at 4–28 min post-exercise significantly lower than baseline cdpPWV (m s^{-1}) Pre-exercise: 8.6 ± 0.5 2 min post-exercise: 8.2 ± 0.41 4 min post-exercise: 7.7 ± 0.5 *Presented as mean ± s.e.	Post *vs.* pre NS P < 0.05

Table 2b (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
				CE: 30 min total. Constant exercise at level equal to HR average of IE	cdpPWV was not significantly decreased after CE cdpPWV ($m\,s^{-1}$) Pre-exercise: 8.7 ± 0.8 2 min post-exercise: 8.5 ± 0.8 4 min post-exercise: 8.5 ± 0.8 *Presented as mean ± s.e.	Post *vs.* pre NS NS
Wang *et al.* [26]	Healthy young men	Non-smoking, active, normotensive with no history of CVD and no medications for diabetes, metabolic disease or CVD	CAVI before and **mmediately post-exercise** compared with non-exercise control session (crossover design) CAVI vascular screening system is used (combines oscillometry, phonocardiogram and electrocardiogram)	Cycle ergometer for 30 min at 25% HRR	Significantly lower values immediately after cycling compared with non-exercise control session CAVI (control): Baseline: 6.6 ± 0.1 30 min: 6.7 ± 0.1 CAVI (cycling): Baseline: 6.5 ± 0.1 Post-exercise: 5.5 ± 0.2 *Presented as mean ± s.e.	$P<0.001$ for cycling *vs.* control post-exercise values (no post *vs.* pre analysis within test)
Zheng *et al.*[27]	Healthy young men	Non-smoking, active, normotensive with no history of CVD and no medications for diabetes, metabolic disease or CVD	CAVI before and **immediately post-exercise** compared to non-exercise control session (crossover design) CAVI vascular screening system is used (combines oscillometry, phonocardiogram and electrocardiogram)	Cycle ergometer for 30 min at 50% HRR (r.p.m. of 60)	Significantly lower values immediately after cycling compared with non-exercise control session CAVI (control): Baseline: 6.8 ± 0.1 30 min: 6.9 ± 0.1 CAVI (cycling): Baseline: 6.8 ± 0.1 Post-exercise: 5.9 ± 0.1 *Presented as mean ± s.e.	$P<0.001$ for cycling *vs.* control post-exercise values (no post *vs.* pre analysis within test)
Zhou *et al.*[31]	Healthy young men	Non-smoking, moderately active (no varsity athletes), taking no medications and no history of CV, respiratory or endocrine disorders	CAVI before and **immediately post-exercise**	Cycle ergometer for 10 min at 50% HRR (performed in subset, $n=6$) Cycle ergometer for 30 min at 50% HRR (performed in all subjects, $n=19$)	CAVI Pre-exercise: 7.1 ± 0.3 Post-exercise: 5.8 ± 0.2 CAVI Pre-exercise: 6.9 ± 0.2 Post-exercise: 5.8 ± 0.1 *Presented as mean ± s.e.	<0.05 for post *vs.* pre $P<0.001$ for cycling *vs.* control post-exercise values

Abbreviations: AI/AIx, augmentation index; BMI, body mass index; b.p.m., beats per minute; brPWV, brachial–radial PWV; CAVI, carotid–ankle vascular index; cdpPWV, carotid–dorsalis pedis PWV; CE, continuous exercise; cfPWV, carotid–femoral PWV; crPWV, carotid–radial PWV; CV, cardiovascular; CVD, cardiovascular disease; faPWV, femoral–ankle PWV; fdpPWV, femoral–dorsalis pedis PWV; fptPWV, femoral-posterior tibial PWV; HR, heart rate; HRR, heart rate reserve; IE, interval exercise; maPWV, midthigh–ankle PWV; PWV, pulse wave velocity; RER, respiratory exchange ratio; RPM, revolution per minute; Ti, transit time of the reflection wave; VO_{2max}, maximal oxygen consumption.
All values are expressed as mean plus/minus s.d., unless otherwise indicated.

Table 3 Central, upper body and lower body peripheral arterial stiffness post-exercise (>5 min)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	*P-value*
Akawaza *et al.*[39]	Healthy older women	Non-smoker, non-obese, postmenopausal, sedentary, free of CVD and not taking any CV or hormone replacement medications	cfPWV pre-exercise and **30 and 60 min post-exercise** (applanation tonometry)	30 min of aerobic cycling exercise at subject's ventilatory threshold	cfPWV ($m\,s^{-1}$) Pre-exercise: 9.49±0.21 30 min post-exercise: 9.64±0.29 60 min post-exercise: 9.70±0.29 *Presented as mean±s.e.	Post *vs.* pre NS NS
Attinà *et al.*[28]	Healthy middle-aged men	Non-smokers or ex-smokers >1 years, non-obese, normotensive, no CV risk factors and not taking any prescribed medications	cfPWV pre-exercise and **10, 40 and 60 min post-exercise** (applanation tonometry)	Incremental aerobic cycling until volitional exhaustion (increase of 20 W min^{-1}) after 2 min of unloaded pedalling	cfPWV ($m\,s^{-1}$) Pre-exercise: 6.5±0.31 10 min post-exercise: 6.5±0.22 40 min post-exercise: 6.6±0.32 60 min post-exercise: 6.5±0.29 *Presented as mean±s.e.	Post *vs. pre* NS NS NS
Boutcher *et al.*[33]	Healthy young men	Normotensive, non-obese, physically active and involved in moderate-intensity exercise 1–3 times per week. *N*=20 had a family history of hypertension (one or two parents, or grandparents treated with hypertension medications)	AIx pre-exercise and **30-mins post exercise** (applanation tonometry)	Exercise on a stationary bike at 60% VO_{2max} for 20 min	AIx (%) HTN family hx **Pre-exercise:** −2.8±2.3 **Post-exercise:** −2.6±2.0 No HTN family hx **Pre-exercise:** −3.6±2.3 **Post-exercise:** −5.4±1.9	Post *vs.* pre NS NS
Burr *et al.*[50]	Healthy young adults	Non-smoker, normotensive, no previously diagnosed heart disease, peripheral vascular disease, diabetes, cancer, pulmonary disease, orthopaedic conditions or use of medications Recreationally active, free from injury and not pregnant	AIx75 and PWV pre-exercise and **15 min** post-exercise (applanation tonometry)	Downhill treadmill running at −12 °C grade at 60% VO_{2max} for 40 min (eccentrically accentuated aerobic exercise)	cfPWV ($m\,s^{-1}$) Pre-exercise: 5.1±0.6 Post-exercise: 5.3±0.6 AIx75 (%) Pre-exercise: −2.0±3.2 Post-exercise: −0.7±2.6	Post *vs.* pre NS NS
Casey *et al.*[13]	Healthy young a middle-aged men	Non-smoker, non-obese, normotensive, sedentary or recreationally active, taking no medications and free from overt CVD	AIx, AP, time of reflected wave and PPA pre-exercise and **2, 4, 6, 8 and 10 min post-exercise** of differing intensities (applanation tonometry)	Cycling at steady-state of 45, 55, and 65% of age-predicted maximal HR (220—age) for 5 min Completed in three separate trials	All measured hemodynamic variables at10 min of recovery were similar to each trial's respective resting values No group differences in any measured variables during 10 min recovery period	NS
Campbell *et al.*[34]	Healthy young men	Non-smokers, normotensive, taking no medications and with no history or symptoms of cardiovascular, pulmonary, metabolic, neurological disease and no hypercholesterolemia	AIx, aortic PWV and femoral-to-tibial PWV before and at three time points **0–5 min, 6–10 min, and 11–15 min post exercise** (applanation tonometry)	Upright cycle ergometer exercise until exhaustion. Start at 60 W (60 r.p.m. pedal frequency, at 3 min increased pedal frequency to 70 b.p.m. and then increased by 30 W every 3 min until 180 W. This was follower by 1 min stages of 20 W increments until volitional exhaustion	**AIx (%)** Pre-exercise: −9±4 6–10 min post-exercise: −27±2 11–15 min post-exercise: −23±3 **Aortic PWV** Pre-exercise: 5.4±0.2 6–10 min post-exercise: 5.6±0.3 11–15 min post-exercise: 5.9±0.3	Post *vs.* pre $P<0.05$ $P<0.05$ NS NS

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					Femoral-to-tibial PWV Pre-exercise: 7.2 ± 0.3 6–10 min post-exercise: 6.2 ± 0.3 11–15 min post-exercise: 6.5 ± 0.3 *Values estimated from line graphs	 $P<0.05$ $P<0.05$
Chandrakumar *et al.*[35]	Healthy young men	Normotensive, not taking medications, no history of chronic disease and active <90 min per week	cfPWV and crPWV pre-exercise and **60 min post-exercise** (applanation tonometry)	Exercise on a cycle ergometer for 30 min (60–80 r.p.m.) at 65% VO_{2max}, with 5 min warm-up and cool down	cfPWV (m s^{-1}) Pre-exercise: 5.29 ± 0.14 60 min post-exercise: 5.52 ± 0.17 crPWV (m s^{-1}) Pre-exercise: 6.48 ± 0.15 60 min post-exercise: 6.45 ± 0.21 AIx (%) Pre-exercise: −4.5 ± 2.5 10 min post-exercise: 1.5 ± 1.0 20 min post-exercise: 5.0 ± 0.5 30 min post-exercise: 4.0 ± 2.5 60 min post-exercise: 2.5 ± 1.5	Post *vs.* pre NS NS Post *vs.* pre associations not reported
Collier *et al.*[36]	Healthy young men	Non-smokers, non-obese, moderately active, not taking any medications and no history of CV or metabolic disease	cfPWV pre-exercise and **40 and 60 min post-exercise** (Doppler ultrasound)	30 min of upright cycle ergometry at 65% of peak oxygen	cfPWV (m s^{-1}) Delta cfPWV at 40 min: −0.45 ± 0.81 Delta cfPWV at 60 min: 0.42 ± 0.61	Post *vs.* pre associations not reported
Dischl *et al.*[14]	Healthy endurance-trained men	No inconspicuous medical history, and not taking any medication Weekly aerobic activity of at least 2 h during the previous 12 weeks	Radial and central Aix, Aid and AId pre-exercise and **5, 15, 25, 35 and 45 min post-exercise** termination (applanation tonometry)	15 min warm-up, followed by maximal intensity treadmill running test (Wasserman graded) 15 min warm-up, followed by 4000 m run at pace of 4.5 min km	See Supplementary Table 3a	See Supplementary Table 3a
Doonan *et al.*[15]	Healthy young men	Non-smoker, non-obese, normotensive, taking no medications affecting arterial stiffness, no history of CV, renal or inflammatory disease or conditions affecting capacity to exercise	cfPWV, AIx75 pre-exercise and **2, 5, 10 and 15 min post-exercise** (applanation tonometry)	Incremental treadmill exercise to volitional exhaustion using Bruce Protocol	cfPWV (m s^{-1}) Pre-exercise: 6.0 ± 0.7 10 min post-exercise: 6.1 ± 0.9 15 min post-exercise: 6.1 ± 0.9 AIx75 (%) Pre-exercise:-11.4 ± 10.0 10 min post-exercise: 6.6 ± 8.2* 15 min post-exercise: 3.2 ± 9.2*	Post *vs.* pre NS NS $P<0.05$ $P<0.05$
Doonan *et al.*[45]	Healthy young adults	Non-smokers, non-obese, normotensive, taking no medications affecting arterial stiffness or contraceptive pills and no history of CV disease, renal or inflammatory disease	cfPWV, AIx and AIx75 pre-exercise and **2, 5, 10, and 15 min post-exercise** (applanation tonometry)	Incremental treadmill exercise to volitional exhaustion using Bruce Protocol	cfPWV (m s^{-1}) See Supplementary Table 4 AIx75 (%) See Supplementary Table 4	See Supplementary Table 4

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	*P-value*
Gkaliagkousi *et al*.[51]	Healthy adults (aged 18–65 years)	Healthy, no history of CVD or significant comorbidilty and no antihypertensive or lipid-lowering medication, aspirin or anti-inflammatory or antiplatelet medication	cfPWV pre-exercise and at **10, 30 and 60 min post-exercise** (applanation tonometry)	Incremental treadmill exercise until exhaustion (Bruce Protocol)	cfPWV ($m\,s^{-1}$) Pre-exercise: 6.9 ± 0.3 10 min post-exercise: 6.6 ± 0.3 30 min post-exercise: 7.0 ± 0.3 60 min post-exercise: 6.7 ± 0.2 *Values estimated from line graphs	NS for all
Hanssen *et al*.[29]	Healthy young men	Non-smoker, normotensive, BMI < 30 $kg\,m^{-2}$, no history, pulmonary or metabolic disease	AIx and AIx75 pre-exercise and **5, 20, 35 and 50 min post-exercise** (applanation tonometry)	HIIT: treadmill exercise (1% inclination) consisting of 10 min warm-up at 70% HR_{max}, followed by 4 × 4 min interval training at 90–95% HR_{max}, with 3 min recovery at 70% HR_{max} between intervals MCT: constant treadmill exercise (1% inclination) at 80% HR_{max} (varied duration to match O_2 uptake during HIIT) Subjects performed both protocols (crossover design) at least 72 h apart	HIIT AIx (%) Pre-exercise: −2 ± 8 20 min post-exercise: −4 ± 8 35 min post-exercise: −6 ± 8 50 min post-exercise: −6.9 ± 8 AIx75 (%) Pre-exercise: −10.8 ± 9 20 min post-exercise: 1.4 ± 9 35 min post-exercise: −4.1 ± 9 50 min post-exercise: −7.4 ± 9 MCT AIx (%) Pre-exercise: −2.6 ± 8 20 min post-exercise: −2.7 ± 8 35 min post-exercise: −2.2 ± 8 50 min post-exercise: −1.9 ± 8 AIx75 (%) Pre-exercise: −11.9 ± 8 20 min post-exercise: −7.9 ± 8 35 min post-exercise: −9.5 ± 8 50 min post-exercise: −10.2 ± 8	Post *vs*. pre associations not reported
Heffernan *et al*.[24]	Healthy African-American men	Non-smokers, taking no medications that affect heart rate or blood pressure, and no CV, metabolic, renal or respiratory disease	cfPWV and fdpPWV pre-exercise and *15 and 30 min post-exercise* (Doppler ultrasound)	Maximal cycling exercise to exhaustion (50 W start, then added 30 W each 2 min)	cfPWV ($m\,s^{-1}$) Pre-exercise: 7.3 ± 0.3 No significant change in cfPWV post-exercise in African-American men (values not provided) fdpPWV ($m\,s^{-1}$) Pre-exercise: 8.2 ± 0.4 No significant change in fdpPWV post-exercise in African-American men (values not provided) *Presented as mean ± s.e.	NS NS
	Healthy Caucasian men	Matched for physical activity level with African-American subjects			cfPWV ($m\,s^{-1}$) Pre-exercise: 5.7 ± 0.3 No significant change in cfPWV post-exercise (values not provided)	NS

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	*P-value*
					fdpPWV ($m\,s^{-1}$) Pre-exercise: 8.4 ± 0.4 Significantly reduced fdpPWV at 15 and 30 min post-exercise compared with baseline (values not provided) *Presented as mean ± s.e.	$P<0.05$
Heffernan *et al.*[24]	Healthy resistance-trained men and age-matched non-trained controls	Non-smokers, BP <140/90 mmHg, not taking any medications, no history of CVD, diabetes, obesity or orthopaedic problems Resistance exercise participants engaged in resistance training more than 3 days per week Controls are sedentary/recreationally active and did not engage in a regular planned exercise program (<1.5 h of unplanned non-vigorous physical activity per week)	cfPWV, fdpPWV, AIx and AIx75 in controls *vs.* resistance-trained men pre-exercise and **10, 20, 30 min post-exercise** (Doppler ultrasound)	Maximal cycle exercise to exhaustion (50 W start and add 30 W every 2 min)	cfPWV ($m\,s^{-1}$) Resistance-trained men: Pre-exercise: 6.4 ± 0.4 10 min post-exercise: 6.5 ± 0.3 20 min post-exercise: 6.6 ± 0.3 30 min post-exercise: 6.6 ± 0.3 Controls: Pre-exercise: 7.1 ± 0.3 10 min post-exercise: 7.0 ± 0.4 20 min post-exercise: 7.1 ± 0.4 30 min post-exercise: 6.7 ± 0.5 fdpPWV ($m\,s^{-1}$) Reduced at 10 min, and remained significantly below pre-exercise at 30 min *in both groups* (values not provided) AIx reduced at 20 and 30 min post-exercise *in both groups* (values not provided) AIx75 (%) Resistance-trained men: Pre-exercise: −10.3 ± 2.3 10 min post-exercise: 4.7 ± 2.4 20 min post-exercise: −3.3 ± 1.2 30 min post-exercise: −3.4 ± 1.7 Controls: Pre-exercise: −6.7 ± 2.6 10 min post-exercise:6.5 ± 2.0 20 min post-exercise: −2.0 ± 1.9 30 min post-exercise: −4.3 ± 1.6 *Presented as mean ± s.e.	Post *vs.* pre NS NS NS NS NS NS $P<0.05$ $P<0.05$ $P<0.05$ $P<0.05$ $P<0.05$ $P<0.05$ $P<0.05$ NS
Heffernan *et al.*[16]	Healthy men	Non-smokers, non-obese, moderately active, not taking medications and with no CV or metabolic complications	cfPWV and fdpPWV pre-exercise and **20 min post-exercise** (Doppler ultrasound)	30 min of upright cycling at 65% VO_{2max}	cfPWV ($m\,s^{-1}$) Pre-exercise: 5.3 ± 0.16 Post-exercise: 4.8 ± 0.25	Post *vs.* pre $P<0.05$

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					fdpPWV (m s^{-1}) Pre-exercise: 8.9 ± 0.25 Post-exercise: 8.3 ± 0.25 *Presented as mean ± s.e.	*P*<0.05
Kingwell *et al.*[17]	Sedentary men	Non-smokers, taking no medications, BMI<27 kg m^{-2}, *BP*<140/90 mm Hg, cholesterol <5.5 mmol l^{-1}, triglycerides <2 mmol l^{-1} and maximal oxygen consumption <50 ml (kgxmin)$^{-1}$	cfPWV, fdpPWV, WBAC at rest and **30 min and 1 h post-exercise** (application tonometry)	30 min cycling on ergometer at 65% VO_{2max}	cfPWV (m s^{-1}) Pre-exercise: 6.2 ± 0.5 30 min post-exercise:5.9 ± 0.3 Decreased 4 ± 2% at 30 min fdpPWV (m s^{-1}) Pre-exercise: 8.3 ± 0.3 30 min post-exercise: 7.5 ± 0.4 Decreased 10 ± 4% at 30 min WBAC (AU) Elevated 66 ± 26% at 30 min and returned to BL by 1 h Pre-exercise: 0.9 ± 0.1 Post-exercise: 1.4 ± 0.3 *Presented as mean ± s.e.	Post *vs.* pre *P*=0.04 *P*=0.01 *P*=0.04
Lane *et al.*[52]	Healthy young adults	Non-smoking, sedentary, normotensive, not taking any chronic medications except oral contraceptives and no chronic disease Women were examined during the early follicular phase, or during the oral contraceptive placebo phase	AIx, cfPWV, fdpPWV pre-exercise, and **15 and 30 min post-exercise** (application tonometry)	Cycling ergometer exercise with a warm-up period (60–100 r.p.m. at 30 W for 30 s, followed by 50 W for 2 min, and then an increase of 30 W every 2 min until exhaustion	Women AIx (%) Pre-exercise: 9.7 ± 1.7 15 min post-exercise: −1.6 ± 1.6 30 min post-exercise: −3.0 ± 2.3 cfPWV (m s^{-1}) Pre-exercise: 5.5 ± 0.8 15 min post-exercise: 5.3 ± 1.0 30 min post-exercise: 5.5 ± 1.2 fdpPWV (m s^{-1}) Pre-exercise: 8.1 ± 0.3 15 min post-exercise: 7.5 ± 0.2 30 min post-exercise: 7.9 ± 0.2 Men AIx (%) Pre-exercise: −1.7 ± 1.8 15 min post-exercise: −8.7 ± 2.1 30 min post-exercise: −9.5 ± 2.3 cfPWV (m s^{-1}) Pre-exercise: 6.1 ± 1.2 15 min post-exercise: 6.2 ± 0.8 30 min post-exercise: 5.9 ± 0.8 fdpPWV (m s^{-1}) Pre-exercise: 8.7 ± 0.3	Post *vs.* pre NS *P*<0.05 *P*<0.05 NS *P*<0.05 *P*<0.05 *P*<0.05 *P*<0.05 NS *P*<0.05

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					15 min post-exercise: 8.2 ± 0.3	$P<0.05$
					30 min post-exercise: 8.5 ± 0.3	$P<0.05$
					*Presented as mean ± s.e.	
Milatz *et al.*[30]	Healthy young men	Non-smokers, not taking antihypertensive medications and free of acute or chronic diseases	aPWV measured pre-exercise and **1, 15,30, 45, and 60 min post-exercise** (oscillometric method)	60 min of endurance cycling exercise on a cycle ergometer at 45% VO_{2max}	aPWV was 3% below baseline at 15 min post-exercise and 2.5% below for the remaining time points	Post *vs.* pre
					aPWV ($m\,s^{-1}$)	
					Pre-exercise: 6.0 ± 0.7	
					15 min post-exercise: 5.8 ± 0.7	NS
					30 min post-exercise: 5.8 ± 0.7	NS
					45 min post-exercise: 5.8 ± 0.7	NS
					60 min post-exercise: 5.8 ± 0.7	$P<0.01$
					*values estimated from bar graphs	
Munir *et al.*[48]	Healthy young adults	Non-smokers, recreationally active, not taking any regular medications, and no history of CVD	Radial and digital pressure waveforms, and cfPWV at rest and **post-exercise (1–3, 15, 30 and 60 min)** (applanation tonometry and servocontrolled finger pressure cuff)	Incremental exercise on bicycle ergometer Start at 25 W, increase by 25 W every 2 min until a) exhaustion, or b) a maximum of 12 min total	cfPWV ($m\,s^{-1}$)	Post *vs.* pre
					Pre-exercise: 7.1 ± 0.2	
					15 min post-exercise:7.1 ± 0.2	NS
					30 min post-exercise: 7.0 ± 0.2	NS
					60 min post-exercise: 7.1 ± 0.2	NS
					Central AI (%)	
					Pre-exercise: 6.2 ± 2.0	
					15 min post-exercise:1.0 ± 1.6*	$P<0.05$
					30 min post-exercise: 1.2 ± 1.8**	$P<0.01$
					60 min post-exercise: 2.9 ± 2.1*	$P<0.05$
					Peripheral systolic AI (%)	
					Radial:	
					Pre-exercise: 54 ± 3.9	
					15 min post-exercise: 42 ± 3.7	$P<0.01$
					30 min post-exercise: 47 ± 3.6	$P<0.01$
					60 min post-exercise: 45 ± 3.5	$P<0.001$
					Finger:	
					Pre-exercise = 51 ± 3.8	
					15 min post-exercise: 45 ± 2.3	NS
					30 min post-exercise: 45 ± 3.1	$P<0.05$
					60 min post-exercise: 47 ± 1.7	$P<0.05$
					Peripheral diastolic AI (%)	
					Radial:	
					Pre-exercise = 37 ± 1.8	
					15 min post-exercise: 25 ± 2.9	$P<0.001$
					30 min post-exercise: 29 ± 1.9	$P<0.01$
					60 min post-exercise: 31 ± 2.85	$P<0.05$
					Finger:	
					Pre-exercise = 37 ± 1.7	

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					15 min post-exercise: 25 ± 2.9	$P<0.001$
					30 min post-exercise: 30 ± 1.8	$P<0.001$
					60 min post-exercise: 30 ± 1.5	$P<0.001$
					*Presented as mean ± s.e.	
Naka *et al.*[41]	Healthy adults	Non-smokers, non-obese, sedentary, taking no medications and no CV complications	brPWV, and maPWV (by oscillometry) pre-exercise and at **every min post-exercise for 60 min** (oscillometry)	Maximal symptom-limited treadmill exercise using Bruce Protocol (until RER < 1.1)	brPWV ($m\,s^{-1}$) Pre-exercise: 8.0 ± 1.2 brPWV dropped to 6% below baseline at 10 min post-exercise (value not provided) Reached steady state of 10% below baseline by 60 min post maPWV ($m\,s^{-1}$) Pre-exercise:7.3 ± 0.9 maPWV decreased to 23% below baseline by 10 min and reached a steady state of 10% below baseline by 60 min (value not provided)	<0.05 for all <0.05 for all
Nickel *et al.*[42]	Healthy older adults	Non-smokers (for at least 3 years), non-active, aged 60 years or older, BMI <30 kg m^{-2} and no CVD	Large and small artery compliance pre-exercise, and **immediately post-exercise** (diastolic pulse contour analysis)	30 min cycling at 50% HRR	No significant change in large artery compliance post-exercise (values not provided) SAEI (ml mm $Hg^{-1} \times 100$) Pre-exercise: 4.2 ± 2.6 Small artery compliance increased 17% from rest to 30 min post-exercise (value not provided)	NS $P<0.001$
Nieman *et al.*[47]	Healthy young adults	Active individuals with a history of regular participation in competitive running races capable of running for 2 h (marathon pace). No history of smoking, heart disease, cancer, diabetes, high blood pressure, arthritis, stroke, or peripheral artery disease	cfPWV and AIx75 pre-exercise, immediately after and at **30, 60, 120, 180, 240 and 300 min post-exercise** (applanation tonometry)	Treadmill running for 2 h at 75% VO_{2max}	Men cfPWV ($m\,s^{-1}$) NS differences 30–300 min post-exercise *vs.* rest condition (data not reported) AIx75 (%) NS differences 30–300 min post-exercise *vs.* rest condition (data not reported) Women cfPWV ($m\,s^{-1}$) Significantly decreased at 30, 60, 240 and 300 min post-exercise *vs.* rest condition (data not reported) AIx75 (%) Significantly decreased 30–	NS NS $P<0.05$ $P<0.05$

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					240 min post-exercise *vs.* rest condition (data not reported)	
Nottin *et al.*[43]	Healthy young adults	Non-smokers, non-obese, taking no medications that effect exercise and no history of hypertension or arterial disease	ULPWV, forearm PWV and brachial PWV at rest and **10 min post-exercise** while sitting upright on cycle ergometer (using continuous wave Doppler and photoplethysmography system)	Two identical sessions, 1 week apart Cycling for 5 min at 70 W, then increase workload to maintain HR at 140 ± 2 b.p.m. for another 8 min	ULPWV ($m\,s^{-1}$) Pre-exercise: 7.0 ± 0.6, 6.8 ± 0.3 10 min post-exercise: 6.7 ± 0.4, 6.7 ± 0.4 Forearm PWV ($m\,s^{-1}$) Pre-exercise: 6.2 ± 0.5, 6.2 ± 0.6 10 min post-exercise: 6.1 ± 0.4, 6.3 ± 0.5 Brachial PWV ($m\,s^{-1}$) Pre-exercise: 8.8 ± 1.8, 8.2 ± 1.8 10 min post-exercise: 8.8 ± 1.8, 8.0 ± 1.6	Post *vs.* pre associations not reported
Payne *et al.*[37]	Healthy young men	No history of cardiovascular or other significant illnesses, and not taking any regular medications	Radial AIx and carotid AIx pre-exercise and **immediately and 10 min post-exercise** (applanation tonometry)	15 min cycling exercise at 65–70% of maximum heart rate	Radial AIx (%) Pre-exercise: 57 ± 14.6 10 min post-exercise: 62 ± 12.3 Carotid AIx (%) Pre-exercise: 18.9 ± 12.2 10 min post-exercise: 29.0 ± 12.5	Post *vs. p*re $P<0.01$ $P<0.001$
Rakobowchuk *et al.*[19]	Healthy young men	Non-smokers, non-obese, normotensive, recreationally active, not taking medications and no CV complications	Femoral artery distensibility *(by ultrasound)* at rest and **immediately post-exercise** fdpPWV at rest and from **2–60 min continuously** post-exercise using *applanation tonometry* at femoral, and infrared blood volume detection device at dorsalis pedis artery	Three sessions: (1) familiarization (2) single sprint interval (1 Wingate anaerobic test) (3) Multiple sprint interval (4 Wingate anaerobic tests)	No statistical difference in femoral artery distensibility post-exercise *vs.* pre-exercise under all conditions (values not provided) Heart-femoral PWV increased significantly (2 min) and returned to baseline levels 20 min into recovery for all exercise conditions (values not provided) fdpPWV decreased significantly (2 min) and returned to baseline levels at 44 min into recovery for all exercise conditions (values not provided)	NS $P<0.001$ $P<0.001$
Ranadive *et al.*[44]	Healthy adults	No history of CV, respiratory or metabolic diseases	crPWV, cfPWV and cdpPWV measured 10 min pre-exercise, and **10 min post-exercise** (a) maximal leg and (b) maximal arm-ergometer VO_{2peak} tests (applanation tonometry)	Maximal arm ergometry: Begin with 15 W workload, increased 15 W every 2 min until test termination	**Arm-ergometry protocol:** crPWV ($m\,s^{-1}$) Pre-exercise: 7.0 ± 0.2 10 min post-exercise: 6.0 ± 0.3 fdpPWV ($m\,s^{-1}$) Pre-exercise: 9.1 ± 0.5	Post *vs.* pre $P<0.05$

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
					10 min post-exercise: 8.1 ± 0.6	NS
					cfPWV (m s^{-1})	
					Pre-exercise: 5.5 ± 0.3	
					10 min post-exercise: 5.7 ± 0.3	NS
				Maximal leg ergometry: Begin with 50 W workload, increased 30 W every 2 min until test termination	**Leg-ergometry protocol:**	Post *vs.* pre
					crPWV (m s^{-1})	
					Pre-exercise: 6.6 ± 0.3	
					Post-exercise: 6.4 ± 0.3	NS
					fdpPWV (m s^{-1})	
					Pre-exercise: 8.7 ± 0.5	
					Post-exercise: 7.6 ± 0.4	$P<0.05$
					cfPWV (m s^{-1})	
					Pre-exercise: 5.6 ± 0.3	
					Post-exercise: 5.5 ± 0.2	NS
Sharman *et al.*[20]	Healthy men	Non-smokers, sedentary or recreationally active, and no history of CVD	PPA before, during and **2, 4, 6, 8,10 min post-exercise** (applanation tonometry)	Cycling on ergometer at 50 r.p.m., beginning at 50%, then increasing to 60%, 70 and 80% of HRmax (4 min at each intensity)	PPA	Post *vs.* pre
					Pre-exercise: 1.57 ± 0.05	
					6 min post: 1.70 ± 0.04	NS
					8 min post: 1.67 ± 0.04	NS
					10 min post: 1.66 ± 0.04	NS
					*Presented as mean ± s.e.	
Sharman *et al.*[38]	Healthy young men	Non-smokers, normotensive, not taking medications, no history of hypertension, CVD, diabetes or hypercholesterolemia	AIx (%) pre-exercise, and **2 and 10 min post-exercise** in a seated position (applanation tonometry)	Cycling on a stationary bicycle ergometer at 50 r.p.m. and 60% of their maximal heart rate for 10 min	AIx (%)	Post *vs.* pre associations not reported
					Pre-exercise: 6 ± 3	
					10 min post-exercise: 1 ± 4	
Tordi *et al.*[23]	Healthy young men	Non-smokers, taking no medication, no CV, pulmonary or metabolic complications	crPWV and cdpPWV before and **for 30 min (2, 4, 6, 8, 10, 12, 14, 16, 20, 24 and 28 min)** after IE and CE (applanation tonometry)	IE: 30 min total (6 consecutive periods of: 4 min at 65% HRmax+1 min at 85% HRmax) CE: 30 min total. Constant exercise at level equal to HR average of IE	crPWV was nonsignificantly elevated at 6–28 min post-IE	NS
					cdpPWV at 6–28 min post-IE was significantly lower than baseline	$P<0.05$
					crPWV at 6–28 min post-CE was nonsignificantly lower than baseline	NS
					cdpPWV at 6–28 min post-CE was not significantly different	NS
Wang *et al.*[26]	Healthy young men	Non-smoking, active, normotensive with no history of CVD and no medications for diabetes, metabolic disease or CVD	CAVI pre-exercise, and **40 min post-exercise** compared with non-exercise control session (crossover design) *CAVI vascular screening system is used* (combines oscillometry, phonocardiogram, and electrocardiogram)	Cycle ergometer for 30 min at 25% HRR	Significantly lower CAVI values 40 min after cycling compared with non-exercise control session	NS for 40 min post-exercise values (cycling *vs.* control, no analysis post *vs.* pre within test)
					CAVI (control):	
					Baseline: 6.6 ± 0.1	
					70 min: 6.6 ± 0.1	
					CAVI (cycling):	

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	*P-value*
					Baseline: 6.5 ± 0.1 40 min post-exercise: 6.4 ± 0.1 *Presented as mean ± s.e.	
Yan *et al.*[53]	Healthy young AA and Caucasian American CA adults	Non-smoking, normotensive, no CV, metabolic, renal or respiratory disease, and not taking any medications	AP, AIx75, cfPWV, dpfPWV, pre-exercise **15 and 30 min post-exercise** (applanation tonometry)	Incremental graded cycle ergometer test to exhaustion (increase of 30 W every 2 min until termination) followed by 2 min of light cycling, a 1 min rest	AP Overall decrease in AP at 15 and 30 min post-exercise in men and women (AA and CA) AIx Overall decrease in AIx at 15 and 30 min post-exercise in men and women (AA and CA) AIx75 Overall increase in Aix75 at 15 min in all men. At 30 min an increase was observed in CA men, but not AA men Overall decrease in Aix75 in women (AA and CA) crPWV Overall increase in crPWV at 15 min in all men. At 30 min an increase was observed in AA men, but a decrease was seen in CA men Overall decrease in crPWV at 15 min in all women. At 30 min an increase was observed in AA men, but not CA men cfPWV Overall decrease in cfPWV at 15 and 30 min post-exercise in men and women (AA and CA) * Statistical significance was only assessed between race (CA and AA), and does not capture whether the change in response to exercise was significant	Post *vs.* pre exercise associations not reported
Zheng *et al.*[27]	Healthy young men	Non-smoking, active, normotensive with no history of CVD and no medications for diabetes, metabolic disease or CVD	CAVI pre-exercise, and **60 min post-exercise** compared with non-exercise control session (crossover design) *CAVI vascular screening system is*	Cycle ergometer for 30 min at 50% HRR (r.p.m. of 60)	Significantly lower values 60 min after cycling compared with non-exercise control session CAVI (control): Baseline: 6.8 ± 0.1	$P<0.001$ for post-exercise values (cycling *vs.* control, no analysis post *vs.* pre within test)

Table 3 (Continued)

Study	*Subjects*	*Inclusion criteria*	*Comparison*	*Exposure*	*Outcome*	P-*value*
			used (combines oscillometry, phono-cardiogram, and electrocardiogram)		90 min: 6.9 ± 0.2 CAVI (cycling): Pre-exercise: 6.8 ± 0.1 60 min post-exercise: 6.7 ± 0.1 *Presented as mean ± s.e.	
Zhou *et al.*[31]	Healthy young men	Non-smoking, moderately active (no varsity athletes), taking no medications and no history of CVD, respiratory or endocrine disorders	CAVI pre-exercise and **10, 20, 30, 40 and 60 min post-exercise**	Cycle ergometer for 10 min at 50% HRR (performed in subset, $n=6$)	CAVI Pre-exercise: 7.1 ± 0.3 10 min post-exercise: 6.0 ± 0.3 20 min post-exercise: 6.1 ± 0.1 30 min post-exercise: 6.4 ± 0.2 40 min post-exercise: 6.7 ± 0.2 60 min post-exercise: 6.9 ± 0.3	Post *vs.* pre $P<0.05$ $P<0.05$ $P<0.05$ NS NS
			CAVI pre- and **60 min post-exercise**	Cycle ergometer for 30 min at 50% HRR (performed in all subjects, $n=19$)	CAVI Pre-exercise: 6.9 ± 0.2 60 min post-exercise: 6.8 ± 0.1 *Presented as mean ± s.e.	 NS

Abbreviations: AI/AIx, augmentation index; AIx75, augmentation index adjusted to heart rate of 75 b.p.m.; AP, augmentation pressure; BMI, body mass index; b.p.m., beats per minute; brPWV, brachial–radial PWV; CAVI, carotid–ankle vascular index; cdpPWV, carotid–dorsalis pedis PWV; cfPWV, carotid–femoral PWV; crPWV, carotid–radial PWV; CV, cardiovascular; CVD, cardiovascular disease; fdpPWV, femoral–dorsalis pedis PWV; HIIT, high-intensity interval training; HR, heart rate; HRR, heart rate reserve; maPWV, midthigh–ankle PWV; MCT, moderate continuous training; PPA, pulse pressure amplification; PWV, pulse wave velocity; RER, respiratory exchange ratio; r.p.m., revolution per minute; SAEI, small artery elasticity index; s.d., standard deviation; s.e., standard error; ULPWV, upper limb PWV; WBAC, whole-body arterial compliance; VO_{2max}, maximal oxygen consumption.
All values are expressed as mean plus/minus s.d., unless otherwise indicated.

in brachial–radial PWV 10 min post-exercise, reaching a significant steady state of 10% below resting values by 60 min. As previously noted in the same study, brachial–radial PWV had been increased 35% at the 3 min post-exercise time point in the same study.[41] Ranadive *et al.*[44] also found significantly decreased carotid–radial PWV 10 min after maximal arm ergometry in healthy volunteers. At 20 and 30 min after cycle exercise, AIx was shown to be significantly reduced compared with resting levels in resistance- and non-resistance-trained men,[16] whereas Munir *et al.*[48] demonstrated significantly decreased central AIx 15–60 min post-exercise.[48] Diastolic AIx was also shown to be increased at multiple upper body peripheral sites 15–45 min following running exercises of various intensities.[14] Nieman *et al.*[47] demonstrated a significantly decreased AIx75 in women 30–240 min following prolonged aerobic exercise; however, this study did not find significant differences in men on the AIx75 post-exercise compared with resting conditions, whereas, Lane *et al.*[52] observed decreased AIx in both men and women 30 min after maximal cycling exercise. One study did nevertheless note a significantly elevated AIx75 at 5, 10 and 15 min post-exercise.[15] Similar overall results were found in measurements of central stiffness: Heffernan *et al.*[16] and Kingwell *et al.*[17] found cfPWV to be significantly reduced compared with rest at 20 and 30 min after cycling exercise, respectively. In line with these findings, Lane *et al.*[52] also observed significantly lower cfPWV in men and women at 15 and 30 min after maximal cycling exercise. Nieman *et al.*[47] also recently demonstrated significantly decreased cfPWV in women 30, 60, 240 and 300 min following prolonged aerobic exercise compared with resting conditions, but no differences were noted in men. By 10 and 20 min post-exercise, significantly elevated cfPWV had also returned to resting values in studies by Doonan *et al.*[15] and Rakobowchuk *et al.*,[19] respectively.

Changes in *lower limb* arterial stiffness measures >5 min post-exercise are similar to those of the upper limbs and larger central arterial segments. At 15 and 30 min post-exercise, Heffernan *et al.*[24] demonstrated a significant decrease in femoral–dorsalis pedis PWV in white men, but not in African-American men. No significant change in cfPWV was noted in this group. Similarly, femoral–dorsalis pedis PWV was found to be significantly decreased 10–30 min after cycling exercise in both resistance-trained and age-matched non-resistance-trained controls.[24] These findings are further confirmed by others who showed decreased femoral–dorsalis pedis PWV for 44 min after high-intensity interval exercise,[19] 4–28 min after cycling intervals,[23] at 10 min after maximal leg ergometry,[44] at 30 min after cycling exercise[17] and 15 and 30 min after a maximal cycling test.[52] Measures of the less conventional midthigh–ankle PWV were also shown to be significantly decreased by 23% at 10 min post-exercise, and reached a steady state of 10% below resting values by 60 min post-exercise.[41] Studies measuring CAVI reported a return to baseline when measured 40 min[26,31] and 60 min[27,31] after 30 min of cycling.

Using pulse contour analysis, Nickel *et al.*[42] recently showed a 17% increase in small artery compliance from rest to 30 min after moderate-intensity cycling exercise. Kingwell *et al.*[17] also found that whole-body arterial compliance was increased by 66% at 30 min post-exercise and remained elevated for up to 60 min following moderate-intensity cycling.

DISCUSSION

We present herein data from 43 studies evaluating the effect of acute aerobic exercise on measures of arterial stiffness in healthy human subjects. We found that the effect of acute aerobic exercise on arterial stiffness is dependent on the anatomical segment being assessed, and on the time at which the measurement is performed following acute aerobic exercise.

The vast majority of included studies implemented primarily lower limb exercise modalities. It was noted that immediately post-exercise (0–5 min), the central and upper body arterial segments demonstrated overall increases in arterial stiffness, whereas measures taken in lower limb segments (proximal to the primary working muscles in most instances) demonstrated immediate decreases in arterial stiffness. As time post-exercise progressed (>5 min), measures of stiffness (of the central, upper and lower body) decreased to or below resting values. However, the magnitude of these post-exercise changes and the timeline over which they occur is variable among different studies.

Mechanisms

Shift in arterial wall properties (structural components). As the heart increases its cardiac output during acute aerobic physical activity, there is an increase in the demands placed on the vascular system. During aerobic exercise, there is an increase in blood pressure, alterations to sympathetic vascular tone and concomitant increase in measures of central arterial stiffness, which may persist early into the recovery period following activity cessation.[13] The physiological alteration caused by exercise leads to changes in the functional properties of the arterial wall.[55] A shift in the load from the more distensible elastin fibers to less compliant collagen fibers in the central arterial segments, alongside constriction of vascular smooth muscle cells, could account for immediate increases in central arterial stiffness observed following exercise.[55] There is also an associated increase in blood flow and resultant shear stress in vessels supplying the working muscles, causing vasodilation and increases to vessel diameter. As the arterial diameter increases, there may again be a resultant shift in the load from elastin to collagen fibers, and could account for early increases in arterial stiffness of upper body arterial segments immediately following exercise.[17]

Alongside vasodilation, exercise leads to reduced total peripheral resistance and an increase in whole-body arterial compliance.[17,56] It has been proposed that a *relaxation* of vascular smooth muscle could work in the opposite manner to increased vascular tone by moving from primarily collagen fibers to more compliant elastin, and could account partially for *decreases* in arterial stiffness seen post-exercise in portions of the arterial tree.[57]

Blood pressure. Blood pressure is acutely increased during aerobic exercise, and decreases in a time-dependent manner following exercise cessation.[58] Multiple studies have, however, demonstrated that the post-exercise decrease in mean arterial pressure is independent of changes in arterial stiffness,[16,17,23] and is, thus, unlikely to be the governing mechanism. Furthermore, studies have noted an acute effect of exercise on CAVI, which is a blood pressure-independent measure of arterial stiffness.[26,27,31]

Central nervous system. Following acute aerobic exercise, there is a reduction in muscle sympathetic nerve activity.[59] As in exercise, in response to stretching, carotid and aortic baroreceptors send afferent signals to the brainstem that act to inhibit sympathetic activity to the periphery,[60] resulting in vessel vasodilation and associated alterations to arterial stiffness as discussed above. Baroreflex sensitivity was shown to be significantly decreased 30 min following cycling exercise, independent of changes to arterial wall components and in the presence of reduced cfPWV and femoral–dorsalis pedis PWV.[16] It is, however, possible that decreases in arterial stiffness may also

be seen concomitantly with sympathoexcitation, as vasodilation has previously been shown to occur alongside sympathetic activation.[61]

Blood markers. With exercise, increased intravascular shear stress and amplitude and frequency of pulsatile flow cause the production and release into the blood of multiple modulators of vascular tone derived by the endothelium.[62] A number of these blood markers may be implicated in the mechanisms governing changes in arterial stiffness following aerobic exercise. There is an alteration in circulating levels of such vasorelaxing factors as nitric oxide,[34] prostaglandins,[62] atrial natriuretic peptide[63] and potential vasoconstrictors such as endothelin-1, angiotensin-II and epinephrine[62] that help mediate vascular smooth muscle tone with exercise. For example, our group has previously demonstrated a significant decrease in circulating endothelin-1 levels in response to acute maximal treadmill exercise in young healthy individuals.[64] There is also current evidence supporting a relationship between arterial stiffness and various blood markers in resting conditions,[65] but further research needs to be conducted investigating the relationship between blood markers and changes in arterial stiffness with exercise.

Clinical implications

It has been extensively demonstrated that chronically increased PWV and AIx are directly and independently associated with an increased risk for CV complications and events.[5,6] Conversely, regular aerobic exercise has notably been shown to reduce the incidence of various CV complications as obesity, diabetes, hypertension and atherosclerosis.[2] Our summarized findings demonstrate the complex relationship between *acute* aerobic stress and changes in arterial stiffness—namely that segments of the arterial tree are differentially affected and respond/recover in a time-dependent manner following physical stress cessation. Therefore, when assessing the overall impact of exercise on arterial stiffness, it is important to consider the arterial segment being analyzed and measurement time point, as failure to contextualize the measurement can lead to conflicting results and misleading clinical inferences. Moreover, arterial stiffness measures that incorporate the arterial segments of the lower limbs, such as carotid–dorsalis pedis, brachial–ankle PWV and CAVI will potentially capture a different response than measures assessing only central stiffness, and this needs to be considered when reporting the effects of acute exercise on arterial stiffness. Furthermore, as AIx is a measure of wave reflection that is greatly influenced by changes in heart rate (HR), it is important to consider whether adjustments for HR have been performed. In this context, our findings have demonstrated opposing effects of acute exercise on AIx and AIx correct for HR (AIx75), and therefore this needs to also be considered when evaluating changes in systemic stiffness after acute exercise.

In the young populations evaluated herein, we observed a transient increase in central stiffening immediately following aerobic exercise. However, such an increase in central stiffening may be a cause for concern in older, more at-risk populations with whom acute periods of strenuous exercise have been associated with a temporary increase in risk for CV events.[66] To our knowledge, no studies to date have assessed the effect of acute aerobic exercise on arterial stiffness in a population with CV disease. On the other hand, a limited number of studies have been conducted in a population with CV risk factors, such as hypertension, dyslipidemia and T2DM.[28,67–74] However, owing to heterogeneity of the patient populations, and use of different measurement methodologies and exercise modalities, it is not feasible to systematically assess the overall effect of acute aerobic exercise on arterial stiffness in such CV risk populations.[28,67–74] Nevertheless, the only study that investigated cfPWV, the 'gold-standard' measure of central arterial stiffness, observed significantly elevated cfPWV immediately after maximal cycling exercise in newly diagnosed, untreated, hypertensive subjects.[28] In line with our findings in a healthy population, these results underline the necessity of vigilant patient evaluation pre-exercise testing or prescription of strenuous exercise in at-risk populations. To confirm the above-mentioned observations, future studies will need to be conducted specifically in patient populations with CV risk factors and CV disease.

Limitations

This review is somewhat limited by the comparability of the included studies. Importantly, there was no standardization of the protocols or methods used between studies. The duration, intensity and modality of exercise varied greatly between protocols. Thus, it was not feasible to evaluate the exercise dose response on arterial stiffness post-exercise. Factors that impact vessel hemodynamics and/or exercise capacity, such as smoking status and the intake of caffeine, alcohol or flavonoid-containing foods differed between protocols, or were altogether not reported. This was also the case in some studies for sex, BMI, and baseline physical activity. The majority of the identified studies enrolled young adults, which may limit the generalizability of summarized findings, yet this allowed us to assess of the normal physiological arterial response to acute aerobic exercise without the presence of confounding CV risk factors. The timing and duration of arterial stiffness measurements post-exercise also varied substantially, as did the methods used to quantify arterial changes. cfPWV is considered the 'gold-standard' in the measurement of arterial stiffness,[5,6] and, thus, other measures, such as β-stiffness index, compliance or CAVI may not be comparable in the assessment of exercise effects. With such limitations, a meta-analysis investigating the overall effect of acute aerobic exercise on arterial stiffness could not be conducted.

CONCLUSION

To our knowledge, this is the first systematic review investigating the effect of acute aerobic exercise on arterial stiffness. Herein, we evaluated relevant results obtained from 43 observational studies. Acute aerobic exercise was shown to have varying effects on arterial stiffness, according to the arterial segment being assessed and the time at which the measure was performed post-exercise. Arterial stiffness of the *central* and *upper body peripheral* arterial segments is increased relative to resting values post-exercise (0–5 min), and thereafter (>5 min) decreases to a level at or below resting values. In the *lower limbs* there is a decrease in arterial stiffness immediately post-exercise (0–5 min), which persists well into the recovery period post-exercise (>5 min). Owing to differences in methodologies and protocols of included studies, a meta-analysis on the effect of acute aerobic exercise on arterial stiffness was not feasible. Future studies evaluating arterial stiffness parameters should include assessment of central, upper body and lower body arterial segments when possible, and follow standardized post-exercise timeline protocols.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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