



A meta-analysis of the effects of strength training on arterial stiffness

review paper

© Wrocław University of Health and Sport Sciences

DOI: <https://doi.org/10.5114/hm.2023.117126>

PABLO GARCÍA-MATEO¹, RODRIGO RAMIREZ-CAMPILLO^{2,3},
ANTONIO GARCÍA-DE-ALCARAZ^{1,4}, MANUEL ANTONIO RODRÍGUEZ-PÉREZ^{1,4}

¹ Faculty of Education Sciences, University of Almería, Almería, Spain

² Department of Physical Activity Sciences, University of Los Lagos, Santiago, Chile

³ Exercise and Rehabilitation Sciences Laboratory, School of Physical Therapy, Faculty of Rehabilitation Sciences, Universidad Andres Bello, Santiago, Chile

⁴ SPORT Research Group (CTS-1024), Centre for Neuropsychological Evaluation and Rehabilitation (CERNEP), University of Almería, Almería, Spain

ABSTRACT

Purpose. Arterial stiffness (AS) describes the mechanical properties of the arterial wall and predicts cardiovascular health. Even if it is known that AS is improved by aerobic exercise, the effects of resistance training (RT) are less clear. Therefore, this meta-analysis aimed to assess the effects of RT on AS.

Methods. A systematic search for randomized controlled trials published until October 2020 was performed in the PubMed, SPORTDiscus, MEDLINE, and Web of Science databases. Overall, 19 studies were selected, with 12.58 ± 0.82 methodological quality points (from a total 15 points) and a total of 626 participants.

Results. No significant long-term effect was noted for RT on AS ($ES = -0.07$; 95% CI: -0.59 to 0.45 ; $p = 0.789$). However, RT induced a significant acute increase in AS ($ES = 1.07$; 95% CI: 1.55 to 0.59 ; $p < 0.001$). No other factors (i.e., age, gender, AS measurement, upper- vs. lower-body RT, training intensity, duration, frequency) had a significant modifying effect on AS in acute or long-term interventions.

Conclusions. Although RT induces an acute AS increase, this effect has no long-term impact, irrespective of the participant's age, sex, or RT variables, such as intensity. However, the clinical implications of acute AS increase after RT are unknown.

Key words: human physical conditioning, strength training, exercise, cardiovascular diseases, vascular stiffness

Introduction

Arterial stiffness (AS) concerns the mechanical characteristics of the wall of the vessels [1]. It applies to the way in which blood pressure, blood flow, and arterial diameter are affected by each heartbeat, having functional consequences on the cardiovascular system [1]. Raised AS alters the normal arterial function, such as buffering and increase in systolic blood pressure, which may lead to abnormal ventricular hypertrophy, reductions in baroreflex sensitivity, and coronary diseases [2, 3]. High AS values (e.g., 12 m/s in young adults) [4] are closely related with a risk of major cardiovascular events [5, 6], cardiovascular diseases

(CVD) [7], and stroke [5]. Moreover, elevated AS increases the risk of developing hypertension [8–10]. In this sense, raised AS may be considered the cause of hypertension, not simply a consequence [9–11]. Hence, AS in young individuals may be a crucial factor to predict cardiovascular health [7]. Pulse wave velocity (PWV), obtained from pulse wave analysis, is the most common way of quantifying AS [1]. PWV can be measured from carotid to femoral arteries (cfPWV) [12], between the ankle and the brachial artery (baPWV) [13], or as an augmentation index (AIx; the higher the AIx, the higher the PWV, hence higher AS) [14]. However, the gold standard of evaluation is at the level of the aorta (aPWV) [15].

Correspondence address: Pablo García-Mateo, SPORT Research Group (CTS-1024), Department of Education, Faculty of Education Sciences, University of Almería, Carretera de Sacramento, s/n, 04120, La Cañada, Almería, Spain, e-mail: pabloogm21@gmail.com, <https://orcid.org/0000-0002-3254-4446>

Received: October 22, 2021

Accepted for publication: July 4, 2022

Citation: García-Mateo P, Ramirez-Campillo R, García-De-Alcaraz A, Rodríguez-Pérez MA. A meta-analysis of the effects of strength training on arterial stiffness. Hum Mov. 2023;24(2):1–17; doi: <https://doi.org/10.5114/hm.2023.117126>.

Nowadays, physical activity is widely known as a fundamental behavioural strategy for treatment and prevention of CVD [15, 16]. Two common training methodologies are the most frequently prescribed: aerobic training and resistance training (RT). Aerobic training exercises have been promoted as a convenient strategy to prevent and reverse AS in healthy adults [16, 17]. Similarly, RT has been recommended for treating CVD-related conditions, such as osteoporosis, sarcopenia [18], impaired glucose and lipid metabolism [19], and related risks, such as falling and functional disability [20]. Moreover, RT may be crucial in controlling hypertension since it reduces blood pressure in both acute [21–23] and chronic [24] ways.

Nevertheless, compared with aerobic training, RT is less commonly prescribed to treat and prevent CVD, probably owing to the doubts existing about the safety in risk patients, which is still in question [25–27]. This, in turn, may be related to the scarcity of research on RT and its potential effects (e.g., benefits) on non-musculoskeletal components, such as cardiovascular function [27], particularly AS. Although previous meta-analyses explored the effects of RT on AS, heterogeneous findings were reported [28–30]. In addition, earlier studies included patients with CVD [28] or mixed samples of CVD patients and healthy people [31]. Also, in the aforementioned meta-analyses, factors affecting the

effects of RT, such as the type of machine or intensity of training, were not explored. Furthermore, to our knowledge, no previous works addressed both acute and long-term conditions. Therefore, this meta-analysis aimed to assess both short- and long-term effects of RT on AS.

Material and methods

Experimental approach to the problem

The guidelines of the PRISMA statement (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) [32] were followed (Appendix 1).

Eligibility criteria

In order to evaluate the studies for eligibility, the PICOS (Participant, Intervention, Comparator, Outcomes, and Study design) approach was used. The selection criteria were defined beforehand (Table 1).

To be included in this meta-analysis, the studies had to be randomized controlled trials evaluating both RT and AS expressed in PWV, AIx, or cardio-ankle vascular index. Also, the interventions had to be exclusively based on RT exercises. In addition, for chronic effects, a minimum period of 4 weeks duration with

Table 1. Selection criteria used in the meta-analysis

Category	Inclusion criteria	Exclusion criteria
Population	Adults (> 18 years of age), normotensive, with normal body mass index	Adults with health problems that prevented sports practice (i.e., osteo-muscular pathology, recent history of surgery or contusion) or a pre-clinical condition (i.e., pre-hypertensive or pre-diabetic)
Intervention	Resistance training interventions. Either a single intervention (acute effects) or a minimum of 2 days per week, 4-week interventions (chronic effects), which are the minimum time and frequency necessary for neuronal and structural adaptations to occur [33]	Exercise interventions not involving resistance training or combining resistance training with another training modality (i.e., aerobic training)
Comparator	Control group (either sedentary, stretching or resistance intervention)	Absence of control group
Outcomes	At least 1 measure of arterial stiffness (e.g., pulse wave velocity, augmentation index, heart-ankle cardiovascular index) before and after the training intervention	Lack of baseline and/or follow-up data
Study design	Randomized controlled trials	Non-controlled trials, descriptive designs without intervention, literature reviews, meta-analyses, letters to the editor, comments, abstracts

a frequency of at least 2 days per week were required, since this has been proved to be the shortest amount of stimulus necessary for structural and neuronal adaptations to occur [33]. For data synthesis, studies were divided into 2 groups: chronic and acute effects.

Information sources

The PubMed, Web of Science, SPORTDiscus, and MEDLINE databases were searched for relevant studies published until October 30, 2020.

Search strategy

The search strategy used was: (“resistance training” OR “strength training” OR “weight training”) AND (“arterial stiffness” OR “arterial elastance” OR “pulse wave velocity” OR “PWV” OR “brachial-ankle pulse wave velocity” OR “baPWV” OR “wave reflection”).

Selection process

In the first screening, a review of title and abstract was performed, and duplicates were removed. Thereafter, the full-text articles were reviewed. Two authors conducted this process independently, and a third researcher was consulted when potential discrepancies arose. The papers excluded were recorded, together with the exclusion details.

Data collection process

A customized spreadsheet was used to gather the data extracted from the papers. In the case of unavailable data, corresponding authors were contacted. If the data could finally not be accessed, the study was excluded. However, when data were displayed in a figure, information was obtained directly from the figures by using a validated software ($r = 0.99$; $p < 0.001$; Web-PlotDigitizer; <https://apps.automeris.io/wpd/>) [34]. Two authors conducted this process independently, and a third researcher was consulted when potential discrepancies arose.

Data items

Basic information from each study was initially extracted: authorship, publication date, study design, and sample size. Besides, sample characteristics and the RT protocols (i.e., training modality [i.e., free weights, machines, combined], main muscle group trained,

number of exercises, intensity, frequency, duration, sets, repetitions, duration of resting intervals) were collected. Mean and standard deviation AS data from before and after the training bouts were obtained.

Risk of bias assessment

The randomized controlled trials were evaluated with a specific validated (intraclass correlation coefficient ≥ 0.91 ; $p < 0.001$) tool for the assessment of study quality and reporting in exercise training studies (TESTEX) [35]. The scale includes sections concerning randomization, blinding, statistical analysis, withdrawals, and activity monitoring.

Synthesis methods

For the classification of the extracted data, the studies were organized into: (a) free-weight, machine, or combined training interventions; (b) programs focused exclusively on the upper muscle training, lower muscle training, or combined; (c) eccentric or concentric muscle actions; (d) fast (1-s lifting phase) or slow-controlled (3-s lifting phase) RT movements; and (e) high ($> 70\%$ of one-repetition maximum [1RM]), moderate (50–69% 1RM), or low ($< 50\%$ 1RM) intensity [36].

Central, peripheral, and systemic AS were established with measurements of central AS (i.e., cfPWV, aPWV, AIx), peripheral AS (i.e., femoral-tibial PWV), and systemic AS (cardio-ankle vascular index, baPWV).

Effect size (*ES*; Hedges' *g*) was calculated from AS means and standard deviations. Data were standardized by using post-score standard deviation. The model of inverse-variance random-effects meta-analysis was used. *ES* was interpreted as trivial (< 0.2), small (0.2–0.6), moderate (> 0.6 –1.2), large (> 1.2 –2.0), very large (> 2.0 –4.0), and extremely large (> 4.0) [37], with 95% confidence intervals (CI). To provide a comparison across participants, the control group was proportionately divided into the intervention groups [38]. Heterogeneity was considered to be low ($< 25\%$), moderate (25–75%), or high ($> 75\%$), on the basis of the I^2 statistic [39].

The potential sources of heterogeneity that might have influenced the results were selected beforehand, through a random-effects model and independent computed single factor analysis. Age, gender, training duration and frequency (weeks and weekly sessions, respectively), training intensity, type of RT equipment (i.e., machine, free weights), body part trained (i.e., full body, upper body), and AS assessment (e.g., cfPWV,

baPWV) were taken into consideration as moderator variables. When appropriate, the median split technique was used to divide single factor analyses [40–42]. The median was computed only when data for a given moderator were available in 2 or more studies.

Reporting bias assessment

The extended Egger’s test [43] was used to assess the risk of bias, in which case, the trim-and-fill method was applied [44] and L0 was considered as the default estimator for missing studies [45]. The Comprehensive Meta-Analysis software (version 2; Biostat, Englewood, NJ, USA) was used for all the analyses. Statistical significance was set at $p \leq 0.05$.

Ethical approval

The conducted research is not related to either human or animal use.

Results

The literature search retrieved 647 studies from databases and literature reviews. Before screening, records were removed for being duplicated ($n = 367$), as well as for other reasons (i.e., language, meta-analysis, reviews, reports, letters to the editor) ($n = 193$), which resulted in 87 records. Then, 8 excluded records were sought for retrieval, of which 3 were finally retrieved, so 90 items were assessed for eligibility. Thereafter, 71 studies were excluded owing to different reasons, detailed in Appendix 2. A total of 19 studies met the selection criteria, involving 626 participants. The article’s screening PRISMA 2020 flowchart is shown in Figure 1 [32]. Of the selected studies, 12 investigated the long-term effects of RT on AS, providing 19 groups that carried out an intervention ($n = 226$) and 12 control groups ($n = 126$). The remaining 7 papers analysed acute effects, providing 8 training groups ($n = 145$) and 7 control groups ($n = 129$).

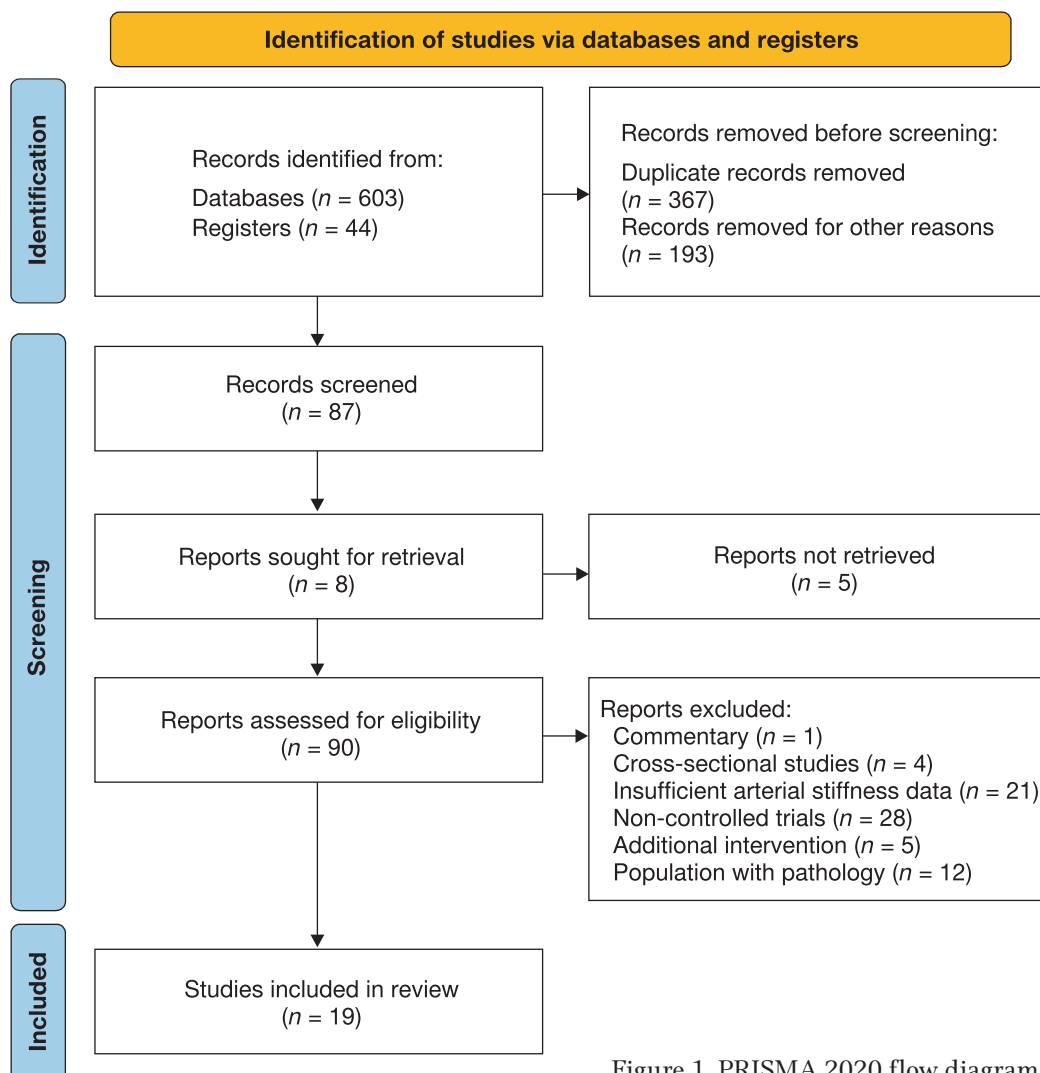


Figure 1. PRISMA 2020 flow diagram of the study

Table 2. Characteristics of the randomized controlled trials: long-term effects

Authors (year)	n, gender	Age (years)	Groups (n)	AS outcome	Pre- / post-intervention results	Effect size (d)	Arterial stiffness ($\Delta\%$)
Au et al. (2017) [46]	46, M	23 \pm 2	G1 (16)	cfPWV	6.4 \pm 0.7 / 5.7 \pm 0.6 m/s	-1.07	\downarrow 10%
			G2 (16)		6.2 \pm 0.6 / 5.8 \pm 0.8 m/s	-0.57	\downarrow 6%
			G3 (14)		5.9 \pm 0.7 / 6.0 \pm 0.7 m/s	0.14	\leftrightarrow
Casey et al. (2007) [47]	42, M/F (23 F)	21.4 \pm 0.6	G1 (24)	cfPWV	6.5 \pm 0.14 / 6.3 \pm 0.19 m/s	-1.20	\leftrightarrow
			G2 (18)		6.9 \pm 0.15 / 7.0 \pm 0.16 m/s	0.65	\leftrightarrow
Cortez-Cooper et al. (2005) [48]	33, F	28.4 \pm 1.3	G1 (23)	cfPWV	7.91 \pm 0.88 / 8.33 \pm 0.96 m/s	0.46	\uparrow 5%
			G2 (10)		7.24 \pm 0.83 / 7.80 \pm 0.66 m/s	0.75	\uparrow 8%
Okamoto et al. (2006) [49]	29, F	19.3 \pm 0.6	G1 (10)	baPWV	9.91 \pm 0.71 / 9.62 \pm 0.83 m/s	0.375	\downarrow 3%
			G2 (10)		9.82 \pm 0.82 / 10.87 \pm 1.01 m/s	1.14	\uparrow 11%
			G1 (9)		9.86 \pm 0.85 / 9.94 \pm 0.70 m/s	0.10	\leftrightarrow
Okamoto et al. (2011) [50]	26, M/F (7 F)	18.55 \pm 0.5	G1 (13)	baPWV	10.95 \pm 1.47 / 10.21 \pm 1.28 m/s	-0.537	\downarrow 7%
			G2 (13)		10.95 \pm 1.47 / 10.95 \pm 1.48 m/s	0	\leftrightarrow
Okamoto et al. (2012) [51]	30, M/F	19.2 \pm 0.7	G1 (10)	cfPWV	6.13 \pm 0.4 / 6.27 \pm 0.54 m/s	0.30	\leftrightarrow
			G2 (10)		6.05 \pm 0.49 / 7.42 \pm 0.38 m/s	3.13	\uparrow 23%
			G3 (10)		6.35 \pm 0.41 / 6.31 \pm 0.76 m/s	-0.07	\leftrightarrow
Raj et al. (2012) [52]	25, M/F (11 F)	68 \pm 5	G1 (13)	ftPWV	10.8 \pm 3.7 / 12.1 \pm 7.4 m/s	0.22	\uparrow 12%
			G2 (12)		14.5 \pm 10.3 / 16.0 \pm 10.8 m/s	0.14	\uparrow 10%
Okamoto et al. (2009) [53]	30, M	19.5 \pm 0.2	G1 (10)	baPWV	10.71 \pm 0.39 / 10.21 \pm 0.31 m/s	-1.42	\downarrow 5%
			G2 (10)		10.52 \pm 0.39 / 11.56 \pm 0.31 m/s	2.95	\uparrow 10%
			G3 (10)		10.68 \pm 0.3 / 10.78 \pm 0.33 m/s	0.317	\leftrightarrow
Yasuda et al. (2014) [54]	19, M/F (15 F)	69.4 \pm 6.5	G1 (9)	CAVI	9.1 \pm 1.4 / 9.0 \pm 0.5 m/s	-0.10	\leftrightarrow
			G2 (10)		8.7 \pm 0.8 / 8.5 \pm 0.1 m/s	-0.35	\leftrightarrow
Okamoto et al. (2009) [55]	30, M/F (11 F)	20.1 \pm 0.4	G1 (10)	baPWV	11.21 \pm 0.40 / 12.66 \pm 0.54 m/s	3.05	\uparrow 13%
			G2 (10)		11.55 \pm 0.35 / 11.46 \pm 0.37 m/s	-0.25	\leftrightarrow
			G3 (10)		11.35 \pm 0.3 / 11.3 \pm 0.37 m/s	0.317	\leftrightarrow
Cortez-Cooper et al. (2008) [56]	24, M/F	52.5 \pm 1.5	G1 (12)	cfPWV	11.09 \pm 0.37 / 10.48 \pm 0.31 m/s	-1.79	\downarrow 5%
			G3 (12)		11.33 \pm 0.55 / 11.32 \pm 0.54 m/s	-0.02	\leftrightarrow
Werner et al. (2019) [57]	30, M	21.7 \pm 3	G1 (10)	cfPWV	6.5 \pm 0.8 / 6.9 \pm 1.5 m/s	0.33	\uparrow 6%
			G2 (10)		7.0 \pm 2.1 / 8.0 \pm 1.7 m/s	0.419	\uparrow 14%
			G3 (10)		6.6 \pm 0.9 / 6.6 \pm 0.9 m/s	0	\leftrightarrow

AS – arterial stiffness, M – male, F – female, G1 – experimental group 1, G2 – intervention group 2 / control, G3 – control group, cfPWV – carotid-femoral pulse wave velocity, baPWV – brachial-ankle pulse wave velocity, ftPWV – femoral-tibial pulse wave velocity, CAVI – cardio-ankle vascular index

Table 3. Characteristics of the randomized controlled trials: acute effects

Authors (year)	n, gender	Age (years)	Groups (n)	AS outcome	Pre- / post-intervention results	Effect size (d)	Arterial stiffness ($\Delta\%$)
Palmiere et al. (2018) [58]	35, M/F (19 F)	22 \pm 3	G1 (35) G2 (35)	aPWV	4.82 \pm 0.39 / 5.12 \pm 0.58 m/s 4.82 \pm 0.43 / 4.86 \pm 0.5 m/s	0.61 0.086	\uparrow 6% \leftrightarrow
Kingsley et al. (2017) [59]	52, M/F (12 F)	23.0 \pm 0.5	G1 (26) G2 (26)	aPWV	5.3 \pm 0.7 / 5.8 \pm 0.7 m/s 5.4 \pm 0.7 / 5.3 \pm 0.6 m/s	0.71 -0.15	\uparrow 9% \leftrightarrow
Lefferts et al. (2015) [60]	40, M/F (6 F)	24 \pm 4	G1 (20) G2 (20)	aPWV	5.1 \pm 0.5 / 6.0 \pm 0.7 m/s 5.1 \pm 0.5 / 5.2 \pm 0.5 m/s	1.48 0.20	\uparrow 18% \leftrightarrow
Augustine et al. (2014) [61]	18, M	24 \pm 6	G1 (9) G2 (9)	aPWV	5.4 \pm 0.5 / 5.6 \pm 0.5 m/s 5.6 \pm 0.7 / 5.5 \pm 0.6 m/s	0.40 -0.16	\leftrightarrow \leftrightarrow
Okamoto et al. (2014) [62]	10, M/F (3 F)	26 \pm 5	G1 (10) G2 (10)	AIx	-5 \pm 2 / -15 \pm 3% -4 \pm 2 / -5 \pm 2%	-3.92 -0.50	\downarrow 10% \leftrightarrow
Yoon et al. (2010) [63]	26, M	20.8 \pm 2.2	G1 (13) G2 (13)	cfPWV	5.85 \pm 0.94 / 6.08 \pm 0.72 m/s 5.91 \pm 0.7 / 5.82 \pm 0.73 m/s	0.275 -0.126	\leftrightarrow \leftrightarrow
Tai et al. (2018) [64]	16, M	23 \pm 3	G1 (16) G2 (16) G3 (16)	AIx	10 \pm 12 / 45 \pm 23% 15 \pm 17 / 35 \pm 15% 16 \pm 16 / 4 \pm 14%	1.91 1.25 -0.798	\uparrow 35% \uparrow 20% \downarrow 10%

AS – arterial stiffness, M – male, F – female, G1 – experimental group 1, G2 – intervention group 2 / control, G3 – control group, aPWV – aortic pulse wave velocity, AIx – augmentation index, cfPWV – carotid-femoral pulse wave velocity

The participants’ chronic and acute outcomes are described in Tables 2 and 3, respectively. Furthermore, a detailed list of acute and long-term RT interventions is presented (Table 4).

Of note, the measurement of AS in all the included studies (Tables 2 and 3) was preceded by a period of complete rest in supine position.

Study quality

The minimum score (from a possible maximum of 15) in the quality assessment was 11 points, and the maximum score was 14 points, with the mean value of all studies of 12.58 \pm 0.82 points (Table 5). The main systematic drawback was not reporting whether the participant’s status was unknown to the outcome assessors (16 studies out of 19).

Long-term effects

A total of 12 studies provided data for AS ($n = 413$). RT did not significantly affect AS ($ES = -0.07$; 95% CI: -0.59 to 0.45; $p = 0.789$; $I^2 = 84.0\%$; Egger’s test $p = 0.002$; Figure 2). When adjusted in accordance with Duval and Tweedie’s method, the results switched

to $ES = -0.40$; 95% CI: -0.99 to 0.19. Each study analysed had a relative weight ranging from 4.3% to 5.7%.

No AS differences ($p = 0.079$) between the groups were found for RT programs applied in participants with a mean age of > 22 years (12 experimental groups; $ES = -0.41$; 95% CI: -1.22 to 0.40; between-group $I^2 = 88.5\%$) compared with under-22-year-old participants (7 experimental groups; $ES = 0.42$; 95% CI: -0.03 to 0.87; between-group $I^2 = 50.4\%$).

In addition, AS did not significantly differ ($p = 0.979$) between RT protocols applied in male participants (6 experimental groups; $ES = 0.02$; 95% CI: -1.17 to 1.21; between-group $I^2 = 88.2\%$) and in female participants (3 experimental groups; $ES = -0.12$; 95% CI: -0.97 to 0.72; between-group $I^2 = 67.5\%$) or mixed (10 experimental groups; $ES = -0.12$; 95% CI: -0.86 to 0.62; between-group $I^2 = 85.8\%$).

Moreover, AS did not differ ($p = 0.312$) between AS measurements incorporating baPWV techniques (7 experimental groups; $ES = -0.49$; 95% CI: -1.63 to 0.65; between-group $I^2 = 89.2\%$) and cfPWV techniques (9 experimental groups; $ES = 0.21$; 95% CI: -0.53 to 0.95; between-group $I^2 = 84.3\%$).

AS did not significantly differ ($p = 0.051$) in RT programs applied over < 12 weeks (10 experimental

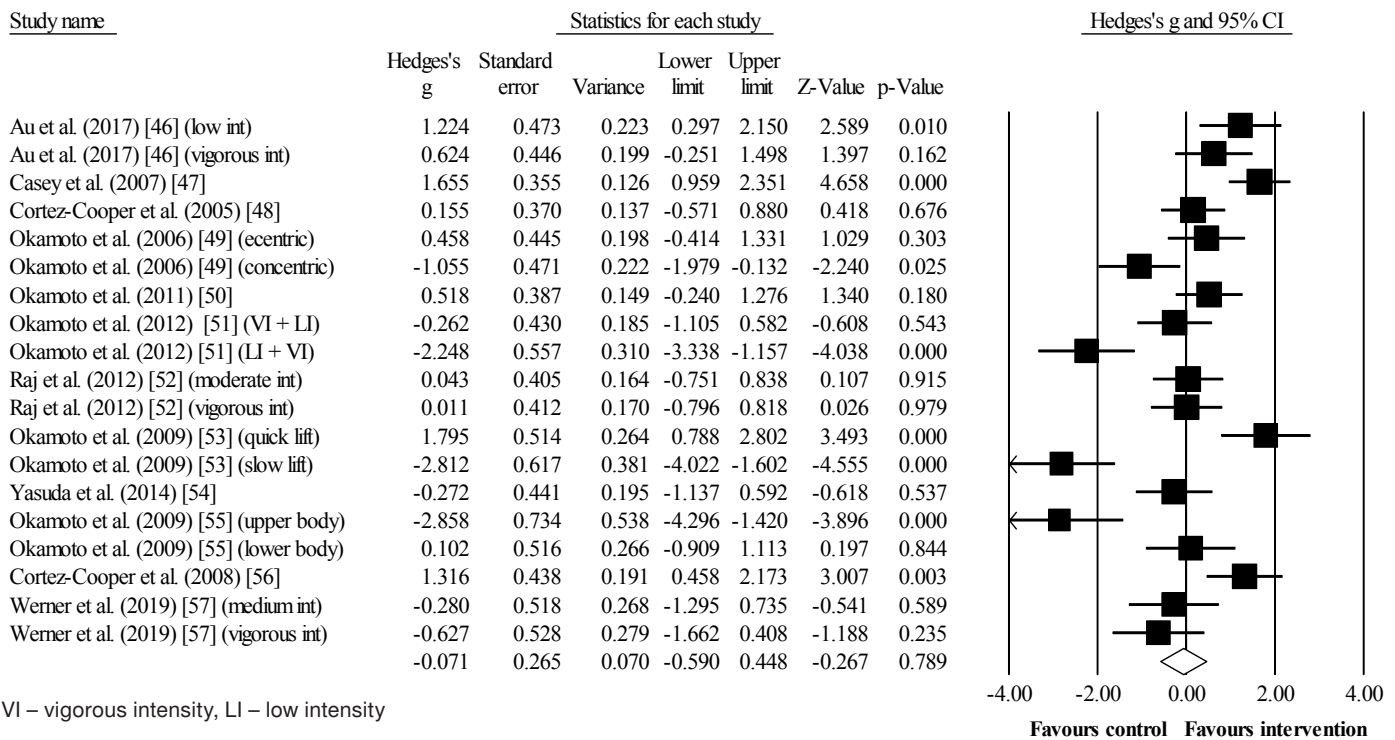
Table 4. Training intervention details

Authors (year)	Weeks	Days/ week	Training type	Exercises	Sets	Repetitions	Intensity	Rest interval (s)
Chronic effects								
Au et al. (2017) [46]	12	2	CB FB [†] / CB FB [‡] / NS [§]	5	3	20–25 [†] / 8–12 [‡]	30–50% RM [†] / 75–90% RM [‡]	60
Casey et al. (2007) [47]	12	3	MA FB [†] / NS [‡]	7	2	8–12	To failure	NR
Cortez-Cooper et al. (2005) [48]	11	4	CB FB [†] / NS [‡]	12	3–6	5–10	To failure	NR
Okamoto et al. (2006) [49]	8	3	FW UT ET [†] / FW UT CT [‡] / NS [§]	1	5	10	100% RM [†] / 75% MVC [‡]	NR
Okamoto et al. (2011) [50]	10	2	CB FB [†] / NS [‡]	8	5	10	50%	30
Okamoto et al. (2012) [51]	10	2	CB FB [†] / CB FB [‡] / NS [§]	5	2–3	10–12	50% & 80% RM [†] / 80% & 50% RM [‡]	30–120
Raj et al. (2012) [52]	16	2	MA FB ET [†] / MA FB CT [‡]	4	2 [†] / 3 [‡]	10 [†] / 5–10 [‡]	75% RM [†] / 50–100% RM [‡]	180
Okamoto et al. (2009) [53]	10	2	CB FB QL [†] / CB FB SL [‡] / NS [§]	6	5	8–10	80% RM	NR
Yasuda et al. (2014) [54]	12	2	MA LT [†] / NS [‡]	2	4	30 [†] / 10 [‡]	20% RM [†] / 30% RM [‡]	90
Okamoto et al. (2009) [55]	10	2	CB UT [†] / CB LT [‡] / NS [§]	5	5	8–10	80% RM	120
Cortez-Cooper et al. (2008) [56]	13	3	MA FB [†] / NS [‡]	10	3	8–12	70% RM	120–180
Werner et al. (2019) [57]	12	3	CB FB [†] / CB FB [‡] / NS [§]	9	3–4 [†] / 2–3 [‡]	10–15 [†] / 3–8 [‡]	50–70% RM [†] / 80–90% RM [‡]	NR
Acute effects								
Palmiere et al. (2018) [58]	-	-	FW UT [†] / NS [‡]	2	5	5–10	5–10 RM	90
Kingsley et al. (2017) [59]	-	-	FW FB [†] / NS [‡]	3	3	10	75% RM	120
Lefferts et al. (2015) [60]	-	-	FW UT [†] / NS [‡]	2	5	5	To failure	90
Augustine et al. (2014) [61]	-	-	FW UT [†] / NS [‡]	2	5	5	To failure	180–300
Okamoto et al. (2014) [62]	-	-	FW UT [†] / NS [‡]	1	3	To exhaustion	40% RM	120
Yoon et al. (2010) [63]	-	-	CB FB [†] / NS [‡]	8	2	15	60% RM	NR
Tai et al. (2018) [64]	-	-	FW UT [†] / FW UT [‡] / NS [§]	1	4	15–30 [†] / 8 [‡]	30% [†] / 70% [‡]	30 [†] / 60 [‡]

CB – combined, FB – full body, NS – no strength, MA – machines, FW – free weights, UT – upper body training, ET – eccentric, LT – lower body training, CT – concentric, QL – quick lift, SL – slow lift, RM – maximum load in 1 repetition, MVC – maximal voluntary contraction, NR – not reported
[†] intervention group 1, [‡] intervention group 2, [§] group 3 (see Table 2)

Table 5. Methodological quality assessment of the studies in accordance with TESTEX [35]

Authors (year)	1	2	3	4	5	6	7	8	9	10	11	12	Total
Chronic effects													
Au et al. (2017) [46]	1	1	1	1	-	2	1	2	1	1	1	1	13
Casey et al. (2007) [47]	1	-	1	1	1	2	1	2	1	-	1	1	12
Cortez-Cooper et al. (2005) [48]	1	-	1	1	-	2	1	2	1	1	1	1	12
Okamoto et al. (2006) [49]	1	1	1	1	-	2	1	2	1	-	1	1	12
Okamoto et al. (2011) [50]	1	1	1	1	1	3	1	2	1	-	1	1	14
Okamoto et al. (2012) [51]	1	1	1	1	-	3	1	2	1	-	1	1	13
Raj et al. (2012) [52]	1	1	1	1	-	1	1	2	1	1	1	1	12
Okamoto et al. (2009) [53]	1	1	1	1	1	3	1	2	1	-	1	1	14
Yasuda et al. (2014) [54]	1	1	1	1	-	2	1	2	1	1	1	1	13
Okamoto et al. (2009) [55]	1	1	1	1	-	2	1	2	1	1	1	1	13
Cortez-Cooper et al. (2008) [56]	1	1	1	1	-	3	1	1	1	-	1	1	12
Werner et al. (2019) [57]	1	1	1	-	-	3	1	2	1	1	1	1	13
Acute effects													
Palmiere et al. (2018) [58]	1	1	1	1	-	3	1	1	1	-	1	1	12
Kingsley et al. (2017) [59]	1	1	1	1	-	3	1	2	1	1	1	1	14
Lefferts et al. (2015) [60]	1	1	1	1	-	1	1	2	1	1	1	1	12
Augustine et al. (2014) [61]	1	1	-	1	-	2	1	2	1	1	1	1	12
Okamoto et al. (2014) [62]	1	1	1	1	-	2	1	2	1	1	1	1	13
Yoon et al. (2010) [63]	1	1	1	1	-	2	1	2	1	-	-	1	11
Tai et al. (2018) [64]	1	1	-	1	-	2	1	2	1	1	1	1	12



VI – vigorous intensity, LI – low intensity

Figure 2. Forest plot of long-term changes in arterial stiffness in healthy participants after resistance training compared with control condition (rest). Black boxes: individual study groups. White diamond: overall results. The relative weight of each study is indicated by the size of the plotted box

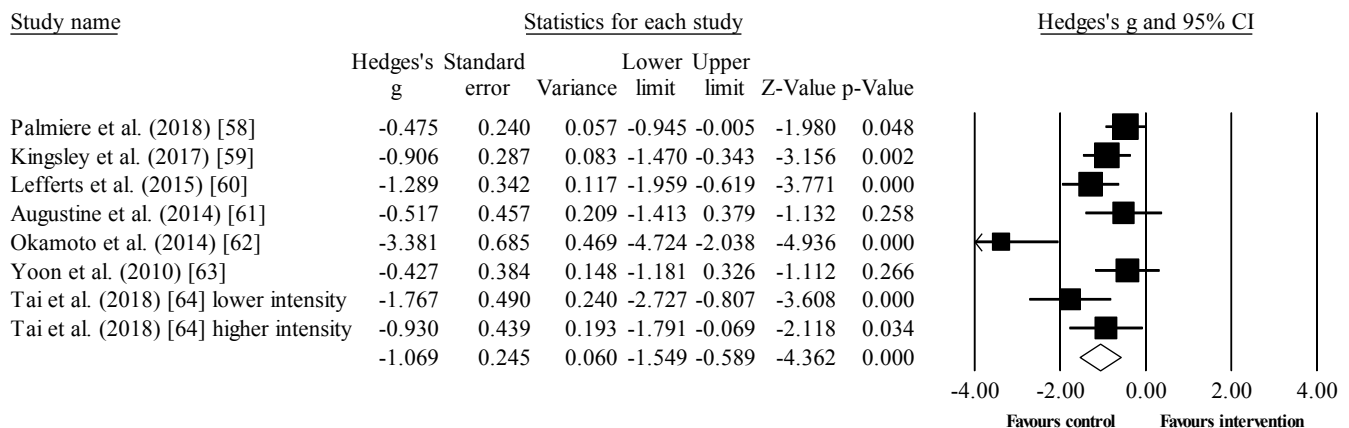


Figure 3. Forest plot of acute changes in arterial stiffness in healthy participants after resistance training compared with control condition (rest). Black boxes: individual study groups. White diamond: overall results. The relative weight of each study is indicated by the size of the plotted box

groups; $ES = -0.56$; 95% CI: -1.39 to 0.28 ; between-group $I^2 = 87.1\%$) and over > 12 weeks (9 experimental groups; $ES = 0.44$; 95% CI: -0.11 to 0.99 ; between-group $I^2 = 72.4\%$).

Similarly, AS did not differ ($p = 0.303$) for RT protocols with 2 sessions per week (12 experimental groups; $ES = -0.28$; 95% CI: -0.99 to 0.43 ; between-group $I^2 = 85.4\%$) and with > 2 sessions per week (7 experimental groups; $ES = 0.27$; 95% CI: -0.49 to 1.02 ; between-group $I^2 = 81.5\%$).

Furthermore, AS did not change ($p = 0.531$) between RT programs incorporating low or medium intensities (5 experimental groups; $ES = 0.25$; 95% CI: -0.27 to 0.78 ; between-group $I^2 = 46.2\%$) and those involving vigorous intensity (12 experimental groups; $ES = -0.04$; 95% CI: -0.77 to 0.70 ; between-group $I^2 = 87.1\%$).

In addition, AS was not significantly different ($p = 0.105$) in RT programs incorporating machine-based exercise (5 experimental groups; $ES = 0.56$; 95% CI: -0.22 to 1.35 ; between-group $I^2 = 79.3\%$) and those with a combination of machine and free weights (12 experimental groups; $ES = -0.33$; 95% CI: -1.06 to 0.41 ; between-group $I^2 = 85.7\%$).

Finally, AS did not significantly differ ($p = 0.423$) between RT programs involving lower-body drills (2 experimental groups; $ES = -0.11$; 95% CI: -0.77 to 0.54 ; between-group $I^2 = 0.0\%$) and upper-body drills (3 experimental groups; $ES = -1.07$; 95% CI: -2.78 to 0.64 ; between-group $I^2 = 87.5\%$) or full-body drills (14 experimental groups; $ES = 0.12$; 95% CI: -0.48 to 0.73 ; between-group $I^2 = 85.0\%$).

Acute effects

Seven studies provided data for AS ($n = 274$). RT significantly increased AS ($ES = -1.07$; 95% CI: -1.55 to -0.59 ; $p < 0.001$; $I^2 = 69.4\%$; Egger's test $p = 0.053$; Figure 3). Each study analysed had a relative weight ranging from 7.7% to 16.2%.

AS did not acutely differ ($p = 0.248$) for RT sessions applied in participants with a mean age of < 23 years (5 experimental groups; $ES = -0.81$; 95% CI: -1.21 to 0.41 ; between-group $I^2 = 41.2\%$) compared with subjects aged > 23 years (3 experimental groups; $ES = -1.63$; 95% CI: -2.96 to -0.30 ; between-group $I^2 = 83.5\%$).

Likewise, AS was not considerably different ($p = 0.391$) between RT sessions applied in male participants (4 experimental groups; $ES = -0.87$; 95% CI: -1.44 to -0.30 ; between-group $I^2 = 43.1\%$) and in males and females combined (4 experimental groups; $ES = -1.31$; 95% CI: -2.12 to -0.49 ; between-group $I^2 = 82.9\%$).

Additionally, AS was not different ($p = 0.230$) for low- or medium-intensity RT sessions (3 experimental groups; $ES = -1.78$; 95% CI: -3.36 to -0.20 ; between-group $I^2 = 86.9\%$) in comparison with vigorous ones (5 experimental groups; $ES = -0.79$; 95% CI: -1.10 to -0.49 ; between-group $I^2 = 10.7\%$).

Finally, AS did not differ ($p = 0.203$) when upper-body RT sessions (6 experimental groups; $ES = -1.26$; 95% CI: -1.93 to -0.59 ; between-group $I^2 = 76.4\%$) were compared with full-body ones (2 experimental groups; $ES = -0.74$; 95% CI: -1.19 to -0.28 ; between-group $I^2 = 0.0\%$).

Discussion

The aim of the present meta-analysis was to evaluate both the long-term and acute effects of RT on AS among healthy participants. The main findings indicate that RT keeps AS stable over a long period, meaning that it seems to be a safe way of training for healthy individuals, regardless of their age or gender, or training characteristics such as type, duration, frequency, and intensity. However, a transitory increase of AS following RT bouts was found as an immediate effect, although this impact does not appear to be clinically significant enough to hesitate about RT effects on cardiovascular health.

From 647 articles retrieved in the electronic database searching, 19 quality-based studies were included, involving 626 participants. The main findings imply that RT did not provoke chronic increases of AS ($ES = -0.07$). This outcome differs from those in previous research [31] that revealed considerable increases (10.7%), but it corroborates a more recent work [29] which did not report such changes (mean difference: -1.33 cm/s, $p = 0.94$). Indeed, RT does not impair cardiovascular health by chronically increasing AS values to a dangerous level. This observation could help to keep building evidence on the appropriateness of including RT in exercise protocols for health. However, this study showed that AS was significantly increased in participants performing an acute bout of RT ($ES = 1.07$) compared with control groups. Since no meta-analysis has been previously performed on that topic, it clarifies a quite unsettled issue, as previous papers [65] and reviews [30] notified.

No moderator factor (i.e., age, gender, RT intensity) disturbed AS either in an acute or in a chronic way. Regarding the participants' age, our findings indicate that there were no significant subgroup differences for long-term or acute effects. This differs from other results [31] that implied significant long-term increases in AS among young participants (14.3%) but not in their older counterparts (> 40 years). That means that RT is as safe for elders as it has been proved to be for young subjects. Gender, on the contrary, has been paid too little attention. In an attempt to give some insight into this aspect, we warn that more intervention groups presented a gender-mixed sample ($n = 10$) compared with male ($n = 6$) and female ($n = 3$) in long-term interventions, whereas no exclusively female groups were available in acute-effect studies (male, $n = 4$; mixed, $n = 4$). The predominance of gender-mixed experimental groups makes gender analysis difficult. Hence, more exclusively female groups in both short-

and long-term interventions are needed to establish whether gender has a decisive influence on the effects of RT on AS or not. In turn, other training factors such as frequency, duration, and kind of training have been shown to have no influence on AS behaviour after RT, in both long and short terms. On the one hand, as it has been stated that a 4-week period with a frequency of 2 days weekly is the shortest stimulus necessary for structural and neuronal adaptations to occur [33], studies with shorter frequencies or durations were excluded, since no arterial adaptations could have been expected. Further, with the present results, we acknowledged that longer durations and higher frequencies did not induce any differences in cardiovascular adaptations compared with shorter or less frequent interventions. On the other hand, before making overall conclusions about the ineffectiveness of applying a specific kind of training program, it should be underlined that some kinds of training did not provide enough data to be analysed (i.e., free-weight RT), so they should be used with caution in risk subjects before stronger evidence is gathered. Namely, consistent acute increases in AS could be influenced by the incorporation of free-weight protocols as a confounder factor. In fact, all acute-effect groups used free-weight protocols, with 1 exception [63], whereas just 1 [49] performed this kind of training in a long-term way. Therefore, we encourage further research to focus on the different kinds of training used in a systematic way, in order to resolve the existing doubts.

Major health organizations recommend RT [25–27]. However, previous research warned that high-intensity RT might promote arterial changes leading to arterial stiffening [48, 49, 66]. Indeed, intermittent increases in blood pressure occur during vigorous RT bouts, up to 320/250 mm Hg [67]. These acute raises affect vessel properties [31]. As a result, the vessel walls cope with an elevated stress, higher AS, and increased blood pressure [67], which might be detrimental to cardiovascular health by damaging the structure of the arteries [8]. Indeed, previous meta-analyses showed long-term AS increases in response to high-intensity RT interventions [29, 31]. Nevertheless, the present work revealed no differences in AS response to RT, either after acute low- or medium-intensity RT ($ES = -1.78$) compared with vigorous-intensity RT ($ES = -0.79$), or after long-term low- or medium-intensity RT ($ES = 0.25$) vs. vigorous-intensity RT ($ES = -0.04$). Therefore, despite the fear that high-intensity RT has always provoked, the results of this study show that it does not imply any increase in AS and that it would therefore be safe for cardiovascular health to apply it

in subjects of all ages, with the necessary progression and adaptation processes. Only specific groups [9, 49, 57] (G2, G1 and G1, respectively) experienced considerable AS increases compared with lower-intensity counterparts, and these were equally distributed among different intensity subgroups. It has been maintained that using exclusively upper limbs might compromise cardiovascular health by increasing AS, as blood noradrenaline concentration raises, causing a vasoconstrictive effect [55]. We identified that 4 out of the 6 groups [55, 60, 64] that experienced AS increases greater than 10% having performed high-intensity programs underwent upper body training exclusively; 3 of them [60, 64] were the only groups that experienced those changes in acute-effect studies. It is of crucial importance to emphasize that the mean relative acute increase in AS was only 4.73% (increase in PWV or AIx) (clinically significant change of 10%) [6]. It is therefore essential to define whether this increase can be considered as threatening to cardiovascular health or not. In that sense, according to Mattace-Raso et al. [10], such levels of AS raise should not be clinically dangerous in adults with low baseline AS levels. However, more studies are definitely warranted in order to check the extent and hazardousness of these AS increases. Among some other considerations, 1 intervention group [51] was not included in our analysis for combining vigorous and low intensity in both intervention programs. These protocols reflect the possible role of low-intensity bouts after high-intensity exercising to buffer the cardiovascular effects, as previous research already proposed to be true with low-intensity aerobic training [49].

Some limitations need to be mentioned. Even if the number of studies is larger than in previous meta-analyses and should be encouraging to draw more robust conclusions, some subgroup analyses were precluded owing to a limited number (i.e., less than 3) of studies available. Secondly, the present results are true for healthy populations and should not be extrapolated to non-healthy ones. For that, analysing pathological populations would broaden the knowledge provided by this meta-analysis. Thirdly, not properly applied or described protocols were at times a result of training programs designed by clinicians, and not coaches. These inconsistencies in exercise prescription might compromise the obtained results. Finally, current observations should be considered with caution owing to moderate-high heterogeneity ($I^2 = 84.0\%$, long-term effects; $I^2 = 69.4\%$, acute effects), probably arising from different RT protocols or different AS assessment protocols.

Conclusions

Long-term RT programs seem safe for healthy individuals, without chronic increases in AS. They appear applicable irrespective of the age and gender of the participants, the assessment protocol for AS, and some RT configurations such as duration, frequency, type of training (e.g., free weights, machine-based, with elastic bands), body part trained (e.g., upper body, lower body), and intensity. However, acute RT bouts transiently increase AS, although the clinical relevance of such increases is unclear.

Practical applications

As long-term RT did not affect AS, it may be suggested that its use may provide well-proved benefits in physical fitness and health, without undue alterations in AS. This may be applicable independently of the subjects' age (i.e., < 22 vs. > 22 years) or sex, or RT program total duration (i.e., < or > 12 weeks). Since RT of any programming variables (e.g., frequency, duration, intensity) induces no harm to AS, different protocols (e.g., low-intensity, high-intensity) may be used safely, in accordance with the individual's needs and preferences. In that sense, some kinds of training, like high-intensity, positive in many health-related aspects, can be applied without hesitation, which clarifies a topic that was still under question. Full-body RT protocols, with free weights, machines, body weight, elastic bands, or related equipment, may provide well-known physical fitness and health benefits, and our findings suggest that such RT protocols will not increase AS. Moreover, we encourage to use high-, moderate-, or low-intensity RT without fear, despite the subjects' gender and age. We only warn that more evidence is needed for the possible acute AS increases provoked by certain protocols. However, this meta-analysis clarifies that, to date, in the context of safety and cardiovascular health prevention, there is no reason or evidence to restrict any kind of RT training owing to short-term AS increases.

Funding

The present work did not receive any funding.

Disclosure statement

No author has any financial interest or received any financial benefit from this research.

Conflict of interest

The authors state no conflict of interest.

References

1. Townsend RR, Wilkinson IB, Schiffrin EL, Avolio AP, Chirinos JA, Cockcroft JR, et al. Recommendations for improving and standardizing vascular research on arterial stiffness. *Hypertension*. 2015;66(3):698–722; doi: 10.1161/HYP.0000000000000033.
2. Monahan KD, Tanaka H, Dinunno FA, Seals DR. Central arterial compliance is associated with age- and habitual exercise-related differences in cardiovascular baroreflex sensitivity. *Circulation*. 2001;104(14):1627–1632; doi: 10.1161/hc3901.096670.
3. O'Rourke M. Arterial stiffness, systolic blood pressure, and logical treatment of arterial hypertension. *Hypertension*. 1990;15(4):339–347; doi: 10.1161/01.HYP.15.4.339.
4. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. 2007 guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Eur Heart J*. 2007;28(12):1462–1536; doi: 10.1093/eurheartj/ehm236.
5. Ben-Shlomo Y, Spears M, Boustred C, May M, Anderson SG, Benjamin EJ, et al. Aortic pulse wave velocity improves cardiovascular event prediction: an individual participant meta-analysis of prospective observational data from 17,635 subjects. *J Am Coll Cardiol*. 2014; 63(7):636–646; doi: 10.1016/j.jacc.2013.09.063.
6. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. *J Am Coll Cardiol*. 2010;55(13):1318–1327; doi: 10.1016/j.jacc.2009.10.061.
7. Thiebaud RS, Fahs CA, Rossow LM, Loenneke JP, Kim D, Mouser JG, et al. Effects of age on arterial stiffness and central blood pressure after an acute bout of resistance exercise. *Eur J Appl Physiol*. 2016;116(1): 39–48; doi: 10.1007/s00421-015-3242-5.
8. Dernellis J, Panaretou M. Aortic stiffness is an independent predictor of progression to hypertension in non-hypertensive subjects. *Hypertension*. 2005;45(3):426–431; doi: 10.1161/01.HYP.0000157818.58878.93.
9. Liao D, Arnett DK, Tyroler HA, Riley WA, Chambless LE, Szklo M, et al. Arterial stiffness and the development of hypertension. The ARIC study. *Hypertension*. 1999; 34(2):201–206; doi: 10.1161/01.hyp.34.2.201.
10. Mattace-Raso FUS, van den Meiracker AH, Bos WJ, van der Cammen TJM, Westerhof BE, Elias-Smale S, et al. Arterial stiffness, cardiovascular baroreflex sensitivity and postural blood pressure changes in older adults: the Rotterdam Study. *J Hypertens*. 2007;25(7):1421–1426; doi: 10.1097/HJH.0b013e32811d6a07.
11. Najjar SS, Scuteri A, Shetty V, Wright JG, Muller DC, Fleg JL, et al. Pulse wave velocity is an independent predictor of the longitudinal increase in systolic blood pressure and of incident hypertension in the Baltimore Longitudinal Study of Aging. *J Am Coll Cardiol*. 2008; 51(14):1377–1383; doi: 10.1016/j.jacc.2007.10.065.
12. Belz GG. Elastic properties and Windkessel function of the human aorta. *Cardiovasc Drugs Ther*. 1995;9: 73–83; doi: 10.1007/BF00877747.
13. The Reference Values for Arterial Stiffness' Collaboration. 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(19):2338–2350; doi: 10.1093/eurheartj/ehq165.
14. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J*. 2006;27(21):2588–2605; doi: 10.1093/eurheartj/ehl254.
15. Taylor RS, Brown A, Ebrahim S, Jolliffe J, Noorani H, Rees K, et al. Exercise-based rehabilitation for patients with coronary heart disease: systematic review and meta-analysis of randomized controlled trials. *Am J Med*. 2004;116(10):682–692; doi: 10.1016/j.amjmed.2004.01.009.
16. Thompson PD, Buchner D, Piña IL, Balady GJ, Williams MA, Marcus BH, et al. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: a statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Circulation*. 2003; 107(24):3109–3116; doi: 10.1161/01.CIR.0000075572.40158.77.
17. Figueiredo T, Rhea MR, Peterson M, Miranda H, Bentes CM, Machado De Ribeiro dos Reis V, et al. Influence of number of sets on blood pressure and heart rate variability after a strength training session. *J Strength Cond Res*. 2015;29(6):1556–1563; doi: 10.1519/JSC.0000000000000774.
18. Karavirta L, Häkkinen A, Sillanpää E, García-López D, Kauhanen A, Haapasaari A, et al. Effects of combined endurance and strength training on muscle strength, power and hypertrophy in 40–67-year-old men. *Scand J Med Sci Sports*. 2011;21(3):402–411; doi: 10.1111/j.1600-0838.2009.01059.x.
19. Smutok MA, Reece C, Kokkinos PF, Farmer C, Dawson P, Shulman R, et al. Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. *Metabolism*. 1993; 42(2):177–184; doi: 10.1016/0026-0495(93)90032-j.
20. Marques EA, Wanderley F, Machado L, Sousa F, Viana JL, Moreira-Gonçalves D, et al. Effects of resistance and aerobic exercise on physical function, bone mineral density, OPG and RANKL in older women. *Exp Gerontol*. 2011;46(7):524–532; doi: 10.1016/j.exger.2011.02.005.
21. Baldi JC, Snowling N. Resistance training improves glycaemic control in obese type 2 diabetic men. *Int J*

- Sports Med. 2003;24(6):419–423; doi: 10.1055/s-2003-41173.
22. Bweir S, Al-Jarrah M, Almalty A-M, Maayah M, Smirnova IV, Novikova L, et al. Resistance exercise training lowers HbA1c more than aerobic training in adults with type 2 diabetes. *Diabetol Metab Syndr*. 2009;1:27; doi: 10.1186/1758-5996-1-27.
 23. Hanson ED, Sheaff AK, Sood S, Ma L, Francis JD, Goldberg AP, et al. Strength training induces muscle hypertrophy and functional gains in Black prostate cancer patients despite androgen deprivation therapy. *J Gerontol A Biol Sci Med Sci*. 2013;68(4):490–498; doi: 10.1093/gerona/gls206.
 24. Rodrigues Matos A, Siqueira Ribeiro H, De Luca Corrêa H, Pimentel Ferreira A, Vieira E. Strength training promotes anthropometric and functional benefits in sedentary subjects: does a personal trainer matter? *Hum Mov*. 2021;22(4):20–27; doi: 10.5114/hm.2021.103286.
 25. Haskell WL, Lee M, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc*. 2007;39(8):1423–1434; doi: 10.1249/mss.0b013e3180616b27.
 26. Nelson ME, Rejeski WJ, Blair SN, Duncan PW, Judge JO, King AC, et al. Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association. *Circulation*. 2007;116(9):1094–1105; doi: 10.1161/CIRCULATIONAHA.107.185650.
 27. Williams MA, Haskell WL, Ades PA, Amsterdam EA, Bittner V, Franklin BA, et al. Resistance exercise in individuals with and without cardiovascular disease: 2007 update: a scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2007;116(5):572–584; doi: 10.1161/CIRCULATIONAHA.107.185214.
 28. Evans W, Willey Q, Hanson ED, Stoner L. Effects of resistance training on arterial stiffness in persons at risk for cardiovascular disease: a meta-analysis. *Sports Med*. 2018;48(12):2785–2795; doi: 10.1007/s40279-018-1001-6.
 29. Ceciliato J, Costa EC, Azevêdo L, Sousa JC, Fecchio RY, Brito LC. Effect of resistance training on arterial stiffness in healthy subjects: a systematic review and meta-analysis. *Curr Hypertens Rep*. 2020;22(8):51; doi: 10.1007/s11906-020-01065-x.
 30. García-Mateo P, García-de-Alcaraz A, Rodríguez-Pérez MA, Alcaraz-Ibáñez M. Effects of resistance training on arterial stiffness in healthy people: a systematic review. *J Sports Sci Med*. 2020;19(3):444–451; PMID: PMC7429424.
 31. Miyachi M. Effects of resistance training on arterial stiffness: a meta-analysis. *Br J Sports Med*. 2013;47(6):393–396; doi: 10.1136/bjsports-2012-090488.
 32. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *Int J Surg*. 2021;88:105906; doi: 10.1016/j.ijssu.2021.105906.
 33. Kraemer WJ, Ratamess NA. Fundamentals of resistance training: progression and exercise prescription. *Med Sci Sports Exerc*. 2004;36(4):674–688; doi: 10.1249/01.mss.0000121945.36635.61.
 34. Drevon D, Fursa SR, Malcolm AL. Inter-coder reliability and validity of WebPlotDigitizer in extracting graphed data. *Behav Modif*. 2017;41(2):323–339; doi: 10.1177/0145445516673998.
 35. Smart NA, Waldron M, Ismail H, Giallauria F, Vigorito C, Cornelissen V, et al. Validation of a new tool for the assessment of study quality and reporting in exercise training studies: TESTEX. *Int J Evid Based Healthc*. 2015;13(1):9–18; doi: 10.1097/XEB.000000000000020.
 36. Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee I-M, et al. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Med Sci Sports Exerc*. 2011;43(7):1334–1359; doi: 10.1249/MSS.0b013e318213efb.
 37. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc*. 2009;41(1):3–12; doi: 10.1249/MSS.0b013e31818cb278.
 38. Higgins JPT, Deeks JJ, Altman DG. Special topics in statistics. In: Higgins JPT, Green S (eds.), *Cochrane handbook for systematic reviews of interventions*. Chichester: John Wiley and Sons; 2008; 481–529.
 39. Higgins JPT, Thompson SG. Quantifying heterogeneity in a meta-analysis. *Stat Med*. 2002;21(11):1539–1558; doi: 10.1002/sim.1186.
 40. Moran J, Sandercock GRH, Ramírez-Campillo R, Meylan C, Collison J, Parry DA. A meta-analysis of maturation-related variation in adolescent boy athletes' adaptations to short-term resistance training. *J Sports Sci*. 2017;35(11):1041–1051; doi: 10.1080/02640414.2016.1209306.
 41. Moran J, Sandercock G, Ramirez-Campillo R, Clark CCT, Fernandes JFT, Drury B. A meta-analysis of resistance training in female youth: its effect on muscular strength, and shortcomings in the literature. *Sports Med*. 2018;48(7):1661–1671; doi: 10.1007/s40279-018-0914-4.
 42. Moran J, Clark CCT, Ramirez-Campillo R, Davies MJ, Drury B. A meta-analysis of plyometric training in female youth: its efficacy and shortcomings in the literature. *J Strength Cond Res*. 2019;33(7):1996–2008; doi: 10.1519/JSC.0000000000002768.
 43. Egger M, Smith GD, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ*. 1997;315(7109):629–634; doi: 10.1136/bmj.315.7109.629.

44. Duval S, Tweedie R. Trim and fill: a simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics*. 2000;56(2): 455–463; doi: 10.1111/j.0006-341x.2000.00455.x.
45. Shi L, Lin L. The trim-and-fill method for publication bias: practical guidelines and recommendations based on a large database of meta-analyses. *Medicine*. 2019; 98(23):e15987; doi: 10.1097/MD.00000000000015987.
46. Au JS, Oikawa SY, Morton RW, Macdonald MJ, Phillips SM. Arterial stiffness is reduced regardless of resistance training load in young men. *Med Sci Sports Exerc*. 2017;49(2):342–348; doi: 10.1249/0000000000001106.
47. Casey DP, Beck DT, Braith RW. Progressive resistance training without volume increases does not alter arterial stiffness and aortic wave reflection. *Exp Biol Med*. 2007;232(9):1228–1235; doi: 10.3181/0703-RM-65.
48. Cortez-Cooper MY, DeVan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS, et al. Effects of high intensity resistance training on arterial stiffness and wave reflection in women. *Am J Hypertens*. 2005;18(7):930–934; doi: 10.1016/j.amjhyper.2005.01.008.
49. Okamoto T, Masuhara M, Ikuta K. Effects of eccentric and concentric resistance training on arterial stiffness. *J Hum Hypertens*. 2006;20(5):348–354; doi: 10.1038/sj.jhh.1001979.
50. Okamoto T, Masuhara M, Ikuta K. Effect of low-intensity resistance training on arterial function. *Eur J Appl Physiol*. 2011;111(5):743–748; doi: 10.1007/s00421-010-1702-5.
51. Okamoto T, Masuhara M, Ikuta K. Low-intensity resistance training after high-intensity resistance training can prevent the increase of central arterial stiffness. *Int J Sports Med*. 2012;34(5):385–390; doi: 10.1055/s-0032-1312604.
52. Raj IS, Bird SR, Westfold BA, Shield AJ. Effects of eccentrically biased versus conventional weight training in older adults. *Med Sci Sports Exerc*. 2012;44(6):1167–1176; doi: 10.1249/MSS.0b013e3182442ecd.
53. Okamoto T, Masuhara M, Ikuta K. Effects of muscle contraction timing during resistance training on vascular function. *J Hum Hypertens*. 2009;23(7):470–478; doi: 10.1038/jhh.2008.152.
54. Yasuda T, Fukumura K, Fukuda T, Uchida Y, Iida H, Meguro M, et al. Muscle size and arterial stiffness after blood flow-restricted low-intensity resistance training in older adults. *Scand J Med Sci Sports*. 2014;24(5): 799–806; doi: 10.1111/sms.12087.
55. Okamoto T, Masuhara M, Ikuta K. Upper but not lower limb resistance training increases arterial stiffness in humans. *Eur J Appl Physiol*. 2009;107(2):127–134; doi: 10.1007/s00421-009-1110-x.
56. Cortez-Cooper MY, Anton MM, Devan AE, Neidre DB, Cook JN, Tanaka H. The effects of strength training on central arterial compliance in middle-aged and older adults. *Eur J Cardiovasc Prev Rehabil*. 2008;15(2):149–155; doi: 10.1097/HJR.0b013e3282f02fe2.
57. Werner TJ, Pellingier TK, Rosette VD, Ortlip AT. Effects of a 12-week resistance training program on arterial stiffness: a randomized controlled trial. *J Strength Cond Res*. 2019;35(12):3281–3287; doi: 10.1519/JSC.0000000000003331.
58. Palmiere S, Wade M, DeBlois J, Lefferts WK, Heffernan KS. Aortic stiffness, central pulse pressure and cognitive function following acute resistance exercise. *Eur J Appl Physiol*. 2018;118(10):2203–2211; doi: 10.1007/s00421-018-3948-2.
59. Kingsley JD, Tai YL, Mayo X, Glasgow A, Marshall E. Free-weight resistance exercise on pulse wave reflection and arterial stiffness between sexes in young, resistance-trained adults. *Eur J Sport Sci*. 2017;17(8):1056–1064; doi: 10.1080/17461391.2017.1342275.
60. Lefferts WK, Hughes WE, Heffernan KS. Effect of acute high-intensity resistance exercise on optic nerve sheath diameter and ophthalmic artery blood flow pulsatility. *J Hum Hypertens*. 2015;29(12):744–748; doi: 10.1038/jhh.2015.12.
61. Augustine J, Tarzia B, Kasprovicz A, Heffernan KS. Effect of a single bout of resistance exercise on arterial stiffness following a high-fat meal. *Int J Sports Med*. 2014;35(11):894–899; doi: 10.1055/s-0033-1363266.
62. Okamoto T, Min S, Sakamaki-Sunaga M. Arterial compliance and stiffness following low-intensity resistance exercise. *Eur J Appl Physiol*. 2014;114(2):235–241; doi: 10.1007/s00421-013-2770-0.
63. Yoon ES, Jung SJ, Cheun SK, Oh YS, Kim SH, Jae SY. Effects of acute resistance exercise on arterial stiffness in young men. *Korean Circ J*. 2010;40(1):16–22; doi: 10.4070/kcj.2010.40.1.16.
64. Tai YL, Marshall EM, Glasgow A, Parks JC, Sensibello L, Kingsley JD. Pulse wave reflection responses to bench press with and without practical blood flow restriction. *Appl Physiol Nutr Metab*. 2018;44(4):341–347; doi: 10.1139/apnm-2018-0265.
65. Nitzsche N, Weigert M, Baumgärtel L, Auerbach T, Schuffenhauer D, Nitzsche R, et al. Acute effects of different strength training protocols on arterial stiffness in healthy subjects. *Int J Sports Sci*. 2016;6(5):195–202; doi: 10.5923/j.sports.20160605.05.
66. Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, et al. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation*. 2004; 110(18):2858–2863; doi: 10.1161/01.CIR.0000146380.08401.99.
67. MacDougall JD. Morphological changes in human skeletal muscle following strength training and immobilization. In: Jones NJ, McCarthey N, McComas AJ (eds.), *Human muscle power*. Champaign: Human Kinetics; 1986; 269–285.

Appendix 1. PRISMA 2020 checklist

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Page 1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Page 2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Page 3
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Page 4
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Page 4
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Page 4
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Page 4
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Page 5
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Page 5
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Page 5
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Page 5
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Page 5
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Page 6
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Page 6
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Page 5
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Page 6
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Page 6
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Page 6
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Page 6
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Page 6
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Page 6

Section and Topic	Item #	Checklist item	Location where item is reported
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Page 6
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Page 6
Study characteristics	17	Cite each included study and present its characteristics.	Page 7
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Page 7
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Page 7
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Pages 8–9
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Pages 8–9
Reporting biases	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Pages 8-9
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Pages 8-9
Certainty of evidence	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Pages 8-9
	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Pages 8-9
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Page 9
	23b	Discuss any limitations of the evidence included in the review.	Pages 11–12
	23c	Discuss any limitations of the review processes used.	Pages 11–12
	23d	Discuss implications of the results for practice, policy, and future research.	Page 12
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	NA
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	NA
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	NA
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Page 12
	26	Declare any competing interests of review authors.	Page 12
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	NA

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ* 2021;372:n71. doi: 10.1136/bmj.n71

For more information, visit: <http://www.prisma-statement.org/>

Appendix 2. Detailed reasons for study exclusion in accordance with PICOS approach

Time of exclusion	Exclusion criteria	Reason for exclusion	<i>n</i>
Duplicates			367
Title and abstract	Participant-related	Population with pathology	41
	Intervention-related	No strength training	27
		Additional intervention	62
		Insufficient duration	3
	Comparator-related	Not applicable	0
	Outcome-related	No arterial stiffness data	21
	Study-design-related	Language other than English	3
		Meta-analyses, reviews, reports, and letters to editor	33
		190	
Full-text	Participant-related	Population with pathology	12
	Intervention-related	Additional intervention	5
	Comparator-related	Non-controlled trials	28
	Outcome-related	Insufficient arterial stiffness data (no PWV or AIx data)	21
	Study-design-related	Commentary	1
		Cross-sectional design	4
		71	
Total			628

PWV – pulse wave velocity, AIx – augmentation index