



Systematic Review

Effects of Dynamic Resistance Training on Arterial Stiffness Assessed by Pulse Wave Velocity: A Systematic Review and Meta-Analysis

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Highlights

Public health relevance—How does this work relate to a public health issue?

- Arterial stiffness is a major predictor of cardiovascular disease and a key target of exercise-based prevention strategies.
- This study clarifies the long-term effects of dynamic resistance training on arterial stiffness across different arterial regions.

Public health significance—Why is this work of significance to public health?

- Dynamic resistance training produced a small, non-significant overall effect on pulse wave velocity, with non-significant segment-specific effects, although the mixed-segment estimate was moderate and imprecise. Thus, the findings indicate neither a statistically significant adverse pooled effect nor consistent improvement in arterial stiffness.
- Exploratory subgroup analyses identified several statistically significant differences; however, the small and highly unbalanced strata and absence of adjustment for multiple testing preclude firm conclusions about moderator effects.

Public health implications—What are the key implications or messages for practitioners, policy makers and/or researchers in public health?

- Resistance training can continue to be prescribed for its well-established musculoskeletal, functional, and metabolic benefits without evidence of a statistically significant adverse pooled vascular effect. However, when performed in isolation, it should not be expected to produce consistent improvements in arterial stiffness.
- Further adequately powered trials should determine whether specific resistance-training configurations produce clinically relevant arterial adaptations and investigate physiological and training determinants beyond conventional FITT-VP descriptors.

Abstract

Arterial stiffness predicts cardiovascular risk, but the chronic effects of dynamic resistance training (RT) remain unclear. This meta-analysis quantified RT effects on arterial stiffness assessed by pulse wave velocity (PWV) and explored moderators. Databases were searched through 26 August 2026. Studies included adults (≥ 19 years) undergoing RT with pre-post PWV data. Random-effects models were used. Fourteen trials included 20 RT interventions



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and 10 controlled comparisons ($n = 379$: 278 RT and 101 control participants; age range 19–63 years; mean age, 33.3 ± 14.3 and 32.4 ± 15.1 years in the RT and control groups, respectively; baseline blood pressure range, 108/64 to 137/90 mmHg). Participants were predominantly physically inactive. Assuming a pre–post correlation of 0.50, pooled effects were non-significant for central PWV ($g = -0.002$; 95% CI, -0.213 to 0.209 ; $p = 0.985$), mixed PWV ($g = -0.548$; 95% CI, -1.168 to 0.071 ; $p = 0.083$), peripheral PWV ($g = -0.110$; 95% CI, -0.436 to 0.217 ; $p = 0.511$), and overall PWV ($g = -0.102$; 95% CI, -0.281 to 0.077 ; $p = 0.265$). Heterogeneity ranged from 43% to 70%. Sensitivity analyses using correlations of 0.30 and 0.70 did not alter statistical significance. Most moderator tests, including those involving participant characteristics and FITT-VP components, were not statistically significant. The few significant subgroup differences were exploratory and arose from small, unbalanced groups without multiplicity adjustment, precluding firm conclusions. Dynamic RT was not associated with a significant change in arterial stiffness; however, imprecision and heterogeneity preclude claims of equivalence or absence of physiological effects. Registration: PROSPERO CRD42022377587.

Keywords: arterial stiffness; resistance training; pulse wave velocity; vascular function; exercise physiology

1. Introduction

Cardiovascular disease remains a leading cause of mortality worldwide, and arterial stiffness is a well-established independent predictor of cardiovascular events and mortality [1,2]. Pulse wave velocity (PWV), particularly carotid–femoral PWV, is considered the gold-standard non-invasive measure of arterial stiffness and provides robust prognostic information beyond traditional risk factors such as blood pressure, diabetes, and dyslipidemia [3,4].

While the beneficial effects of aerobic exercise on arterial stiffness are well established [5–8], the impact of resistance training (RT) remains unclear. Dynamic RT may acutely increase arterial stiffness, possibly due to transient elevations in blood pressure and mechanical stress on arterial walls [6]. However, chronic adaptations are inconsistent, with studies reporting increased [9–13], reduced [14,15], or unchanged PWV [16–18] following RT interventions. This inconsistency has become one of the main unresolved issues in the field, raising the question of why apparently similar RT interventions produce divergent vascular responses.

Whether this heterogeneity reflects true physiological differences in vascular adaptation or merely differences in study populations, exercise prescription, and methodology remains unknown. Meta-analytic approaches provide an opportunity to synthesize these findings and examine potential moderators. However, existing meta-analyses have yielded conflicting results: some reported increased central PWV in younger populations [19], whereas others found no significant changes in healthy or clinical samples [6–8,20,21], and only one observed an overall reduction in PWV [22]. Thus, beyond determining whether RT affects arterial stiffness, an equally important question is whether the observed inconsistencies can be explained by differences in participant characteristics or exercise prescription.

Importantly, most previous analyses did not simultaneously consider individual factors (e.g., nutritional status and resting blood pressure) and training characteristics, such as selected FITT-VP exercise prescription variables. The few studies that addressed these aspects reported inconsistent findings. For example, Miyachi [19] observed increased central arterial stiffness following high-intensity RT ($\geq 70\%$ 1 RM) in young adults, but not

in middle-aged individuals. In contrast, Zhang et al. [22] reported reductions in central PWV following low- to moderate-intensity RT, whereas high-intensity protocols had no effect. Similarly, Saz-Lara et al. [6] found no moderating effects of training duration or frequency, although light-intensity RT (30–50% 1 RM) appeared to reduce arterial stiffness in populations with elevated cardiovascular risk. Collectively, these findings suggest that neither participant characteristics nor conventional descriptors of exercise prescription have yet provided a consistent explanation for the heterogeneity observed across central, mixed, and peripheral arterial segments.

Despite the growing body of evidence, important gaps remain. No previous meta-analysis has directly compared the effects of resistance training across central, mixed, and peripheral arterial segments within a unified framework, as prior studies have either combined data across arterial sites [8,19–23] or focused on specific regions, such as central or peripheral arteries [6,7]. In addition, the combined influence of participant characteristics and exercise prescription variables (FITT-VP) on PWV responses has not been systematically evaluated. Consequently, it remains uncertain whether the inconsistent findings reported in the literature reflect true physiological differences in vascular adaptation or simply methodological and clinical heterogeneity across studies.

Accordingly, the present meta-analysis aimed to (i) quantify the effects of dynamic resistance training on arterial stiffness assessed by PWV across distinct arterial beds in individuals with normal and above-normal blood pressure, and (ii) determine whether differences in participant characteristics, methodological quality, and exercise prescription (FITT-VP) explain the heterogeneity observed in PWV responses across studies.

2. Materials and Methods

2.1. Study Design

This meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [24] and AMSTAR-2 [25] statements. The review protocol was prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO; CRD42022377587), and no amendments were made after registration.

2.2. Eligibility Criteria

Studies were eligible if they: (i) assessed overall, central, mixed, or peripheral pulse wave velocity (PWV) before and after resistance training (RT); (ii) reported at least one component of the FITT-VP framework (frequency, intensity, time, type, volume, or progression); and (iii) provided sufficient data to calculate effect sizes for PWV changes. Studies from all countries were considered. Trials involving participants with cardiovascular disease other than hypertension, or with chronic conditions such as diabetes, stroke, cancer, or HIV, were excluded. Metabolic syndrome without established diabetes was not an exclusion criterion.

To minimize measurement-related heterogeneity, eligible PWV outcomes were restricted to tonometric or Doppler-based measurements obtained at prespecified arterial sites. Oscillometric PWV measurements were excluded because their device-specific waveform-detection and path-length estimation procedures may not be directly comparable with the prespecified methods.

For studies with multiple intervention arms, only exercise and control groups (when available) were included to minimize confounding from non-exercise interventions.

2.3. Search Strategy

PubMed, Web of Science, Scopus (including EMBASE), SportDiscus, and CINAHL were initially searched from inception through September 2025. A bridge search covering

the period from 1 October 2025 to 26 August 2026 was subsequently conducted using the same databases, search strategies, and eligibility criteria. The complete strategy and results for both searches are provided in Text Supplement S1.

The search was restricted to adults (≥ 19 years). Although studies in any language were eligible, all included articles were published in English. Records were screened in three stages (title, abstract, and full text) against predefined eligibility criteria (Text Supplement S2). Two investigators (RC and MO) independently screened titles, abstracts, and full texts, with high agreement (Cohen's $\kappa = 0.82$, $p < 0.0001$). Disagreements were resolved by consensus or, when necessary, through consultation with a third investigator (PF). Study authors were not contacted for additional or missing information, and no automation tools were used in the data collection process.

2.4. Data Extraction and Reliability

Data were independently extracted by two investigators (RC and MO) using a standardized coding form. Agreement was assessed using Cohen's κ for categorical variables and intraclass correlation coefficients (ICC) for continuous variables, indicating high reliability (mean $\kappa = 0.86$; mean ICC = 0.88). Discrepancies were resolved through consensus with a third investigator (PF).

All eligible pre-post PWV outcomes reported by each study were sought and extracted. When a study reported more than one arterial segment, each eligible PWV measure was classified and included in the corresponding central, mixed, or peripheral synthesis, as follows: central (carotid–femoral PWV, cfPWV), mixed (carotid–brachial, carotid–radial, carotid–ankle, or brachial–femoral PWV), and peripheral (brachial–radial, femoral–ankle, or brachial–ankle PWV).

Other extracted variables included study design, methodological quality, sample size, age, sex, baseline blood pressure, physical activity status, health status, PWV assessment method, and RT frequency, intensity, duration, type, volume, and progression. When exercise intensity was not directly reported, it was estimated from the available training parameters according to ACSM guidelines. No assumptions were made for other missing information, which was recorded as not reported.

Baseline blood pressure status was classified according to the 2025 AHA/ACC Guideline [26], with participants categorized as having normal or above-normal blood pressure based on the mean baseline values reported by the original studies. Study samples with mean baseline systolic blood pressure < 120 mmHg and mean diastolic blood pressure < 80 mmHg were categorized as having normal blood pressure, whereas those with mean systolic blood pressure ≥ 120 mmHg or mean diastolic blood pressure ≥ 80 mmHg were categorized as having above-normal blood pressure.

Physical activity status was not assessed using a standardized instrument across studies. Participants were classified according to the descriptions provided in the original reports, such as physically inactive, sedentary, recreationally active, or active. When available, baseline cardiorespiratory fitness (VO_2max) was extracted as supplementary information but was not considered a direct measure of physical activity.

2.5. Effect Size Calculation

Hedges' g was used to quantify changes in PWV within and between RT and control conditions [27]. For non-controlled studies, effect sizes reflected standardized pre–post changes; in controlled trials, they represented the standardized difference in change between RT and control groups. Standard deviations of change were calculated from pre- and post-intervention standard deviations assuming a within-participant correlation of

$r = 0.50$. Values reported as standard errors were converted to standard deviations. Negative values indicated reductions in PWV, whereas positive values indicated increases.

Studies were assigned to the central, mixed, or peripheral PWV synthesis according to the arterial segment assessed. When more than one eligible PWV outcome was reported, each arterial segment contributed separately to its corresponding synthesis. Controlled and non-controlled comparisons were analyzed according to their respective study designs. For trials including multiple intervention arms (e.g., different exercise intensities or training modes), effect sizes were calculated separately for each intervention. When multiple interventions shared a single control group, each comparison was treated as an independent experimental condition because the interventions represented distinct exercise protocols.

Studies without sufficient information to calculate an effect size were excluded, and missing outcome data were not imputed. Values reported as mean $\pm$ SEM were converted to mean $\pm$ SD. Exercise intensity was standardized as a percentage of one-repetition maximum according to ACSM guidelines [28]. Sensitivity analyses repeated the complete calculation using pre–post correlations of $r = 0.30$ and $r = 0.70$ to evaluate the robustness of the findings to the assumed within-participant correlation.

2.6. Study Quality Assessment, Risk of Bias, and Certainty of Evidence

Methodological quality and potential risk of bias were independently assessed by two reviewers (MO and JR) using the TESTEX scale [29]. Disagreements were resolved by consensus with a third reviewer. No automation tools were used. The TESTEX scale includes 12 criteria (maximum score = 15). Studies were classified as high (≥ 12), moderate (7–11), or low quality (≤ 6). Specific criteria were applied for intention-to-treat analysis and maintenance of exercise intensity, requiring either no sample loss or documented progression in training load.

Publication bias was evaluated through funnel plots and Egger’s regression test [30], with confirmation using Duval and Tweedie’s trim-and-fill method [31]. Certainty of evidence was assessed using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) approach [32], considering risk of bias, inconsistency, indirectness, imprecision, and publication bias, and classified as high, moderate, low, or very low.

2.7. Statistical Analyses

All analyses were performed using the software Comprehensive Meta Analysis version 2.2 (Biostat, Englewood, NJ, USA) under a random-effects model, selected a priori because clinical and methodological heterogeneity was expected across participant characteristics, arterial segments, and RT protocols. Descriptive data are presented as mean $\pm$ standard deviation. Statistical heterogeneity was quantified using the I^2 statistic, with values $\geq 50\%$ indicating moderate-to-high heterogeneity [33,34].

When moderate-to-high heterogeneity was identified, subgroup analyses were conducted according to study design (controlled vs. non-controlled), methodological quality (above vs. below the median TESTEX score), participant characteristics (age < 40 vs. ≥ 40 years, normal vs. above-normal blood pressure, and baseline PWV below vs. above the median), blood pressure reduction (yes vs. no), and FITT-VP exercise prescription variables (frequency, intensity, follow-up duration, and total training volume), dichotomized at the median. Baseline blood pressure status was classified according to the 2025 AHA/ACC Guideline [26]. Exercise intensity was standardized as a percentage of one-repetition maximum according to ACSM guidelines [28] when not directly reported.

Physical activity status was not included as a moderator because it was derived from heterogeneous qualitative descriptions rather than from a standardized assessment

instrument. Moreover, objective cardiorespiratory fitness data were available in only six studies, precluding reliable subgroup or meta-regression analyses.

To facilitate clinical interpretation, standardized effect sizes were converted to absolute PWV changes (m/s) by multiplying Hedges' *g* by the corresponding standard deviation for each moderator level [35].

Study and intervention characteristics were summarized in tables, whereas individual and pooled effect estimates with 95% confidence intervals were presented in forest plots. Publication bias was assessed by visual inspection of funnel plots and Egger's regression test.

3. Results

3.1. Study Selection

The bridge search identified 595 additional records; however, no additional trial met the eligibility criteria. Across the original and updated searches, 1323 records were identified, of which 14 trials met the inclusion criteria, yielding 20 RT interventions, including 10 controlled comparisons [9,11,16,17,36–45]. The included studies were published between 2005 and 2023 (mean publication year: 2013 ± 5.4). The complete study selection process is summarized in Figure 1.

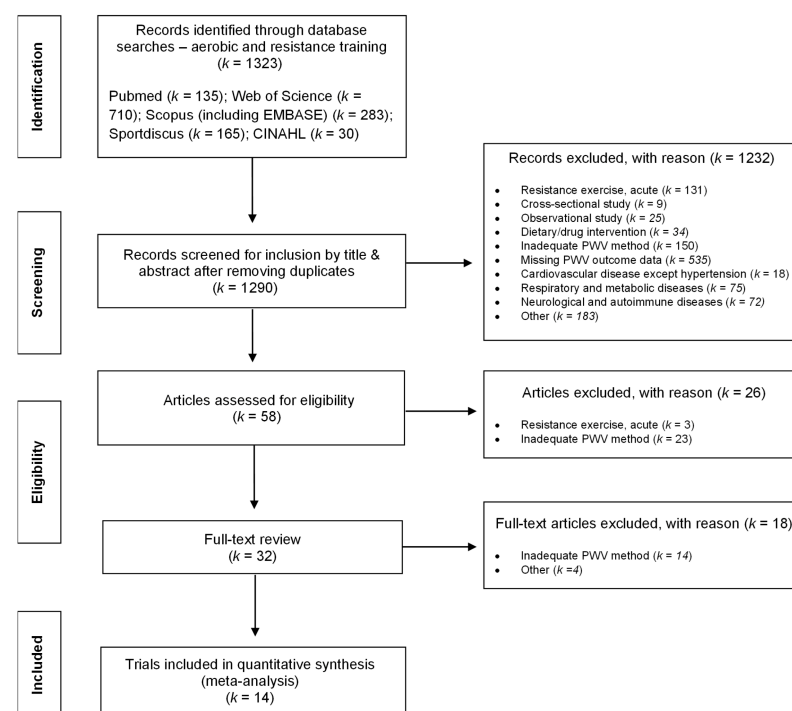


Figure 1. Flow diagram, illustrating the details of the search strategy, screening of potentially qualifying reports (k), the selection of the included trials and the reasons for study exclusion.

3.2. Participant and Study Characteristics

Study characteristics are summarized in Table 1. The included trials comprised 379 participants, of whom 278 were allocated to RT interventions and 101 to control groups. Participants across the RT and control groups were aged 19–63 years; participants allocated to RT had a mean age of 33.3 ± 14.3 years, whereas those allocated to control groups had a mean age of 32.4 ± 15.1 years. Sex was reported for 240 participants allocated to RT, including 117 men and 123 women. RT intervention-arm sample sizes ranged from 8 to 24 participants. Participant ethnicity was rarely reported; geographically, 11 trials were conducted in North America, two in Asia, and one in Europe.

Table 1. Characteristics of included resistance exercise training interventions (14 trials with 20 interventions).

Study		Sample Characteristics					Exercise Characteristics (FITT)				
Reference	Group	Sample	BP Status	PA Status	Age (Years)	Sex (%fem)	Frequency (d/wk)	Weeks Total	Intensity	Volume	Type
Cortez-Cooper et al. (2005) [11]	RE	23	Normal	Inactive	29.0 ± 1.0	100	4	11	MVF	12 exerc.; 3 sets, 5–10 reps (periodized)	Conv
	CG	10	Normal	Inactive	27.0 ± 2.0	100	-	-	-	-	-
Casey et al. (2007) [17]	RE	24	Normal	Inactive	21.0 ± 2.4	55	3	12	MVF	7 exerc.; 2 sets, 8–12 reps (periodized)	Conv
	CG	18	Normal	Inactive	22.0 ± 3.0	45	-	-	-	-	-
Collier et al. (2008) [9]	RE	15	Above normal	Active	47.0 ± 7.7	67	3	4	65% 10 RM	12 exerc.; 3 sets, 10 reps	Conv
Yoshizawa et al. (2009) [16]	RE	11	Normal	Inactive	47.0 ± 6.6	100	2	12	60% 1 RM	6 exerc.; 3 sets, 10 reps	Conv
	CG	12	Normal	Inactive	47.0 ± 10.5	100	-	-	-	-	-
Heffernan et al. (2009) [36] (1)	RE (Afro Americans)	19	Normal	Inactive	22.0 ± 1.7	0	3	6	MVF	5 exerc.; 3 sets, 8–15 reps (periodized)	Conv
Heffernan et al. (2009) [36] (2)	RE (Caucasians)	18	Normal	Inactive	23 ± 3.0	0	3	6	MVF	5 exerc.; 3 sets, 8–15 reps (periodized)	Conv
Collier et al. (2011) [37] (1)	RE (Men)	10	Above normal	Active	44.0 ± 3.0	0	3	4	65% 10 RM	9 exerc.; 3 sets, 10 reps	Conv
Collier et al. (2011) [37] (2)	RE (Women)	10	Above normal	Active	52.0 ± 7.0	100	3	4	65% 10 RM	9 exerc.; 3 sets, 10 reps	Conv.
Beck et al. (2013) [38]	RE	15	Above normal	Inactive	21.1 ± 2.3	27	3	8	60% 1 RM	7 exerc.; 2 sets, 8–12 reps (periodized)	Conv
	CG	15	Above normal	Inactive	21.1 ± 2.3	34	-	-	-	-	-
Rossow et al. (2014) [39] (1)	RE (Young adults)	16	Normal	Inactive	22.0 ± 2.0	100	3	8	80% 1 RM	6 exerc.; 3 sets, 8–10 reps	Conv
Rossow et al. (2014) [39] (2)	RE (Middle-aged adults)	13	Normal	Inactive	57.0 ± 3.0	100	3	8	80% 1 RM	6 exerc.; 3 sets, 8–10 reps	Conv
Okamoto et al. (2015) [40]	RE	9	Normal	Inactive	23.0 ± 3.0	NI	3	16	MVF 75% 1 RM	1 exerc.; 3 sets, 10 reps (periodized)	Conv

Table 1. Cont.

Study		Sample Characteristics					Exercise Characteristics (FITT)				
Reference	Group	Sample	BP Status	PA Status	Age (Years)	Sex (%fem)	Frequency (d/wk)	Weeks Total	Intensity	Volume	Type
Lin et al. (2016) [41]	RE (placebo)	11	Normal	Inactive	57.0 ± 6.6	64	1	12	MVF	1 exerc.; 1 set, 10 reps	Conv (Exc.)
DeVallance et al. (2016) [42] (1)	RE	13	Above normal	Inactive	51.0 ± 10.8	NI	3	8	60–80% 1 RM	6 exerc.; 3 sets, 8–12 reps (periodized)	Conv
	CG	16	Above normal	Inactive	51.0 ± 16.0	NI	-	-	-	-	-
DeVallance et al. (2016) [42] (2)	RE	16	Normal	Inactive	49.0 ± 12.6	NI	3	8	60–80% 1 RM	6 exerc.; 3 sets, 8–12 reps (periodized)	Conv
	CG	12	Normal	Inactive	44.0 ± 10.6	NI	-	-	-	-	-
Jürgenson et al. (2019) [43]	RE	19	Normal	Active	28.2 ± 6.1	0	4	12	60–80% 1 RM	12 exerc.; 6 sets, 12–4 reps (periodized)	Conv
Werner et al. (2021) [44] (1)	RE (High intensity)	10	Normal	Inactive	22.9 ± 2.9	0	3	12	80–90% 1 RM	9 exerc.; 2–3 sets, 3–8 reps	Conv
	CG	10	Normal	Inactive	21.2 ± 2.8	0	-	-	-	-	-
Werner et al. (2021) [44] (2)	RE (High volume)	10	Normal	Inactive	22.9 ± 2.9	0	3	12	50–60% 1 RM	9 exerc.; 2–3 sets, 10–15 reps (periodized)	Conv
	CG	10	Normal	Inactive	21.2 ± 2.8	0	-	-	-	-	-
Morgan et al. (2023) [45] (1)	RE (High intensity)	8	Normal	Inactive	28.8 ± 1.2	100	5	11	80–90% 1 RM	3 exerc.; 2–3 sets, 3–8 reps	Conv
	CG	8	Normal	Inactive	20.9 ± 1.2	100	-	-	-	-	-
Morgan et al. (2023) [45] (2)	RE (High volume)	8	Normal	Inactive	19.8 ± 1.7	100	5	11	50–60% 1 RM	3 exerc.; 2–3 sets, 10–15 reps (periodized)	Conv
	CG	8	Normal	Inactive	20.9 ± 1.2	100	-	-	-	-	-

RE: Resistance training; CG: control group; BP: Blood pressure; d/wk: days a week; Conv: Conventional; Exc.: emphasis on eccentric muscle contraction; MVF: Maximum voluntary fatigue; NI: no information; PA: Physical activity; 1 RM: one repetition-maximum; 10 RM: ten repetitions-maximum; exerc.: exercises; sets: series; reps: repetitions. Numbers in parentheses indicate interventions within a given trial.

Physical activity status was classified according to the qualitative descriptions provided in the original studies rather than through a common standardized assessment instrument. Eleven trials included participants described as physically inactive or sedentary ($n = 224$), whereas three included participants described as physically active ($n = 54$). Seven trials used controlled designs, and seven used uncontrolled pre–post designs. Among the controlled trials, three included multiple RT intervention arms, whereas four compared a single RT intervention group with a control group.

Participants generally had a BMI of $26.1 \pm 3.2 \text{ kg/m}^2$ and body fat of $24.2 \pm 10.5\%$. Muscular strength was assessed using one-repetition maximum (1 RM) tests in 10 studies, although baseline values were inconsistently reported. Cardiorespiratory fitness was assessed in six studies and ranged from 18 to $25 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, indicating generally low aerobic capacity. Most were free from cardiovascular disease, although approximately 20% ($n = 63$) had diagnosed conditions and a small proportion ($n = 8$) were receiving pharmacological treatment.

Baseline blood pressure was reported in 14 trials (20 interventions). According to the 2025 AHA/ACC Guideline criteria [26], most participants had normal blood pressure ($n = 215$; $117.5 \pm 6.7/72.8 \pm 6.3 \text{ mmHg}$), whereas a smaller subgroup had above-normal blood pressure ($n = 63$; $129.8 \pm 6.7/76.2 \pm 4.2 \text{ mmHg}$). Across studies, baseline blood pressure ranged from 108/64 to 137/90 mmHg.

3.3. Exercise Intervention Characteristics

Interventions lasted 9.3 ± 3.3 weeks (range: 4–16), with a frequency of 3.2 ± 0.9 sessions/week and intensity of $68 \pm 17\%$ 1 RM ($\approx 6.4 \pm 1.6$ METs). Protocols typically included 6.7 ± 3.3 exercises per session, 3.0 ± 0.9 sets, and 9.6 ± 2.0 repetitions per set. Most interventions (80%) involved whole-body training, whereas 20% targeted isolated muscle groups, including isolated biceps curl exercise in Okamoto et al. [40] and eccentric leg extension exercise in Lin et al. [41].

Dynamic RT was used in nearly all interventions ($k = 19$). Intensity was primarily reported as %1 RM, although some studies used %10 RM or did not report intensity. To standardize comparisons, intensities were converted to METs or estimated from training parameters. Based on these estimates, most interventions were classified as vigorous (≥ 6 METs), with the remainder categorized as low-to-moderate intensity, although variability was considerable (2.0–8.0 METs). Training progression was absent in approximately half of the interventions, whereas the remainder incorporated progressive increases in intensity, volume, or both.

3.4. Overall PWV Change Following Resistance Training

Table 2 summarizes the raw PWV values used in the calculations. Fourteen trials (20 RT interventions; $n = 278$) contributed 31 eligible effect estimates: 20 central, six mixed, and five peripheral estimates. For Lin et al. [41], only the placebo arm ($n = 11$) was included.

Central PWV was assessed in 14 trials (20 intervention estimates). Although individual studies showed changes in both directions, the pooled central effect was close to zero and non-significant.

Mixed PWV was evaluated in four controlled trials (six effect estimates). The pooled estimate favored training to a moderate degree but was imprecise and did not reach statistical significance.

Peripheral PWV contributed five effect estimates and showed no statistically significant pooled change. These results should be interpreted from the pooled estimates and confidence intervals rather than from the direction of individual pre–post changes alone.

Table 2. Mean pulse wave velocity changes following resistance exercise training ($k = 14$ with 20 interventions).

Study		PWV Assessment		PWV Change Following Training (m/s)										
Reference	Intervention	Sample	Instrument Device	Outcome		Central PWV			Mixed PWV			Peripheral PWV		
				Measure	Units	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
Cortez-Cooper et al. (2005) [11]	RE	23	T	cfPWV	m/s	7.9	8.3	$\uparrow 0.4^*$						
	CG	10				7.2	7.8	$\uparrow 0.6^*$						
Casey et al. (2007) [17]	RE	24	T	cfPWV crPWV faPWV	m/s	6.5	6.3	$\downarrow 0.2$	8.4	8.4	$\leftrightarrow 0.0$	9.5	9.5	$\leftrightarrow 0.0$
	CG	18				6.9	7.0	$\uparrow 0.1$	8.4	8.3	$\downarrow 0.1$	9.0	9.0	$\leftrightarrow 0.0$
Collier et al. (2008) [9]	RE	15	D	cfPWV faPWV	m/s	11.0	12.7	$\uparrow 1.7^*$				11.5	12.5	$\uparrow 1.0^*$
Yoshizawa et al. (2009) [16]	RE	11	T	cfPWV	m/s	9.2	9.0	$\downarrow 0.2$						
	CG	12				9.2	9.5	$\uparrow 0.3$						
Heffernan et al. (2009) [36] (1)	RE (Afro-Americans)	19	T	cfPWV	m/s	6.1	5.9	$\downarrow 0.2$						
Heffernan et al. (2009) [36] (2)	RE (Caucasians)	18	T	cfPWV	m/s	5.6	5.7	$\uparrow 0.1$						
Collier et al. (2011) [37] (1)	RE (Men)	10	D	cfPWV	m/s	12.0	13.9	$\uparrow 1.9^*$						
Collier et al. (2011) [37] (2)	RE (Women)	10	D	cfPWV	m/s	9.2	9.6	$\uparrow 0.4$						
Beck et al. (2013) [38]	RE	15	T	cfPWV crPWV faPWV	m/s	7.0	6.8	$\downarrow 0.2$						
									8.8	7.8	$\downarrow 1.0^*$	10.4	9.4	$\downarrow 1.0^*$
	CG	15	T	cfPWV crPWV faPWV	m/s	7.0	7.0	$\leftrightarrow 0.0$						
									8.8	8.8	$\leftrightarrow 0.0$	10.4	10.2	$\downarrow 0.2$
Rossow et al. (2014) [39] (1)	RE (Young)	16	T	cfPWV faPWV	m/s	5.8	5.6	$\downarrow 0.2$				8.5	8.6	$\uparrow 0.1$
Rossow et al. (2014) [39] (2)	RE (Middle-aged)	13				7.9	7.5	$\downarrow 0.4$				9.0	8.3	$\downarrow 0.7$
Okamoto et al. (2015) [40]	RE	9	T	cfPWV	m/s	7.1	8.7	$\uparrow 1.6^*$						
Lin et al. (2016) [41]	RE (placebo)	11	T	cfPWV	m/s	7.3	7.5	$\uparrow 0.2$						

Table 2. Cont.

Study		PWV Assessment				PWV Change Following Training (m/s)								
Reference	Intervention	Sample	Instrument Device	Outcome		Central PWV			Mixed PWV			Peripheral PWV		
				Measure	Units	Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
DeVallance et al. (2016) [42] (1)	RE	13	T	cfPWV	m/s	7.8	7.7	↓ 0.1						
	CG	16	T	cfPWV	m/s	7.9	8.0	↑ 0.1						
DeVallance et al. (2016) [42] (2)	RE	16	T	cfPWV	m/s	6.9	7.0	↑ 0.1						
	CG	12				6.5	6.7	↑ 0.2						
Jürgenson et al. (2019) [43]	RE	19	T	cfPWV	m/s	6.2	6.5	↑ 0.3						
Werner et al. (2021) [44] (1)	RE (High intensity)	10	T	cfPWV crPWV	m/s	7.0	8.0	↑ 1.0	9.7	10.7	↑ 1.0			
	CG	10				6.6	6.6	↔ 0.0	9.4	9.8	↑ 0.4			
Werner et al. (2021) [44] (2)	RE (High volume)	10	T	cfPWV crPWV	m/s	6.5	6.9	↑ 0.4	9.2	9.3	↑ 0.1			
	CG	10				6.6	6.6	↔ 0.0	9.4	9.8	↑ 0.4			
Morgan et al. (2023) [45] (1)	RE (High intensity)	8	T	cfPWV crPWV	m/s	6.1	6.6	↑ 0.5	9.3	9.5	↑ 0.2			
	CG	8				6.4	8.4	↑ 2.0 *	9.8	12.6	↑ 2.8 *			
Morgan et al. (2023) [45] (2)	RE (High volume)	8	T	cfPWV crPWV	m/s	6.8	6.3	↓ 0.5	10.4	9.7	↓ 0.7			
	CG	8				6.4	8.4	↑ 2.0 *	9.8	12.6	↑ 2.8 *			

RE: resistance training group; CG: control group; PWV: pulse wave velocity; D: Doppler; T: tonometry; cfPWV: carotid–femoral pulse wave velocity; crPWV: carotid–radial pulse wave velocity; faPWV: femoral–ankle pulse wave velocity. ↑: increase; ↔: stability; ↓: decrease. *: Significant post- versus pre-intervention difference ($p < 0.05$). Numbers in parentheses indicate interventions within a given trial. Values reported as SEM were converted to SD ($SD = SEM \times \sqrt{n}$) for effect-size calculations. For Lin et al. [41], only the placebo arm ($n = 11$) was used. The faPWV outcome reported by Yoshizawa et al. [16] was excluded because it was obtained using an oscillometric method, which was not eligible.

3.5. Pooled Effects of Resistance Training on PWV

Using the primary assumption of $r = 0.50$, RT was not associated with a statistically significant change in central PWV ($g = -0.002$; 95% CI, -0.213 to 0.209 ; $p = 0.985$), mixed PWV ($g = -0.548$; 95% CI, -1.168 to 0.071 ; $p = 0.083$), peripheral PWV ($g = -0.110$; 95% CI, -0.436 to 0.217 ; $p = 0.511$), or overall PWV ($g = -0.102$; 95% CI, -0.281 to 0.077 ; $p = 0.265$) (Figure 2). The mixed-segment estimate was moderate in magnitude but imprecise and did not reach statistical significance.

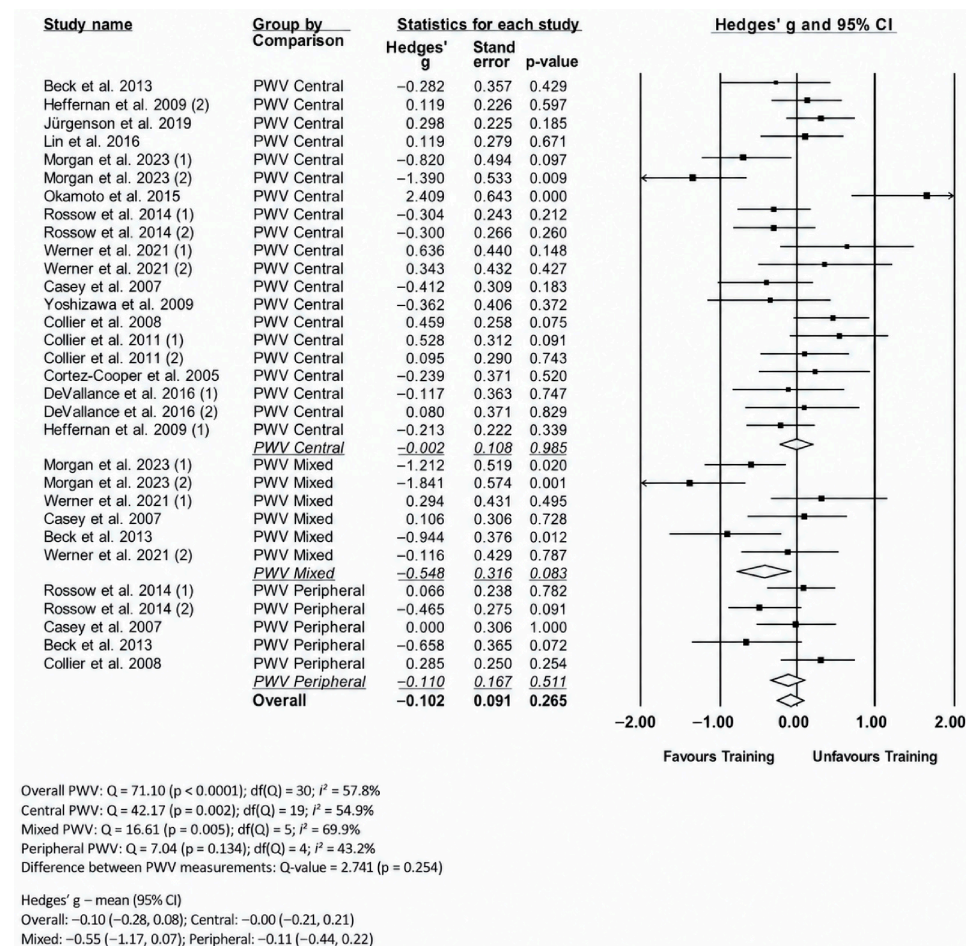


Figure 2. Effects of dynamic resistance training on arterial stiffness assessed by PWV [9,11,16,17,36–45]. Hedges' g values are standardized pre-post effects; negative values favor training. The primary analysis assumed a pre-post correlation of $r = 0.50$ and used a random-effects model. CI: confidence interval; PWV: pulse wave velocity. Numbers in parentheses indicate interventions within a given trial.

Statistical heterogeneity was moderate for central PWV ($I^2 = 55\%$), mixed PWV ($I^2 = 70\%$), and the overall synthesis ($I^2 = 58\%$), and lower for peripheral PWV ($I^2 = 43\%$). Sensitivity analyses assuming $r = 0.30$ and $r = 0.70$ yielded overall effects of $g = -0.079$ (95% CI, -0.238 to 0.080 ; $p = 0.328$) and $g = -0.140$ (95% CI, -0.352 to 0.072 ; $p = 0.195$), respectively; none of the segment-specific estimates became statistically significant.

3.6. Study Quality, Risk of Bias, and Certainty of Evidence

Visual inspection of the funnel plot did not indicate marked asymmetry (Figure 3). Egger's regression test was not statistically significant (intercept = -1.375 , SE = 1.015 , $t = -1.354$, $df = 29$, $p = 0.186$), and the Duval and Tweedie trim-and-fill procedure estimated no missing effect estimates ($k_0 = 0$). Accordingly, the trim-and-fill-adjusted pooled effect was identical to the observed estimate (Hedges' $g = -0.102$; 95% CI: -0.281 to 0.077 ;

$p = 0.265$). These findings should nevertheless be interpreted cautiously because several trials contributed more than one effect estimate, limiting the independence assumed by conventional funnel-plot methods and their ability to detect publication bias.

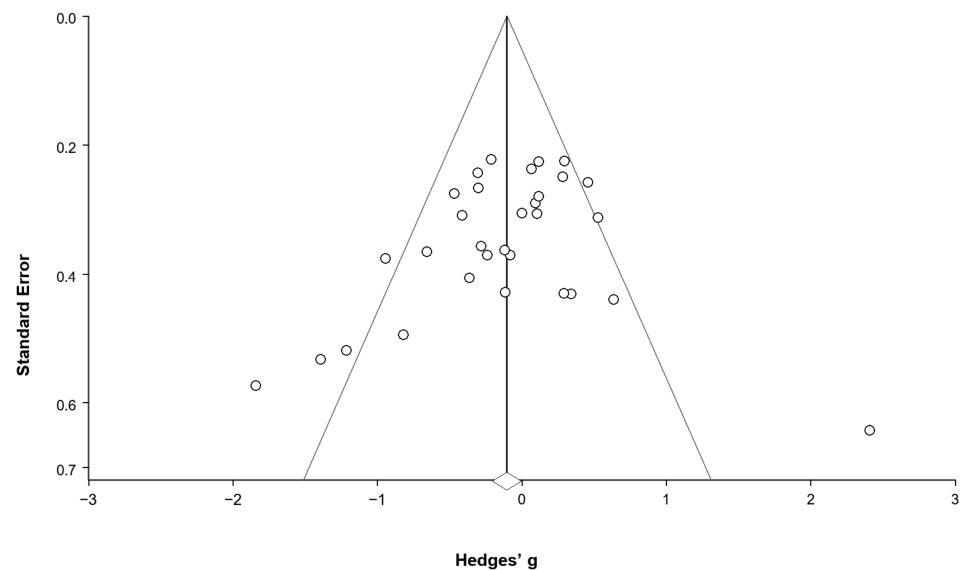


Figure 3. Funnel plot of Hedges' g effect estimates for the effects of dynamic resistance training on central, mixed, and peripheral pulse wave velocity. The analysis included 31 effect estimates from 14 trials. The white diamond represents the observed pooled effect. The trim-and-fill procedure estimated no missing effects ($k_0 = 0$); therefore, the adjusted pooled effect was identical to the observed estimate.

The certainty of evidence ranged from moderate to very low (GRADE assessment, Supplementary Table S2). Evidence from controlled comparisons was rated as moderate for overall and central PWV and low for mixed and peripheral PWV, mainly because of imprecision and, for mixed and peripheral PWV, inconsistency. Evidence from non-controlled comparisons was rated as very low because of study-design limitations, imprecision, and residual heterogeneity. Publication bias was not considered a reason for downgrading the certainty of evidence because Egger's test was non-significant and the trim-and-fill analysis did not impute missing effects. However, the relatively small number of trials and the inclusion of multiple, non-independent effect estimates from some studies reduced the sensitivity of these assessments.

3.7. Moderators of PWV Changes Following Resistance Training

Exploratory moderator analyses are presented in Table 3. In the overall synthesis, statistically significant between-stratum differences were observed for study design ($Q = 10.09$; $p = 0.001$) and methodological quality ($Q = 7.35$; $p = 0.007$). In the central synthesis, study design differed between strata ($Q = 5.15$; $p = 0.023$). In the mixed-PWV synthesis, significant differences were observed for methodological quality ($p = 0.041$), blood pressure reduction ($p = 0.014$), training frequency ($p = 0.002$), and total training volume ($p = 0.014$). These findings are exploratory because several strata contained only one or two effect estimates, comparisons sharing control groups were treated as independent, and no adjustment was made for multiple testing.

Table 3. Hedges' g effect sizes ('g'), difference tests (Q), and estimated reduction in overall, central, and mixed pulse wave velocity by moderator after dynamic resistance training in trials with moderate to high heterogeneity ($I^2 > 50\%$) (14 trials with 20 interventions).

Moderator	Overall Pulse Wave Velocity					Central Pulse Wave Velocity					Mixed Pulse Wave Velocity				
	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)
Design															
Controlled	18	−0.329 (−0.580, −0.078)	10.09 (0.001)	46.6	−0.45	10	−0.237 (−0.527, 0.054)	5.15 (0.023)	28.2	−0.28	6	−0.548 (−1.168, 0.071)	-	69.9	−0.29
Non-controlled	13	0.114 (−0.112, 0.341)		58.9	0.24	10	0.168 (−0.110, 0.446)		63.2	0.25	-	-		-	-
TESTEX															
<11 points	19	0.027 (−0.150, 0.205)	7.35 (0.007)	46.0	0.04	14	0.042 (−0.191, 0.275)	0.48 (0.490)	55.2	0.06	1	0.106 (−0.494, 0.706)	4.17 (0.041)	0.0	0.07
≥11 points	12	−0.430 (−0.826, −0.034)		63.8	−0.55	6	−0.172 (−0.693, 0.349)		60.6	−0.20	5	−0.713 (−1.421, −0.005)		67.9	−1.08
Age															
<40 y	21	−0.182 (−0.431, 0.067)	2.28 (0.131)	64.5	−0.19	12	−0.044 (−0.372, 0.285)	0.96 (0.327)	66.9	−0.04	6	−0.548 (−1.168, 0.071)	-	69.9	−0.77
≥40 y	10	0.041 (−0.176, 0.259)		27.5	0.09	8	0.079 (−0.148, 0.307)		11.8	0.17	-	-		-	-
BP rest															
Normal	23	−0.130 (−0.342, 0.082)	1.22 (0.269)	57.9	−0.24	15	−0.061 (−0.322, 0.201)	2.68 (0.101)	59.6	−0.07	5	−0.469 (−1.191, 0.253)	2.48 (0.116)	71.7	−0.48
Above-normal	8	−0.031 (−0.377, 0.315)		60.2	−0.06	5	0.186 (−0.112, 0.485)		17.1	0.39	1	−0.944 (−1.680, −0.208)		0.0	−0.99
PWV rest															
≤median	17	−0.177 (−0.390, 0.036)	1.58 (0.208)	46.2	−0.18	10	−0.106 (−0.390, 0.177)	0.80 (0.370)	47.9	−0.13	4	−0.485 (−1.111, 0.141)	0.04 (0.843)	61.1	−0.63
>median	14	0.006 (−0.299, 0.311)		67.3	0.01	10	0.113 (−0.211, 0.437)		62.6	0.19	2	−0.740 (−2.831, 1.351)		88.7	−1.39

Table 3. Cont.

Moderator	Overall Pulse Wave Velocity					Central Pulse Wave Velocity					Mixed Pulse Wave Velocity				
	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)	k	Hedges g (CI 95%)	Q (p-Value)	I ² (%)	Δ PWV (m/s)
BP reduction															
No	20	−0.097 (−0.324, 0.129)	0.84 (0.359)	54.4	−0.14	13	−0.047 (−0.347, 0.252)	3.74 (0.053)	58.4	−0.06	4	−0.299 (−1.072, 0.474)	5.99 (0.014)	71.3	−0.47
Yes	9	−0.164 (−0.560, 0.232)		70.9	−0.33	5	0.151 (−0.243, 0.544)		52.1	0.32	2	−1.036 (−1.633, −0.440)		0.0	−0.93
Frequency															
≤3 d/wk	25	−0.012 (−0.181, 0.157)	3.72 (0.054)	46.4	−0.02	16	0.061 (−0.159, 0.280)	0.60 (0.440)	50.6	0.09	4	−0.169 (−0.707, 0.370)	9.71 (0.002)	51.9	−0.21
>3 d/wk	6	−0.778 (−1.496, −0.060)		77.9	−0.71	4	−0.434 (−1.164, 0.297)		73.3	−0.41	2	−1.495 (−2.249, −0.740)		0.0	−2.67
Intensity															
Light/moderate	12	0.041 (−0.273, 0.354)	2.43 (0.119)	67.2	0.07	8	0.207 (−0.162, 0.577)	3.17 (0.075)	66.7	0.33	2	−0.556 (−1.366, 0.254)	0.55 (0.457)	52.6	−0.53
Vigorous	19	−0.186 (−0.399, 0.028)		48.8	−0.22	12	−0.134 (−0.377, 0.110)		38.9	−0.12	4	−0.586 (−1.524, 0.352)		78.5	−0.99
Follow-up															
≤24 sessions	17	−0.082 (−0.253, 0.089)	0.01 (0.926)	35.3	−0.13	12	−0.015 (−0.182, 0.152)	0.15 (0.703)	7.3	−0.02	1	−0.944 (−1.680, −0.208)	2.48 (0.116)	0.0	−0.85
>24 sessions	14	−0.129 (−0.522, 0.264)		72.0	−0.16	8	0.056 (−0.515, 0.626)		76.8	0.05	5	−0.469 (−1.191, 0.253)		71.7	−0.74
Total volume															
≤4320 a.u.	20	−0.097 (−0.311, 0.117)	0.07 (0.788)	59.4	−0.16	14	0.018 (−0.224, 0.260)	0.00 (0.999)	54.7	0.03	2	−1.036 (−1.633, −0.440)	5.99 (0.014)	0.0	−1.54
>4320 a.u.	11	−0.123 (−0.466, 0.220)		58.8	−0.16	6	−0.075 (−0.553, 0.403)		62.9	−0.11	4	−0.299 (−1.072, 0.474)		71.3	−0.43

BP: Blood pressure; PWV: Pulse wave velocity (medians—overall: 7.9; central: 7.0 m/s; mixed: 9.3 m/s); Total volume: product of the number of sessions × sets × repetitions × number of exercises; Δ PWV converted to m/s. For each moderator and level of interest, ‘g’ was arithmetically transformed using the standard deviation corresponding to the sample mean; a.u.: arbitrary units. Two interventions from one trial investigating central PWV (Heffernan et al. [36]) were excluded because BP reduction was not specified. Physical activity status was not analyzed as a moderator because assessment and classification methods were heterogeneous across studies; objective cardiorespiratory fitness data were reported in only six studies. Results are exploratory; no multiplicity adjustment was applied.

Age, baseline blood pressure, baseline PWV, training intensity, and intervention exposure did not show statistically significant between-stratum differences in any of the reported syntheses. The overall training-frequency comparison approached significance ($Q = 3.72$; $p = 0.054$).

4. Discussion

The meta-analysis indicates no statistically significant pooled effect of dynamic RT on arterial stiffness. The central and overall standardized effects were small, whereas the mixed-segment estimate was moderate but imprecise and crossed the null; peripheral PWV also showed a small, non-significant estimate. These findings were robust to sensitivity analyses using alternative pre-post correlations. Accordingly, the results support the absence of a detected pooled effect within the available evidence, rather than equivalence or proof that RT has no physiological influence on arterial stiffness.

These overall neutral findings deserve consideration in light of previous meta-analyses, which have reported conflicting effects of RT on arterial stiffness [6–8,19–22]. Although these discrepancies might initially appear contradictory, they likely reflect differences in study populations, arterial regions assessed, and exercise protocols rather than a consistent physiological effect of RT itself. Acute resistance exercise may transiently increase arterial stiffness because of marked elevations in blood pressure, mechanical stress, and sympathetic activation during exercise; however, these short-term responses do not necessarily translate into adverse chronic vascular adaptations [6,19]. Studies reporting increased PWV have often involved relatively high-intensity protocols performed by young, healthy adults [19], whereas investigations employing lower loads, higher training volumes, or participants with above-normal blood pressure or cardiovascular risk have generally reported neutral or modestly beneficial effects [6,7,20,22]. The apparently conflicting observations may therefore represent protocol- or population-specific responses rather than evidence of a consistently detrimental chronic effect of RT. Moreover, transient increases in vascular load during individual sessions may be counterbalanced by favorable adaptations in vascular function over time, resulting in the overall neutral effect on PWV observed in the present meta-analysis.

Several mechanisms may contribute to this balance between potentially beneficial and adverse vascular responses. On the one hand, RT may improve endothelial function, nitric oxide bioavailability, metabolic profile, insulin sensitivity, and the regulation of vascular tone [15,36,37,42,46–49]. On the other hand, high-load, low-repetition protocols and the use of the Valsalva maneuver may produce substantial acute elevations in blood pressure and mechanical stress [50–52]. Proposed chronic adverse mechanisms include sustained sympathetic activation and increased vascular tone [19,51], inflammatory responses, particularly in individuals with unfavorable metabolic profiles [53], and structural alterations in the arterial wall, such as increased collagen deposition and reduced elastin content [19,51]. The magnitude and direction of these responses may depend on the interaction between the characteristics of the training stimulus and the physiological profile of the participant, with lower-load, higher-repetition protocols potentially attenuating adverse vascular responses [19,50].

Nevertheless, compared with aerobic exercise, the intermittent and relatively short-duration nature of RT may provide a less sustained increase in blood flow and endothelial shear stress, thereby limiting the stimulus required for consistent structural remodeling of the arterial wall [5–7,20,54]. RT protocols may also provide insufficient frequency or cumulative volume to induce detectable structural vascular adaptations [47]. Thus, even when local vasodilatory capacity and nitric oxide-mediated responses are preserved [14,20], the net chronic stimulus may be insufficient to produce a consistent change in PWV. This

interpretation provides a plausible physiological explanation for both the negligible pooled effects and the failure of participant- and protocol-related variables to account for between-study heterogeneity.

The estimates remain broadly consistent with previous systematic reviews and meta-analyses reporting minimal or non-significant changes in PWV after RT [6,20]. However, the moderate mixed-segment estimate and its wide confidence interval underscore that the available evidence is not sufficiently precise to establish vascular equivalence. Unlike earlier reviews that often examined overall PWV or particular arterial sites [7,19,21,22], the present analysis evaluated central, mixed, and peripheral arterial segments in a unified framework.

The subgroup analyses yielded statistically significant differences by study design and methodological quality in the overall synthesis, and by study design in the central synthesis. Controlled comparisons produced a pooled overall estimate favoring RT, whereas non-controlled estimates were centered on the null. Higher-TESTEX studies also produced a more negative overall estimate than lower-TESTEX studies. These contrasts should not be interpreted causally, because study design and methodological quality overlap with other study characteristics, several comparisons shared control groups, and the number of studies was small.

Baseline blood pressure status and baseline PWV did not significantly differentiate the pooled effects. Participants with above-normal blood pressure were represented by only eight effect estimates overall and one mixed-PWV estimate, limiting precision. Thus, the analysis does not establish that baseline vascular or blood pressure status is irrelevant; it indicates only that statistically reliable differences were not detected in the available dataset.

The significant subgroup tests also require caution. The analysis involved repeated testing across moderators and arterial regions without adjustment for multiplicity. Some significant mixed-PWV contrasts were based on strata containing only one or two estimates and were strongly influenced by Beck and Morgan. These findings should therefore be considered hypothesis-generating and require confirmation in prospectively powered datasets.

Compared with previous meta-analyses, the present study evaluated a broader combination of participant characteristics and exercise-prescription variables within a unified analytical framework [6–8,19–22]. The analyses suggest that methodological features and selected training descriptors may contribute to observed variability, particularly for mixed PWV, but the sparse and unbalanced evidence prevents firm conclusions about physiological moderation. Future studies should evaluate these candidate moderators using adequately powered models and should report cumulative hemodynamic load, endothelial function, autonomic regulation, and arterial wall remodeling more consistently [54,55].

Among FITT-VP variables, training frequency and total training volume differentiated mixed-PWV estimates, whereas training intensity and intervention exposure did not. The significant frequency and volume contrasts were driven by very small strata and are not independent because the same Morgan interventions contributed to both classifications. They should not be interpreted as dose-response evidence.

Conventional FITT-VP descriptors may therefore be insufficient to characterize the vascular stimulus generated by RT. Variables such as time under tension, movement velocity, proximity to muscular failure, inter-set recovery, transient pressor responses, the use of the Valsalva maneuver, and cumulative internal training load may be more informative than external descriptors such as frequency or percentage of one-repetition maximum alone. Vascular adaptations may also depend on the multidimensional and longer-term interaction of these factors rather than on any single training component. Future trials should report these variables more consistently and incorporate them into comprehensive models of exercise prescription, together with mechanistic outcomes such as endothelial function, autonomic regulation, arterial wall composition, and cumulative hemodynamic load.

From a clinical and public health perspective, our findings provide no evidence of a statistically significant adverse pooled effect of conventional dynamic RT on arterial stiffness. RT remains effective for improving muscular strength, body composition, functional capacity, and metabolic health. Nevertheless, non-significance should not be interpreted as proof of equivalence or complete vascular neutrality, particularly because heterogeneity and imprecision persisted.

To our knowledge, this is the first meta-analysis to systematically investigate whether participant characteristics and exercise-prescription variables explain the inconsistent effects of RT on arterial stiffness across multiple arterial segments. This analytical perspective represents an advance beyond estimating pooled effects alone.

Several limitations should be acknowledged. The calculation of standardized pre-post effects required an assumed within-participant correlation because change-score variances were generally unavailable; sensitivity analyses using $r = 0.30$ and $r = 0.70$ did not alter the significance of the primary pooled estimates. Moderator analyses involved small and markedly unequal strata and repeated comparisons without multiplicity adjustment. Because multiple estimates from the same trials and comparisons sharing control groups were treated as independent, the precision of the pooled, moderator, and publication-bias analyses may have been overestimated. Moderate-to-high heterogeneity persisted, participants with above-normal blood pressure were underrepresented, RT protocols varied substantially, and intervention durations were generally short. Accordingly, statistically significant subgroup differences should be treated as exploratory rather than confirmatory evidence of moderation.

5. Conclusions

Dynamic RT was not associated with a statistically significant pooled change in arterial stiffness across central, mixed, peripheral, or overall PWV. The central, peripheral, and overall estimates were small; the mixed-segment estimate was moderate but imprecise and crossed the null. Exploratory subgroup analyses identified several between-stratum differences, but sparse, unbalanced, and partially non-independent data preclude firm conclusions regarding moderator effects.

RT can therefore continue to be prescribed for its well-established musculoskeletal and metabolic benefits, as the present synthesis provides no evidence of a statistically significant adverse pooled effect on arterial stiffness. Conversely, the findings do not support prescribing RT in isolation with the expectation of consistently improving arterial stiffness. Further adequately powered trials are required to determine whether specific RT configurations produce clinically relevant arterial adaptations.

Future research should investigate whether multidimensional characteristics of the training stimulus, rather than conventional FITT descriptors alone, account for the variability in vascular adaptations observed after resistance training.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/ijerph23101247/s1>, Search Strategy; Inclusion and Exclusion Criteria; PRISMA 2020 Checklist [56]; Table S1: Quality of studies on resistance training and arterial stiffness according to TESTEX criteria ($k = 14$); Table S2: Quality Grading of Recommendations Assessment Development and Evaluation (GRADE).

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Abbreviations

The following abbreviations are used in this manuscript:

ACSM	American College of Sports Medicine
AMSTAR-2	A MeaSurement Tool to Assess Systematic Reviews 2
BMI	Body Mass Index
cfPWV	Carotid–Femoral Pulse Wave Velocity
CG	Control Group
CI	Confidence Interval
FITT-VP	Frequency, Intensity, Time, Type, Volume, and Progression
GRADE	Grading of Recommendations Assessment, Development and Evaluation
ICC	Intraclass Correlation Coefficient
MET	Metabolic Equivalent of Task
PWV	Pulse Wave Velocity
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO	International Prospective Register of Systematic Reviews
RT	Resistance Training
TESTEX	Tool for the Assessment of Study Quality and Reporting in Exercise
1 RM	One-Repetition Maximum
10 RM	Ten-Repetition Maximum

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