Pflügers Archiv - European Journal of Physiology (2019) 471:467–478
https://doi.org/10.1007/s00424-018-2235-8

INVITED REVIEW

Impact of high- and low-intensity resistance training on arterial stiffness and blood pressure in adults across the lifespan: a review

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Received: 2 August 2018 / Revised: 23 October 2018 / Accepted: 4 November 2018 / Published online: 13 November 2018

Abstract

Resistance training (RT) is performed for improvements in body composition in young healthy adults and for health benefits in middle-aged and older adults. Traditionally, RT is prescribed at moderate- to high-intensity to promote benefits on skeletal muscle mass and strength in middle-aged and older adults without considering the vascular effects. Recent evidence suggests that muscle strength may be more protective than muscle mass for cardiovascular disease prevention and that muscle strength can be importantly improved with low-intensity RT. The main purpose of this review was to examine the effects of RT intensity on arterial stiffness and blood pressure (peripheral and central) in young and older adults. Although small increases in central arterial stiffness (carotid β and carotid-femoral pulse wave velocity [PWV]) have been reported in young and middle-aged men, this review suggests that low- and high-intensity RT may not affect arterial stiffness whereas low-intensity RT may decrease systemic arterial stiffness (brachial-ankle PWV) in young healthy adults or not affect arterial stiffness in middle-aged and older adults. Independently of the intensity, RT may be effective to reduce blood pressure (peripheral and central) in middle-aged and older adults with at least elevated blood pressure at baseline. Further studies are needed to examine the impact of RT on arterial stiffness, central blood pressure, and wave reflection in middle-aged and older adults.

Keywords Resistance training · Arterial stiffness · Blood pressure · Intensity

This article is part of the special issue on Exercise Physiology: future opportunities and challenges in Pflügers Archiv – European Journal of Physiology

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Introduction

Arterial stiffening is a major age-related risk factor for the development of systolic hypertension [7] and cardiovascular disease (CVD) [90]. Arterial stiffness can be measured non-invasively as pulse wave velocity (PWV) in various arterial segments. Carotid-femoral PWV (cfPWV) is the gold standard measure of aortic stiffness [60], although it does not include the ascending aorta. Alternatively, brachial-ankle PWV (baPWV) is a composite of cfPWV and peripheral arterial stiffness, predominantly femoral-ankle (leg) PWV [82]. Both cfPWV and baPWV are positively associated with age and systolic blood pressure (BP) and are predictors of CVD and mortality [13, 85, 90, 91]. It is estimated that an increase in cfPWV or baPWV by 1.0 m/s increases the risk of total cardiovascular events (12–14%), mortality (13–15%), and all-cause mortality (13–15%) [90, 91]. Central (aortic and carotid) BP are more relevant risk factors than brachial BP for the development of hypertension and CVD because they exert a pressure load on the left ventricle, brain, and other organs [12, 77, 92]. The direction of the association between PWV

and BP is age-related [30]. In young adults, increased BP is the precursor of arterial stiffness, while in middle-aged and older adults this association is inverted. Elevated cfPWV contributes to augment left ventricular afterload via increases in the pulsatile components of aortic BP (systolic BP [SBP] and pulse pressure [PP]), leading to isolated systolic hypertension, ventricular hypertrophy, and heart failure in middle-aged and old adults [20, 39, 45]. In contrast, an increase in PWV may be determined by a concurrent increase in the steady components of BP (mean arterial pressure and diastolic BP) in young adults [59]. It is estimated that for each 10 mmHg increase in aortic SBP, the risk of cardiovascular events increases by 8.8% [89].

Increased cfPWV has been linked to altered body composition, both increased adiposity [80] and reduced muscle mass [1, 61]. Aortic stiffness also exhibits an inverse relationship to upper-body muscular strength in heathy young males [24]. Recent work further suggests that this relationship exists in young and older men and women and that relative muscle strength is more closely related to cfPWV than either muscle mass or absolute muscle strength alone [26]. Several studies have shown an inverse association between low leg muscle mass and baPWV [46, 61, 79]. Additionally, the negative relationship between reduced limb muscle mass and prevalence of hypertension [32] and the protective effect of high muscle strength against all-cause mortality in hypertensives [2] support the notion that the skeletal muscle fitness plays a beneficial role on vascular health. Since leg muscle mass is a strong determinant of reduced aortic and leg arterial stiffness in older adults [80], improvements in mass and/or strength may result in cardiovascular benefits by affecting PWV and BP.

Unlike participation in aerobic activities, which may benefit both cardiorespiratory fitness as well as reduce arterial stiffness and BP, the effects of muscle strengthening activities (e.g., resistance training [RT]) on arterial stiffness are less clear and controversial. Some experimental trials have found RT to cause an increase in arterial stiffness [57] while other studies observed no changes in arterial stiffness following RT [10]. Similarly, meta-analyses on this topic vary with one concluded no effect of RT on arterial stiffness [3] while another concluded high-intensity RT would increase arterial stiffness in young healthy individuals, especially in those with low levels at baseline [56]. In contrast, high-intensity RT did not change arterial stiffness in middle-aged adults [56]. Despite the mixed evidence regarding the effects of RT on arterial stiffness, it would seem that activities that benefit skeletal muscle health would also be beneficial for overall cardiovascular health based on the aforementioned relationships between the skeletal muscle and vasculature. Skeletal muscle and vascular health may both be influenced by inflammation [79] and endothelial function [6]. It is also possible that changes in vascular health precede changes in skeletal muscle health via reductions in skeletal muscle blood flow [1].

The American College of Sports Medicine recommends RT at 65–85% of one-repetition maximum (1RM) as a means for healthy adults to increase muscular fitness (e.g., strength and mass) [76]. To increase muscular strength, a relatively low (~ 50% of the one-repetition maximum, 1RM) RT intensity can be effective for novice trainees. In contrast, relatively high resistance exercise intensities (> 80% 1RM) may be necessary to enhance muscular strength and mass in experienced trainees [76]. While the physical stress of high-intensity resistance exercise benefits the structure and function of many bodily systems, primarily skeletal muscle and bone, it also imposes a unique stress on the cardiovascular system. For instance, high-intensity (> 90% 1RM) bilateral leg press exercise can cause arterial pressures to rise to ~ 320/250 mmHg [49]. Repeated acute increases in BP during high-intensity resistance exercise [49] may elicit structural and functional remodeling in the arterial and ventricular walls as well as chronic changes in resting BP. During high-intensity resistance exercise is difficult to avoid the Valsalva maneuver, which by itself can increase cfPWV [35]. Changes in arterial stiffness may be one consequence of alterations in arterial structure [96]. Numerus recent studies have examined the effects of high-intensity RT on PWV and BP in younger and older adults (Table 1). The following sections review the impact of high-intensity RT and low-intensity RT on arterial stiffness and resting BP in young and older adults.

The effects of high-intensity resistance training on arterial stiffness in young, middle-aged, and older adults

One of the first studies to investigate the effects of high-intensity RT on arterial stiffness observed an increase in carotid β stiffness following 4 months of whole-body (2 upper and 3 lower) exercise training to concentric failure in young, healthy men [57]. Similar increases in carotid β stiffness as well as cfPWV have occurred in young women after high-intensity RT [19]. However, other studies have shown no change in carotid β stiffness [34, 74], cfPWV or peripheral PWV (faPWV and carotid-radial) following RT in previously sedentary young adults (Fig. 1) [10, 34, 74]. The contradictory findings may be due to variations in the RT protocol. Further research in this area has explored different RT protocols and has demonstrated that the concentric component elicits the increase in PWV [62]. Additionally, upper but not lower body RT results in increased PWV [67]. Another consideration is the modality of RT, machine based or free weight based. Free weight-based RT mostly used by experienced trainees typically requires more core stabilization compared to machine based exercise which only allows movement through a fixed range

Table 1 Characteristics of participants and high-intensity resistance training protocols assessing chronic effects on arterial stiffness and blood pressure

Study	Age (y)	RT, *n*	Characteristics	Groups	%1RM or RM	Duration (weeks)	RT Protocol (sets × reps)	Arterial stiffness	BP
Young									
Miyachi et al. 2004 [57]	20–38	14	Healthy♂	RT, NE	80%	16	6 exercises 3 × 8–12	↑Carotid β	↔SBP/DBP ↔cSBP/cDBP
Cortez-Cooper et al. 2005 [18]	29 ± 1	23	Healthy♀	RT, NE	5–10RM	11	12 exercises 3 × 5–10	↑cfPWV ↔faPWV	↔SBP/DBP
Rakobowchuk et al. 2005 [74]	23 ± 4	28	Healthy♂	RT	≥ 80%	12	4–5 exercises 2-3 × 5–12	↔Carotid β	↔SBP/DBP
Casey et al. 2007 [10]	21 ± 1	24	13♂11♀	RT, NE	8–12RM	12	7 exercises 2 × 8–12	↔cfPWV ↔faPWV ↔crPWV	↔SBP/DBP ↔aSBP/aDBP
Heffeman et al. 2009 [34]	22 ± 1	19 AA, 18 W	Healthy♂	AA, W	8–12RM	6	5 exercises 3 × 8–15	↔cfPWV ↔Carotid β AA:↑Brachial β W:↔Brachial β	↔SBP/DBP ↓aSBP/aDBP
Okamoto et al. 2009 [67]	20 ± 1	10 10	Healthy ULRT:7♂3♀ LLRT:6♂4♀	ULRT, LLRT, NE	80%	10	5 exercises 1 × 8–10	ULRT:↑baPWV LLRT:↔baPWV	↔SBP/DBP
Beck et al. 2013 [5]	18–35	15	PreHT 11♂4♀	NMTC, PHRT, PHET, PHTC	8–12RM	8	7 exercises 2 × 8–12	↔cfPWV ↓crPWV ↓faPWV	↓SBP/DBP ↓aSBP/aDBP
Okamoto et al. 2013 [69]	19 ± 1	10 10	Healthy ALRT:5♂5♀ BLRT:5♂5♀	ALRT, BLRT, NE	50 & 80%	10	5 exercises 5 × 10	ALRT:↔cfPWV ↔faPWV BLRT:↑ cfPWV ↔faPWV	↔SBP/DBP
Croymans et al. 2014 [21]	20–23	28	Healthy♂	RT, NE	8–12RM	12	6–8 exercises 2-3 × 6–15	↔cfPWV	↓SBP/DBP ↓aSBP/aDBP
Middle-aged and older									
Taaffe et al. 2007 [83]	65–78	17	Healthy/PreHT 12♂5♀	1 vs. 3 sets	8RM	20	7 exercises 1 or 3 × 8	–	↓SBP/DBP ↓aSBP/aDBP
Collier et al. 2008 [15]	30–60	15	PreHT/HT 5♂10♀	RT, AT	65% of 10RM	4	9 exercises 3 × 10	↑cfPWV ↑faPWV	↓SBP/DBP
Cortez-Cooper et al. 2008 [18]	52 ± 2	13	Healthy 10♂3♀	RT, CT, FT	70%	13	10 exercises 1 × 8–12	↔cfPWV	↔SBP/DBP
Yoshizawa et al. 2011 [94]	32–59	11	Healthy♀	RT, AT, NE	60%	12	6 exercises 3 × 10	↔cfPWV ↔faPWV	↔SBP/DBP
Figueroa et al. 2011 [28]	47–68	12	Healthy♀	CT, NE	60%	12	9 exercises 1 × 12	↓baPWV	↓SBP/DBP
Jefferson et al. 2015 [38]	65–79	32	Healthy/PreHT 14♂18♀	Diet+RT, RT	70%	20	8 Exercises 3 × 10	↔baPWV	↔SBP/DBP
Rossow et al. 2014 [78]	50–64	13	Healthy/ PreHT♀	Young Older	80%	8	6 exercises 3 × 8–12	↔cfPWV ↔faPWV	↔SBP/DBP

♀, female; ♂, male; *PWV*, pulse wave velocity; *BP*, blood pressure; *preHT*, prehypertensive; *HT*, hypertensive; *AT*, aerobic training; *RT*, resistance training; *CT*, combined resistance and aerobic training; *FT*, flexibility training; *AA*, African-American; *W*, white; *UL*, upper-limb RT; *LL*, lower-limb RT; *NE*, non-exercising control; *ALRT*, low-intensity resistance training after high-intensity resistance training; *BLRT*, low-intensity resistance training before high-intensity resistance training; *NMTC*, normotensive healthy non-exercising control; *PHRT*, prehypertensive resistance training; *PHET*, prehypertensive endurance training; *PHTC*, prehypertensive time control; *SBP*, systolic BP (aSBP, aortic SBP; cSBP, carotid SBP); *DBP*, diastolic BP (aDBP, aortic DBP; cDBP, carotid DBP); *PWV* (faPWV, femoral-ankle PWV or femoral-dorsalis pedis; *baPWV*, brachial-ankle PWV; *cfPWV*, carotid-femoral PWV; *crPWV*, carotid-radial pulse wave velocity); *β*; beta-stiffness

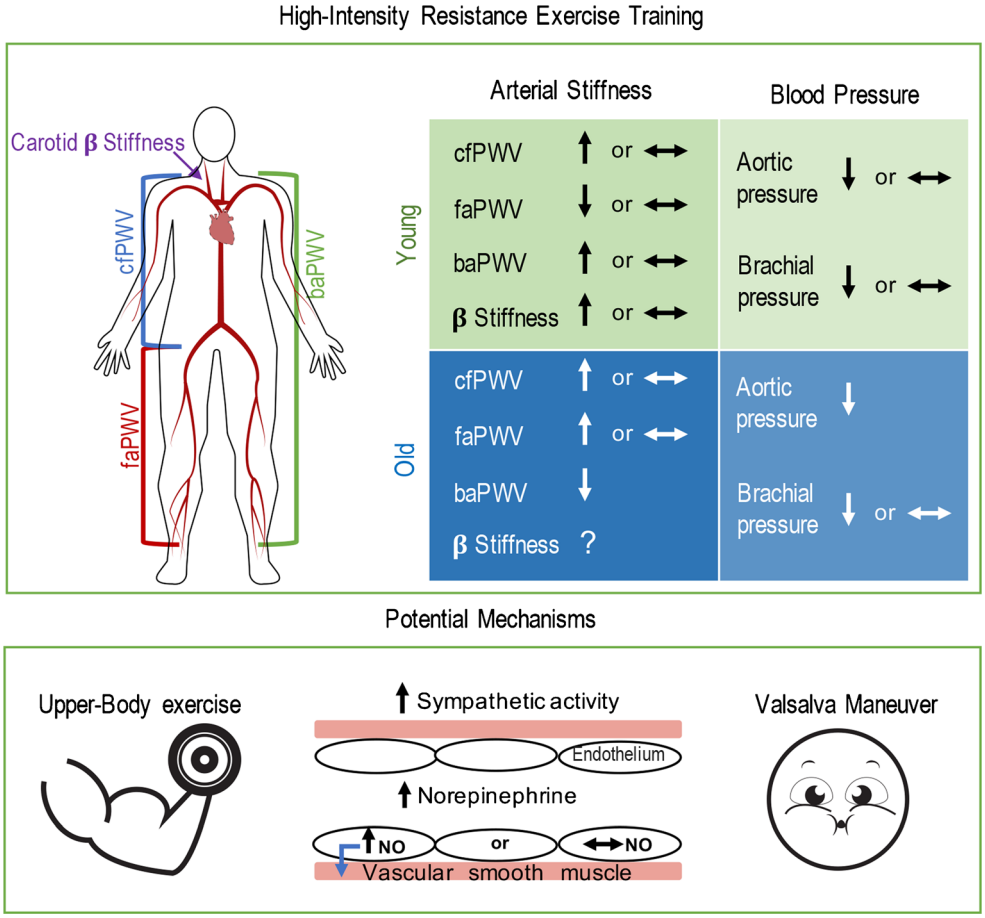

Fig. 1 Potential mechanisms for the increases in arterial stiffness in young, middle-aged and older adults. High-intensity resistance training and concurrent Valsalva maneuver increase blood pressure acutely and sympathetic activity chronically, contributing to increase arterial stiffness. In contrast, decreases in PWV may be explained by improved endothelial NO-mediated vasodilatory function. *Abbreviations:* cfPWV, carotid-femoral pulse wave velocity; faPWV, femoral-ankle pulse wave velocity; baPWV, brachial-ankle pulse wave velocity; β stiffness, carotid artery beta-stiffness; NO, nitric oxide

of motion. With greater core stabilization demands and thus potentially greater increases in intra-abdominal pressure during exercise, theoretically, this modality of RT could cause greater increases in arterial stiffness in young individuals. However, one only study has examined the effects of primarily high-intensity free weight (e.g., squats, deadlifts, overhead press, etc.) training on arterial stiffness [34]. This study did not observe any changes in arterial stiffness following 8 weeks of RT in young healthy African American and Caucasian men [34]. Collectively, whole-body high-intensity RT in young individuals who have low baseline levels of arterial stiffness may increase arterial stiffness by ~ 11% [56]. However, an increase in arterial stiffness of that magnitude in young, healthy individuals may not be enough to cause adverse cardiovascular events [56].

Although the increase in arterial stiffness following high-intensity resistance exercise in young individuals may not pose a significant health risk, research has examined training methods that could attenuate the increase in stiffness. Maintaining, rather than increasing, the RT volume over a 12-week period of high-intensity RT prevent increases in arterial stiffness [10]. The addition of low-intensity (50% 1RM) following high-intensity (80% 1RM) exercises has been shown to prevent the increases in arterial stiffness that occur following high-intensity RT [69]. It is suggested that lower and moderate-intensity resistance exercise attenuates the elevations in arterial BP and sympathetic nervous system activity that may occur following high-intensity resistance exercise leading to an increase in arterial stiffness (Fig. 1) [69]. Additionally, a combination of RT with aerobic training may be an effective means to prevent arterial stiffening as a meta-analysis has shown a small reduction in arterial stiffness followed combined training [58]. Notably, Figueroa et al. [28] observed a reduction in systemic arterial stiffness (baPWV) in post-menopausal women (~ 54 years) following a combination of moderate-intensity (60% 1RM) circuit type resistance exercise followed by 20 min of treadmill walking. This is still in contrast to a more substantial reduction in PWV following aerobic training alone [58]. Overall, it appears that combining high-intensity RT with either aerobic training or low-intensity RT and/or careful manipulation of the RT volume may be an effective strategy to prevent any potential arterial stiffening effects of high-intensity RT.

With aging and menopause, there is a progressive increase in cfPWV and baPWV [88, 95]. Increases in arterial stiffness following high-intensity RT in older adults with already elevated arterial stiffness may be of a greater concern for cardiovascular risk, although this apprehension is commonly

ignored by researchers in RT studies. Three studies have shown no effect of RT on arterial stiffness in middle-aged and/or older adults [18, 78, 94]. Cortez-Cooper et al. [18] observed no change in arterial stiffness following 13 weeks of machine based RT using an intensity of 70% 1RM in middle aged (~ 52 years) men and women. Yoshizawa et al. [94] also found no effects of 12 weeks of moderate-intensity (60% 1RM) resistance exercise on arterial stiffness in middle-aged women. Finally, Rossow et al. [78] observed no change in arterial stiffness following high-intensity (80% 1RM) RT in older (~ 57 years) women. As mentioned, Figueroa et al. [28] observed a decrease in arterial stiffness following combined RT and aerobic training in post-menopausal women. In contrast, one study observed an increase in arterial stiffness (~ 14.5% increase in cfPWV and faPWV) following 4 weeks of moderate-intensity (~ 50% 1RM or 65% 10RM) RT in pre-hypertensive and stage-1 hypertensive middle-aged men and women [15]. A meta-analysis which pooled the aforementioned studies concluded that RT in middle-aged subject does not increase arterial stiffness [56]. Notably, the studies which observed no change [78, 94] or a decrease [28] in arterial stiffness following RT studied women exclusively or women comprised the majority (~ 77%) of the participants in the RT group [18]. The study that observed an increase in arterial stiffness following RT included a majority of men (~ 67%) in the RT group. Thus, in middle-aged and older individuals, it is possible that men are more susceptible to increases in arterial stiffness following RT compared to women although there is a need for more evidence in this topic. Additionally, there is a lack of evidence on how high-intensity (> 80% 1RM) RT effects arterial stiffness in older adults, especially men.

The effects of high-intensity resistance training on blood pressure in young, middle-aged, and older adults

Hypertension is a primary risk factor for cardiovascular diseases [40]. Non-pharmacological strategies for hypertension include physical activity, primarily aerobic exercise supplemented by resistance exercise [52]. The BP-lowering effects of aerobic exercise are well-established [42]. In contrast, fewer studies have investigated the effects of high-intensity RT on BP (Fig. 1). The mode of RT as well as other RT variables may affect the magnitude of the change in resting BP following training.

One of the earlier studies examining the effects of high- (75–85% 1RM) and moderate-intensity (55–65% 1RM) RT in older adults (~ 68 years) revealed that both training intensities were effective in reducing BP although the magnitude of the decrease was greater following moderate-intensity compared to high-intensity RT [87]. However, pooled analysis of multiple studies examining the effect of various RT exercise intensities on BP did not find a significant effect of RT intensity on the change in BP [17]. Of note, the majority of RT studies have used moderate to high training intensities (60–80% 1RM) and thus the effect of different training intensities is not entirely clear. With respect to the type of resistance exercise, isometric or dynamic, a meta-analysis concluded that isometric RT results in greater reductions in BP than either traditional dynamic resistance exercise or even aerobic exercise [9].

The effect of RT on resting BP does not appear to vary between younger and older participants [16]. One investigation which compared the effects of 8-week high-intensity RT on brachial BP observed no reductions in either younger or older women without hypertension [78]. Similarly, 12 to 18 weeks of RT was ineffective to elicit brachial BP reductions in normotensive young adults [10] or in postmenopausal women [11]. Data from these studies may suggest that BP reductions after RT are minimal and insignificant in normotensive participants [17].

While most research on the BP lowering effects has measured brachial BP as the primary outcome, central (aortic) BP is better related to future cardiovascular events [73]. Non-invasive estimation of central BP requires measurement of pressure waveforms from other sites in the arterial tree [53]. Despite the technology becoming more available for estimation of central BP, only a handful of studies have specifically investigated the effects of high-intensity RT on central BP. One of the aforementioned studies which investigated the effects of whole-body resistance exercises without increases in volume [10] did not observe any changes in central BP following 12 weeks of RT in young men and women. In contrast, other investigations observed a central BP-lowering effect of RT. Croymans et al. [21] found central aortic BP was reduced in sedentary and overweight young men following 12 weeks of RT. Similarly, 8-week high-intensity RT was effective in reducing central aortic SBP (~ 10 mmHg) in young adults with elevated BP and stage-1 hypertension [5]. Moreover, Taaffe et al. [83] also observed reductions in central aortic BP following 20 weeks of high-intensity RT in older (~ 70 years) men and women with elevated BP or stage-1 hypertension. Finally, Heffernan et al. [34] also observed reduction in central BP (carotid and aortic SBP) following 8 weeks of high-intensity RT in young African-American and Caucasian men. These studies observed no changes in central arterial stiffness [5, 34, 83]. Of note, the three studies which observed reductions in central BP all included participants with relatively high aortic SBP (> 105 mmHg) [21, 34, 83] compared to the study which did not observe changes in aortic BP in which participants had relatively low central BP (~ 102/74 mmHg) [10]. Importantly, reductions in central BP following RT occur independent of changes in brachial BP or central arterial stiffness [5, 21, 83]. Notably, one of the studies reported that

reduction of aortic BP was attributed to decreases in pressure wave reflection and peripheral (arms and legs) arterial stiffness in young adults [5].

With the limited number of studies that have examined the central BP effects of RT, there is not enough information to conclude if the modality, intensity, or other training variables may impact the anti-hypertensive response to training. Thus, there is a need for more research on the impact of RT on central BP.

The effects of low-intensity resistance training on arterial stiffness and blood pressure in young adults

Several studies have reported that low-intensity RT at 30–50% of 1RM promotes gains in muscle strength and mass that are similar to those reported with high-intensity RT [54, 84, 86]. Low-intensity RT at 40–50% of 1RM with slow movement (≥ 3 s concentric and eccentric phases) and shorter inter-set rest periods (30 s) also increases muscular size and strength as effective as conventional high-intensity RT [63, 84, 86]. In addition, low-intensity RT to exhaustion can increase muscular size and strength to the same degree as high-intensity RT [54].

In contrast to high-intensity RT, several studies have shown that low-intensity RT effectively decreases arterial stiffness (baPWV) in healthy young adults (Fig. 2) [63, 68, 70]. An interesting effect of low-intensity RT when performed after high-intensity RT in the same session is its capacity to suppress the increase in arterial stiffness (baPWV) induced by high-intensity RT alone [69]. Interestingly, arterial stiffness increases when the intensity of RT is reversed. These findings therefore suggest that arterial stiffening is not suppressed by low- before high-intensity RT. Thus, although low- after high-intensity RT suppresses the increase in arterial stiffness, high-intensity RT might neutralize this anti-stiffening effect of low-intensity RT. On the other hand, increased arterial stiffness might depend on the method (e.g., body part, contraction type) of RT. In fact, upper limb RT increases arterial stiffness (baPWV) while lower limb RT has no effect [23, 67]. Moreover, concentric RT (lifting phase) increases arterial stiffness, whereas eccentric RT (lowering phase) does not affect PWV [62]. Furthermore, high-intensity RT with rapid lifting and slow lowering might prevent arterial stiffening [64]. These studies have reported no effect of low-intensity RT on peripheral BP since participants were healthy normotensive adults (Fig. 2). Based on these findings, the intensity of RT, muscles exercise performed, and type of contraction required to maintain cardiovascular health might have to be carefully prescribed, especially in middle-aged older adults with hypertension, CVD, and other chronic diseases or risk factors (e.g., type 2 diabetes, prediabetes, metabolic syndrome) with increased PWV associated with aging and health conditions.

The effects of low-intensity resistance training on arterial stiffness and blood pressure in middle-aged and older adults

The effects of low-intensity RT on arterial stiffness and hemodynamics have been assessed following 4–52 weeks in middle-aged and older adults (40–72 years of age), although the number of studies are limited (Table 2 and Fig. 2). In short-term RT (4–12 weeks) studies, authors reported no impact on arterial stiffness. Fahs et al. [25] utilized unilateral knee extension exercise to examine the effects of low-intensity (30% 1RM) RT with and without blood-flow restriction in normotensive middle-aged adults. Although they reported no effect on SBP or faPWV following 6 weeks of low-intensity RT (Table 2), there were significant increases in vascular conductance (26.2%) in the leg trained without blood-flow restriction. These findings suggest that an RT program with a single-leg exercise for the quadriceps may be not sufficient to improve whole leg arterial stiffness and BP in normotensive adults. Similarly, 16 weeks of low-intensity RT did not cause significant reductions in aortic SBP (− 2 mmHg) in apparently healthy older adults [93]. However, older women tended to have lower SBP than men after RT.

Important factors that influence reductions in BP with RT include BP at baseline and sex. Studies have shown that the BP lowering effect of RT is not evident in individuals with normal BP, and on the other hand, there is greater BP reduction in individuals with hypertension, followed by those with elevated BP [48]. There is a growing number of studies suggesting that women may benefit from low-intensity RT more than men [14, 27, 29, 65]. Evidence suggests that only 4 weeks of low- to moderate-intensity (80% of 10RM) RT may induce more vascular benefits in middle-aged women than men with elevated BP or hypertension [14]. Despite similar reductions in SBP in men and women, RT evoked an increase in cfPWV in men but not in postmenopausal women. In contrast, low-intensity RT decreased systemic PWV (baPWV) but not SBP in normotensive middle-aged women [65]. Although women in the previous two studies [14, 65] had similar age, inclusion of women with normal BP explains the lack of reduction in BP with a decrease in baPWV [65]. To examine potential sex differences on the impact of low-intensity (45% 1RM) RT on aortic BP and wave reflection in older adults, Williams et al. randomized men and women aged 60–75 years to RT and flexibility training in a crossover design [93]. They reported no significant vascular changes following 16 weeks of RT. Although significant reductions in wave reflection indexes

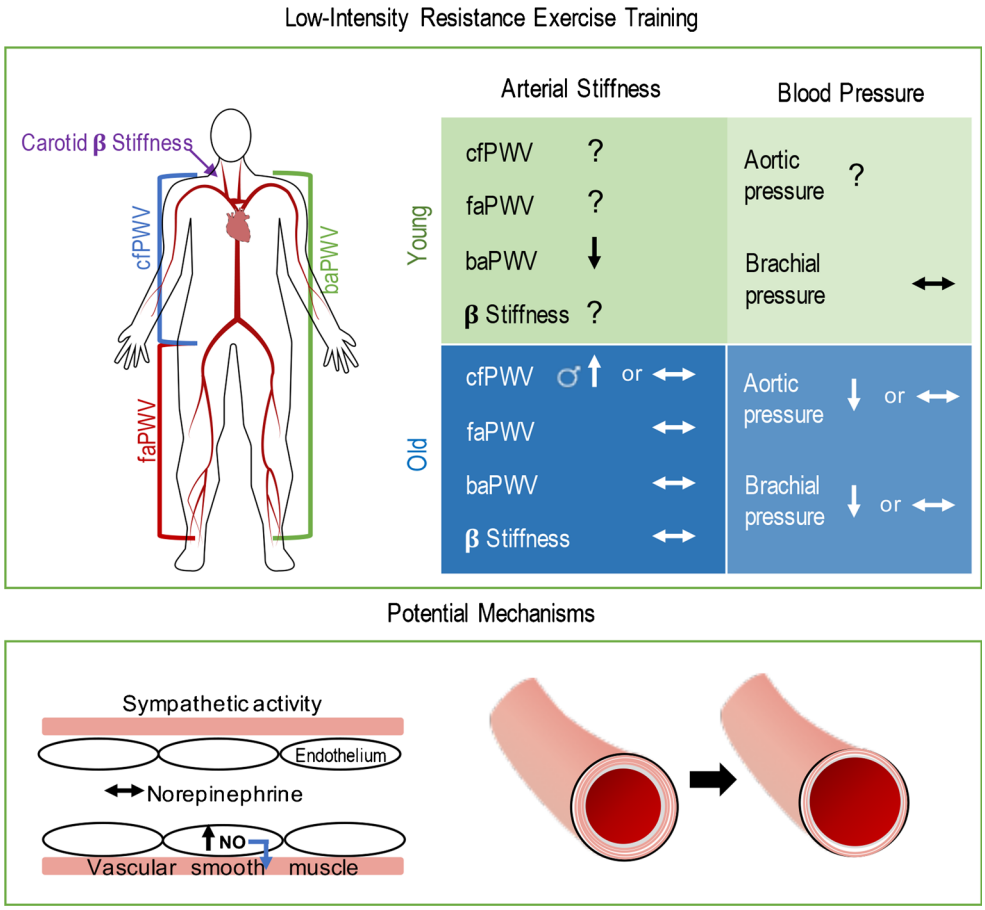

Fig. 2 Potential mechanism for the decrease in PWV in young adults. Low-intensity resistance training does not change plasma norepinephrine levels and increases endothelial NO-mediated vasodilatory function, providing a restrain to sympathetic-mediated vasoconstriction. *Abbreviations:* cfPWV, carotid-femoral pulse wave velocity; faPWV, femoral-ankle pulse wave velocity; baPWV, brachial-ankle pulse wave velocity; β stiffness, carotid artery beta-stiffness; NO, nitric oxide

including augmented pressure (22.2%) and augmentation index (16.0%) were apparent in older women, but not men, the decrease in aortic SBP (4 mmHg) in women was not statistically significant. It was suggested that the duration of the RT might have not been long enough to evoke greater reductions in aortic SBP. More importantly, it has been proposed that upper-body, but not lower-body resistance exercises increase PWV acutely and chronically [23, 67], which may attenuate decreases in BP. Given that RT consisted of three exercises for trunk muscles and one for thigh muscles in the study by Williams et al. [93], more preference should be given to leg resistance exercises. In contrast, it was found that aortic SBP significantly decreased (7 mmHg) following 12 weeks of low-intensity RT with slow movement in obese early postmenopausal women aged 47–60 years with elevated BP and hypertension. Women in this RT intervention performed three sets of four leg exercises. Despite reduction in aortic SBP, low-intensity RT was inefficient to reduce arterial stiffness measured as cfPWV, baPWV, and faPWV, which is in agreement with previous studies that used high-intensity RT [5, 34, 83]. In prehypertensive and never-medicated hypertensive older adults, low- to moderate-intensity RT for 12 weeks reduced aortic SBP and DBP [36]. Although cfPWV was not measured in the previous study, the decrease in forward wave pressure (Pf) may indicate that reduction of proximal aorta stiffness would have contributed to reductions in aortic SBP and Pf. Given that participants in the previous study were predominantly (82%) older women, RT may be considered a more effective strategy in women with elevated hypertension [14, 27]. In a subsequent study in older obese adults aged 65–79 years, moderate-intensity RT (70% 1RM) did not reduced baPWV and brachial SBP after 5-months intervention [38]. An important limitation of the previous study was high variability in the baPWV response to RT, which might be due to inclusion of both sexes and participants with normal BP and hypertension. The only study to our knowledge to report reductions in BP in older men utilized 52 weeks of low-intensity (45% 1RM) whole-body RT [43]. Kim and Kim [43] reported a significant decrease in SBP (6.3%) and diastolic BP (5.2%) with a concurrent increase in brachial artery compliance (2.5%) in hypertensive older (72 years of age) men. At this time, we cannot state whether or not males need longer RT programs to have beneficial vascular

Table 2 Characteristics of participants and low-intensity resistance training protocols assessing chronic effects on arterial stiffness and blood pressure

Study	Age (y)	RT, *n*	Characteristics	Groups	%1RM	Duration (weeks)	RT Protocol (sets × reps)	Arterial stiffness	BP
Young									
Okamoto et al. 2008 [63]	19 ± 1	10	Healthy♂	RT, NE	40–50	8	9 Exercises 5 × 10	↓baPWV	–
Okamoto et al. 2011 [68]	19 ± 1	13	Healthy 10♂ 3♀	RT, NE	50	10	8 Exercises 5 × 10	↓baPWV	↔SBP/DBP
Middle-aged and older									
Collier et al. 2011 [14]	40–60	20	PreHT/HT 10♂10♀	RT, AT	50	4	9 Exercises 3 × 10	♂↑cfPWV ♀↔cfPWV ↔faPWV	♂↓SBP/↔DBP ♀↓SBP/DBP
Figueroa et al. 2013 [27]	54 ± 1	13	PreHT/HT Obese♀	Diet, RT, Diet+RT	40	12	4 Exercises 2-3 × 18–22	–	↓SBP/DBP ↓aSBP/aDBP
Figueroa et al. 2013 [29]	54 ± 1	14	PreHT/HT Obese♀	Diet, RT, Diet+RT	40	12	4 Exercises 2–3 × 18–22	↔cfPWV ↔faPWV ↔baPWV	–
Hefferman et al. 2013 [36]	61 ± 1	11	PreHT/HT 2♂ 9♀	RT, NE	40–60	12	9 Exercises 2 × 12–15	–	↓SBP/DBP ↓aSBP/aDBP
Kim and Kim 2013 [43]	68–72	20	HT♂	RT	45	52	8 Exercises 2 × 10	↑Brachial compliance	↓SBP/DBP
Williams et al. 2013 [93]	60–75	25	PreHT/HT♂♀	RT, ST	45	16	4 Exercises 2–3 × 8–12	–	↔aSBP/aDBP

♀, female; ♂, male; *PWV*, pulse wave velocity; *BP*, blood pressure; *preHT*, prehypertensive; *HT*, hypertensive; *AT*, aerobic training; *RT*, resistance training; *NE*, non-exercising control; *SBP*, systolic blood pressure; *aSBP*, aortic SBP; *aDBP*, aortic diastolic BP; *faPWV*, femoral-ankle PWV; *baPWV*, brachial-ankle PWV; *cfPWV*, carotid-femoral PWV

adaptations when compared to their female counterparts. Investigations observing the effects of low-intensity RT on central hemodynamics and arterial stiffness (PWV) in older adults are severely limited. Taken together, evidence to date suggests that it may be more difficult to regress structural changes in the arterial wall than BP with RT in older adults.

Potential mechanisms

Although the mechanism by which high-intensity RT increases central artery stiffness is not entirely clear, it may be due to elevations in plasma norepinephrine (NE) [67], exaggerated elevations in BP during exercise [47, 49], and repeated use of the Valsalva maneuver (Fig. 1) [35]. A single bout of RT consisting of one set to exhaustion at 75% of 1RM for nine whole-body exercises or four sets at 70–80% of 1RM for two upper-body exercises have increased central carotid (beta) or aortic (cfPWV) stiffness in healthy young adults [22, 23]. Acute intermittent elevations of BP during resistance exercise sessions may influence the increase in central artery stiffness after high-intensity RT.

Some mechanisms have been proposed to explain the reduction in arterial stiffness after low-intensity RT (Fig. 2). Okamoto et al. [66] found that plasma NE remained unchanged and significantly increased after acute low- and high-intensity resistance exercise, respectively. In addition, plasma NE concentrations increase more after 10 weeks of upper- than lower-limb low-intensity RT [67]. Moreover, Okamoto et al. [67] also determined that arterial stiffness (baPWV) was positively correlated with plasma NE concentrations in the upper-limb after RT. Thus, the increase in arterial stiffness might result from complex interactions between sympathetic activity and vascular smooth muscle tone. Therefore, low-intensity RT might reduce arterial stiffness by providing restraint to sympathetic adrenergic vasoconstriction. Thus, high-intensity RT enhances, whereas low-intensity RT with slow lifting and lowering and short inter-set rest periods attenuates the vasoconstriction stimulated by sympathetic activity.

High-intensity RT does not favorably affect endothelial function in healthy young men [41, 64, 75]. However, improvements in brachial artery or forearm endothelial function have been found after moderate- and high-intensity RT in overweight premenopausal women [44, 71], young [4] and middle-aged [14, 15] adults with elevated BP and hypertension. Positive effects on endothelial function have been shown after low-intensity RT with slow lifting and lowering and short inter-set rest periods even in healthy adults [63, 68]. The continuous generation of force during exercise at 40% maximum voluntary

contraction suppresses both muscle blood inflow and outflow due to an increased intramuscular pressure, causing intramuscular hypoxia [8, 55]. Vascular tone is influenced by the balance between local (endothelial nitric oxide [NO] and muscle hypoxia-related metabolites) vasodilators and sympathetic vasoconstrictor reflexes to maintain adequate muscle blood flow and arterial BP [37]. Endothelin-1 (ET-1), a potent endothelial-derived vasoconstrictor, may partially explain the increased cfPWV in resistance-trained male athletes [72]. The mechanisms underlying vascular benefits of RT remain unclear in humans. The primary mechanism may involve an increase in BP and shear stress stimulation on endothelial cells during exercise bouts. Repeated periods of increased blood flow and shear stress during training mechanically stimulates endothelial NO synthesis [31]. Improvements in vascular function after 6 months of RT and aerobic training were evident in the exercised limb, suggesting that vascular benefits may be associated with increased local shear stress rather than the exercise modality [81]. Increased NO bioavailability by RT may decrease production of ET-1 [50, 51]. In rats, RT failed to decrease cfPWV and increase NO production, suggesting that RT does not activate NO signaling pathway (endothelial NO synthase/Akt phosphorylation) in endothelial cells as occurred with endurance training [33]. Human studies are needed to investigate molecular-signaling mechanisms of the impact of low- and high-intensity RT on PWV. Collectively, the studies examined in this review support the notion that low- to high-intensity RT may reduce BP by improving endothelial function (Figs. 1 and 2).

Conclusions

This review article highlights evidence on the effects of high- and low-intensity RT on arterial stiffness and BP in young and older adults. Increased central arterial stiffness and SBP are associated with aging and cardiovascular diseases. In addition, the age-related loss of skeletal muscle mass/strength is also associated with arterial stiffening and hypertension. Therefore, RT should be the primary exercise modality to improve these age-related vascular and muscular abnormalities. Although the potential for adverse effects of high-intensity RT on central arterial stiffness exists, most of the studies have shown that high-intensity RT does not affect arterial stiffness whereas low-intensity RT can decrease or not change arterial stiffness. Importantly, RT independently of the intensity has demonstrated effectiveness to improve BP (peripheral and central) and peripheral artery endothelial function. Further studies are needed to examine the impact of RT on central arterial stiffness, BP, and wave reflection in middle-aged and older adults.

Acknowledgments We appreciate the technical assistance provided by Arun Maharaj and Dr. Amber McCord.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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