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Structural and Functional Adaptations of Blood Vessels to Running: A Literature Review

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ABSTRACT

Running is commonly practiced and linked to cardiovascular advantages, but its effects on vascular structure and function are not well understood. This review gathers existing evidence regarding vascular adaptations related to running, emphasizing endothelial function, arterial stiffness, arterial diameter, and intima-media thickness (IMT). Regular moderate running typically promotes endothelial function, often shown through maintained or improved flow-mediated dilation (FMD), although immediate post-exercise responses differ based on race length, training level, and arterial location. Studies on pulse wave velocity (PWV) show varied results: marathon training over time might lead to greater arterial stiffness, while ultramarathon athletes frequently exhibit PWV levels beneath age-related expectations, indicating adaptations that depend on individual conditions and training load. Structural remodeling, especially the increase of conduit artery diameter in the lower limbs, is consistently observed in longitudinal studies resulting from ongoing hemodynamic stimuli experienced during running. In contrast, IMT results are varied; some studies show some evidence of decreased or stabilized wall thickness, whereas others demonstrate slight increased, probably affected by metabolic or hemodynamic elements like exercise-related hypertension. Together, this information suggests that vascular adaptations to running vary and are influenced by factors such as training volume, individual conditions, and differences in the methodology of studies. Additional longitudinal studies with standardized vascular evaluations are needed to better understand the mechanistic pathways and clinical effects of running and vascular adaptations.

Keywords: running; endothelial function; flow-mediated dilation; pulse wave velocity; arterial stiffness; arterial remodeling; intima-media thickness

INTRODUCTION

In recent years, running has become one of the most prevalent forms of physical activity in society. Running is an accessible form of exercise that is becoming increasingly common. The health benefits of physical activity are well known. According to the World Health Organisation, all adults should undertake 150–300 minutes of moderate-intensity, or 75–150 minutes of vigorous-intensity physical activity, or some equivalent combination of moderate-intensity and vigorous-intensity aerobic physical activity per week [1]. Therefore, a review has been conducted on the health benefits of running, particularly on the cardiovascular system. Regular and moderate running can significantly reduce the risk of death from any cause, including the risk of cardiovascular disease (CVD). Physical activity in the form of moderate running affects heart function better and prevents arrhythmia [2].

Several studies have reported that vascular function is related to a person's quality of life. Brunner et al. (2011) found a negative correlation between Pulse Wave Velocity (PWV), a parameter of vascular function, and the physical component of quality of life [3]. In addition, another research found that vascular status components measured using the Ankle Brachial Index (ABI) and cardio-ankle vascular index (CAVI) showed improved quality of life [4]. Therefore, it is important to maintain vascular health.

One physical activity that can affect vascular health is running. A study reported that running at the right intensity also improves the functioning of blood vessels, makes arteries more elastic, and lowers blood pressure, which is especially important for older people [3]. Several previous studies have examined the impact of running on vascular function.

Therefore, this review aims to summarise the current evidence on vascular adaptations to running based on a wide range of studies, including studies on recreational running, marathon running, and ultra-endurance running (UER).

FLOW MEDIATED DILATION

Flow-mediated dilation (FMD) is the most widely used non-invasive method to assess endothelial function by measuring the ability of an artery to dilate in response to increased blood flow and shear stress. The primary mechanism underlying FMD depends on the bioavailability of nitric oxide (NO), synthesized by endothelial nitric oxide synthase (eNOS) in response to elevated shear forces along the endothelial wall. Activation of eNOS increases NO production, which diffuses into vascular smooth muscle cells and causes endothelium-dependent vasodilation.

Consequently, higher FMD values reflect preserved endothelial health, whereas reduced FMD is considered an early marker of endothelial dysfunction and atherosclerotic risk. FMD is typically measured using high-resolution ultrasound after a 5-minute ischemic cuff occlusion, which induces reactive hyperemia and a transient increase in blood flow [5]. FMD values are highly sensitive to physiological factors such as age, sex, habitual physical activity, and metabolic status, and can be favorably modified by long-term aerobic exercise that repeatedly elevates shear stress and enhances endothelial vasodilatory capacity [6].

Evidence from studies investigating runners suggests that there is a relationship between running and FMD. In recreational populations, habitual running is generally associated with enhanced endothelial function. For instance, a cross-sectional study reported significantly higher brachial artery FMD in trained runners than in untrained runners, indicating a protective vascular effect of regular running [7]. While this finding is consistent with exercise physiology theory, the study is cross-sectional and has a small sample. Another study that compared regularly trained marathon runners and recreationally active controls has aligned results, with marathon runners having higher FMD compared to controls [8,9]. Beak et al. (2022) compared low-intensity running performed with or without blood flow restriction (BFR) among recreational runners. Although no significant difference in brachial FMD was detected post-training, both groups showed numerically higher post-intervention brachial FMD values, suggesting potential endothelial adaptation to consistent low-intensity running even in the absence of high mechanical load, though the study did not conduct formal pre-post statistical testing [10]. Another study evaluated runners completing a 50 km ultramarathon and measured brachial artery FMD at baseline, 1 hour, and 24 hours post-race. It was found that there were no significant changes in FMD at 1 hour and 24 hours after a 50 km ultramarathon. This is as a consequence of the runner's arterial diameter may already be structurally larger to accommodate the repetitive functional responsiveness needed to maintain blood pressure during exercise. Therefore, the change in the diameter would not be to the same degree as an untrained individual [11]. In addition, another study that assessed superficial femoral artery FMD in ultramarathon participants over distances of 25-km, 50-km, 80-km, and 160-km, observed a significant post-race decrease in FMD only in the 50 km group, whereas other distances showed no significant changes, suggesting that transient endothelial cell damage may occur under specific race conditions but is not a universal effect of prolonged running [12]. These divergent results may reflect heterogeneity in measurement sites (brachial vs. femoral arteries), participant conditioning, or race-specific metabolic stress, suggesting that acute FMD responses are context-dependent and difficult to generalize. Naylor et al. (2021) revealed that limb-specific structural and functional vascular adaptation is evident in athletes, which may be influenced by exercise modality, as there were differences in femoral artery FMD despite no significant differences in brachial FMD between runners and weightlifters compared to controls [13]. Another study reported that highly trained runners had lower baseline brachial FMD compared with moderately trained individuals due to a larger increase in basal brachial diameter, highlighting that very high training volumes may be associated with structural or functional adaptations that reduce resting FMD [14].

Overall, the literature suggests that recreational running is generally associated with preserved or enhanced endothelial function, but the evidence has important limitations that reduce the strength of this conclusion. Many studies rely heavily on cross-sectional designs, limiting causal inference, and often include small sample sizes with heterogeneous training intensities, durations, and participant fitness levels. Methodological variability in FMD measurement, such as differences in artery site, imaging technique, and timing relative to exercise, further complicates interpretation. Acute exercise effects are also inconsistent, varying with race, distance, intensity, and baseline conditioning. Although habitual recreational running appears beneficial, high training volumes may lead to lower resting FMD, which does not necessarily indicate impaired vascular health but could reflect structural adaptations. To clarify the relationship between running, endothelial adaptation, and vascular risk, future research should prioritize prospective, randomized interventions with standardized FMD protocols.

PULSE WAVE VELOCITY

Pulse wave velocity (PWV), particularly carotid femoral PWV (cfPWV), is the current reference standard for assessing central arterial stiffness and reflects the speed at which the systolic pressure wave travels along the aorta. Because PWV is highly sensitive to acute hemodynamic conditions, any interpretation of changes must consider both vascular tone and the underlying structural properties

of the arterial wall [15]. In the context of recreational running, accumulating evidence suggests that running induces acute functional responses and longer-term structural adaptations that may lead to either reductions or increases in arterial stiffness depending on training history, training load, and individual physiological characteristics. For instance, a study reported that completing a half-marathon did not produce significant group-level changes in cfPWV when measured 18–22 hours post-race in either recreational or high-level runners. Nevertheless, the individual direction and magnitude of cfPWV change were significantly associated with race time and athlete category: faster and more highly trained runners exhibited mild increases in cfPWV during recovery, whereas slower, less trained runners tended to show stable or slightly reduced values. These findings emphasize that even within a single endurance event, recovery-related arterial stiffness responses are heterogeneous and strongly influenced by the athlete's physiological profile, possibly reflecting differences in sympathetic activation, hydration status, vascular tone, and the extent of exercise-induced muscle damage or inflammation [16]. On another study that compare regularly trained marathon runners and recreationally active controls showed increased PWV in trained marathon runners. Thus marathon runners have increased aortic stiffness, it may be as a result of of elastic component damage due to excessive exercise burden. However, it may also represent an adaptive process that leads to a higher, but favorable travel for waves, and possibly relates to marathon runners reach their peak later in life compared to other athletes [8].

Acute and chronic adaptations have been explored through cross-sectional comparisons. A widely cited study observed that chronic effect of exercise in marathon runners with many years of high volume endurance training exhibited higher cfPWV (mean 6.89 m/s) compared with matched recreationally active controls (6.33 m/s), suggesting that effects of exercise may be detrimental beyond a certain point (increase arterial stiffness). Mechanistically, several. repeated and, particularly, excessive stress (partially due to lower heart rate and resultant increased stroke volume and aortic distension) imposed on the elastic elements of the aortic wall may lead to their mechanical fatigue. Study that corroborates this explanation is that the intensity of exercise (min/day) was an independent determinant of increased aortic stiffness [17].

Other plausible mechanisms include increased inflammatory state that has been observed in marathon runners after the race and has been shown to unfavorably affect arterial stiffness. Furthermore, increased oxidative stress, as it has been shown in endurance and ultra-endurance athletes may have a contributing role. Importantly, acute effect of marathon was it does not appear to have an effect on changes of wave reflection indexes as PWV. The higher heart rate and peripheral vasodilation after the race could have influenced the blood volume within the aorta and consequently the dimension and elastic properties of the aorta contributing to the lack of change in PWV values before and after the race in the marathon runners. As hydration state is shown to influence both arterial stiffness and wave reflections, dehydration in marathon runners after the race may have partially affected the measurements [17].

More recent evidence provides an additional perspective. A study evaluated ultramarathon runners preparing for a 161 km event and found that their mean cfPWV was 6.5 ± 1.0 m/s, with 86% of athletes exhibiting values below age-predicted normative averages. This finding demonstrates that even in the context of very high training volumes, lifelong endurance running can coexist with favorable central arterial stiffness [18]. The apparent discrepancy with Vlachopoulos et al. (2010) may be explained by differences in athlete phenotype, recovery and hydration status at assessment, distribution of training intensity (high-volume but predominately low-intensity running among ultramarathoners), or unmeasured confounders such as genetics, habitual blood pressure, arterial geometry, or dietary patterns. Additionally, another study showed that cfPWV can acutely decrease after prolonged endurance running, with recreational marathoners exhibiting reductions from approximately 8.5 to 7.9 m/s within two hours post-race. interestingly, this response was more closely associated with BMI than with performance level, suggesting that adiposity and metabolic factors may modulate vascular responsiveness even among active individuals [19].

In summary, current evidence demonstrates that the impact of recreational endurance running on central arterial stiffness is highly context-dependent, with acute and chronic effects that vary according to training load, athlete phenotype, and hemodynamic conditions at the time of measurement. Acute studies show minimal group-level change in cfPWV after endurance events, but significant interindividual variability driven by differences in autonomic recovery, vascular tone, hydration, and post-exercise inflammatory or mechanical stress. Long-term findings are equally heterogeneous: while veteran high-volume "recreational" marathoners exhibit elevated cfPWV suggestive of cumulative pulsatile stress and arterial remodeling, ultramarathon runners with similarly high lifetime mileage demonstrate cfPWV values at or below age-predicted norms. These mixed results likely reflect differences in training intensity distribution, metabolic health, habitual blood pressure, recovery quality, and underlying genetic vascular traits. Methodologically, most studies are limited by small samples, inconsistent timing of cfPWV assessment, inadequate adjustment for distending pressure, and variable categorization of "recreational" runners, which obscure causal inference and increase heterogeneity. Overall, from the current literatures, it can be assumed that endurance running universally enhances arterial compliance, suggesting an individualized, threshold-dependent adaptation

pattern that remains incompletely understood and requires more longitudinal and standardized investigation.

ARTERIAL DIAMETER

In vascular physiology research, common measurements of arterial diameter include the lumen diameter of large conduit arteries such as the femoral and brachial arteries, typically assessed by high-resolution ultrasound. These structural parameters (e.g., resting lumen diameter) reflect chronic remodeling of the artery in response to long-term hemodynamic stimuli. A larger resting diameter in conduit arteries may indicate outward (expansive) remodeling, which often results from sustained increases in blood flow and shear stress. Such remodeling is considered beneficial, as it enables a greater capacity for blood delivery to muscles during exercise and may reduce wall shear stress at rest [6].

Study conducted by Kapilevich et al. (2020) divided subjects into five groups with different training characteristics (sedentary, two different groups of active subjects, two different groups of well-trained runners and weightlifters). Basal brachial artery diameter between the five groups was not different. However, in response to physical work capacity test, brachial basal diameter was significantly increase and larger increase was observed in well-trained athletes [14].

There is limited yet informative evidence indicating that running, particularly long-distance or marathon-specific training, contributes to structural remodeling of conduit arteries. The most direct evidence comes from the longitudinal study by Hafner et al. (2016), who investigated first-time marathoners undergoing a standardized 4–6-month marathon training program. Using high-resolution B-mode ultrasound, they measured popliteal and brachial artery lumen diameter (LD) and intima-media thickness (IMT) at baseline and after completion of the training period. Participants adhered to individualized running programs progressively increasing weekly mileage toward a marathon target. The main finding was a significant increase in popliteal artery LD in the running group (from 4.73 ± 0.72 mm to 5.11 ± 0.72 mm, $p = 0.002$), whereas non-running controls showed no change. The popliteal artery demonstrated the greatest remodelling, consistent with limb-specific hemodynamic load during running. Furthermore, brachial artery, or non-trained limb, dimensions were significantly altered by the 12 weeks of training suggest that arterial adaptations to exercise training can be manifested in the non-trained limb (e.g. brachial artery) as well as the trained limb (e.g. popliteal artery). Hafner et al. (2016) interpreted these results as evidence that arterial dimension adaptations to marathon training are dependent on training volume and that higher exercise intensities may extend the influence of localized mechanisms into arteries that are peripheral to the trained limbs.. However, sample sizes were modest, and the study relied on self-reported training logs, introducing variability in running exposure [20].

In Naylor et al. (2021), a large cross-sectional study comparing elite male endurance athletes (including competitive distance runners) with non-athlete controls, femoral artery structure was assessed using standardized ultrasound imaging with surface area normalized correction for body size. Although causality cannot be established in this design, the authors observed that runners displayed significantly larger femoral artery diameters after normalization for body surface area (3.51 mm in runners vs. 3.25 mm in controls, $p < 0.05$). Their methodological strength included strict categorization of athletes by sport type (endurance, strength, power), careful body-size correction, and measurement of both brachial and femoral arteries to evaluate limb-specificity. The greater femoral diameter in runners suggests chronic exposure to high-flow locomotor limb exercise promotes conduit artery expansion [13]. However, as the athletes were already well-trained, the enlargement could partially reflect selection effects (i.e., individuals with naturally larger arteries excelling in endurance sports), and the absence of baseline or longitudinal data makes it impossible to confirm training-induced structural change.

Taken together, these studies suggest that running is associated with outward remodeling of conduit arteries, particularly in the lower limbs, as shown by increased lumen diameter in individuals who regularly participate in sustained running. Nevertheless, the number of running-specific interventional studies remains small. The marathon-training study by Hafner et al. (2016), while providing the strongest longitudinal evidence, was not randomized and may be influenced by selection bias, variability in adherence, and uncontrolled differences in baseline physical activity. Similarly, the elite athlete comparison by Naylor et al. (2021) is limited by its cross-sectional design, which obscure causal inference and raises the possibility that individuals with naturally larger femoral arteries may self-select into endurance running. There is also high heterogeneity across studies in training intensity, weekly mileage, duration of training history, and participant characteristics, all of which limit the extent of generality of the findings. Furthermore, Kapilevich et al. (2020) and many available studies do not account for important confounders such as cross-training habits, nutritional status, genetic predisposition, or lifetime running exposure, making it difficult to fully isolate the independent effect of running on arterial structure and function.

INTIMA-MEDIA THICKNESS

Intima media thickness (IMT) of large arteries, particularly the common carotid artery and peripheral arteries such as the popliteal or femoral artery, is routinely measured by high-resolution B-mode ultrasound as a non-invasive biomarker of

arterial structure and subclinical vascular disease. IMT combines the thickness of the intimal and medial layers of the arterial wall and reflects cumulative exposure to mechanical stress, metabolic challenge, and hemodynamic load over time [21]. Clinically, increased IMT is strongly associated with a higher risk of atherosclerosis and cardiovascular events, while stable or reduced IMT progression is considered protective, as it suggests favorable vascular remodeling or slower arterial aging [8].

Evidence specifically on running and IMT remains limited but informative. In a well-controlled longitudinal intervention, Hafner et al. (2016) recruited 14 first-time marathon trainees and 11 control participants, measuring brachial, popliteal, and carotid IMT (and other wall metrics) before and after 12 weeks of structured marathon training. They used B-mode ultrasound to assess total wall thickness (TWT), wall-to-lumen ratio (W:L), and IMT, along with lumen diameter. After training, they observed a significant decrease in brachial TWT (from 0.99 mm to 0.84 mm, $p = 0.007$) and a 0.07 mm reduction in brachial IMT ($p = 0.032$), whereas popliteal IMT reduction (~0.03 mm) was not statistically significant [20]. Carotid IMT did not change significantly, suggesting that local (limb) adaptations may be more sensitive to the running stimulus than central arteries. In another long-term study, Müller et al. (2017) followed 38 male marathon runners over approximately 3.8 years and found that carotid IMT increased by 0.05 ± 0.09 mm, though this progression was unrelated to running volume or competition frequency; instead, higher blood glucose levels predicted greater thickening. Complementing these findings, a study compared marathon runners with recreationally active non-runners and reported significantly lower carotid IMT in the runners (0.56 ± 0.11 mm vs. 0.63 ± 0.07 mm), indicating favorable arterial wall remodeling associated with sustained running exposure [9]. Another earlier cross-sectional study by Kardara et al. (2011) similarly showed that long-term marathon runners had lower common carotid IMT (0.58 mm vs. 0.63 mm) compared with controls, despite demonstrating higher aortic stiffness, an observation termed the "marathon paradox." Additionally, Yoon and Kim (2024) studied long-distance runners stratified by their blood pressure response during maximal running tests and found that runners with exercise-induced hypertension had significantly higher carotid IMT than runners with normal responses suggest that exercise induced hypertension is a factor that accelerates the changes in IMT of long-distance runners rather than the effects of high intensity exercise, with the increase in carotid IMT has been shown to be an important indicator of elevated blood pressure during exercise [22].

In summary, the primary evidence indicates that running may have small impact on arterial wall thickness, especially in non-central (limb) arteries or brachial sites. However, the studies on carotid IMT are show different results: some studies report lower IMT in runners while others show slight progression or no clear association. The limited number of running-specific longitudinal trials limit the ability to make strong causal claims. Furthermore, cross-sectional designs carry risk of selection bias (runners who maintain high training volumes may inherently have more favorable vascular profiles) and may not fully adjust for confounding factors such as metabolic health, blood-pressure response, and lifetime training exposure. It is also difficult to determine if running directly slows, stabilises, or conversely accelerates artery wall thickening due to the use of surrogate measurements (IMT) without standardised ultrasonography methods and uneven follow-up times between studies.

CONCLUSIONS

Running induces a wide spectrum of vascular adaptations that vary according to training volume, intensity, and individual conditions. While moderate habitual running generally supports endothelial function and promotes favorable structural remodeling, responses in arterial stiffness and intima–media thickness remain heterogeneous. These findings suggest that vascular adaptations to running are not uniform but context-dependent, highlighting the need for more standardized, longitudinal studies to clarify their mechanistic and clinical significance.

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