Improvements in arterial stiffness and flow-mediated dilatation by concurrent training are independent of body weight changes

Mejoras en la rigidez arterial y dilatación mediante ejercicio físico combinado son independientes de la reducción del peso corporal

Authors

Cristian Alvarez [1]
Pedro Delgado-Floody [2]
Christian Campos-Jara [1]
Cristóbal Durán-Marín [1]
Daniel Jerez-Mayorga [3]
David C. Andrade [4]
Cristian Caparros-Manosalva [5,6]
Gabriel Marzuca-Nassr [2,6]
Omar Andrade-Mayorga [2]
Gabriel Rojas Rojas [1]

[1] Universidad Andrés Bello (Chile)
[2] Universidad de La Frontera (Chile)
[3] University of Granada (Spain)
[4] Universidad de Antofagasta (Chile)
[5] University of Talca (Chile)
[6] Interuniversity Center for Healthy Aging (Chile)

Corresponding author:
Cristian Alvarez
cristian.alvarez@unab.cl

Received: 13-07-25
Accepted: 03-09-25

How to cite in APA

Alvarez, C., Delgado-Floody, P., Campos-Jara, C., Durán-Marín, C., Jerez-Mayorga, D., Andrade, D. C., Caparros-Manosalva, C., Marzuca-Nassr, G., Andrade-Mayorga, O., & Rojas Rojas, G. (2025). Improvements in arterial stiffness and flow-mediated dilatation by concurrent training are independent of body weight changes. *Retos, 72*, 835-849. https://doi.org/10.47197/retos.v72.117115

Abstract

Introduction: Although exercise is known to improve vascular outcomes associated with weight loss, there is limited evidence of whether similar benefits occur in the absence of weight loss.
Objective: To examine the effects of 6-week concurrent training on pulse wave velocity (PWV) and flow-mediated dilation (FMD) in adults who were responders and nonresponders to 'weight loss' to exercise.
Methodology: A secondary analysis of an experimental randomized controlled clinical trial was conducted in 60 adult participants (BMI: 29.7 kg/m^2) reported into 3 groups; weight loss responders' (WLR$_{ET}$, *n*=14), 'weight loss nonresponders to exercise' (WLNR$_{ET}$, *n*=14), and a control group (CG, *n*=30). Participants underwent a 6-week intervention consisting of three sessions per week of concurrent high-intensity interval and resistance training where delta changes (Δ) of pulse wave velocity (ΔPWV) and flow-mediated dilation (ΔFMD) were reported.
Results: After intervention and comparing groups WLR$_{ET}$ vs. WLNR$_{ET}$, there were similar significant changes in outcomes; ΔPWV (-0.9 vs. -0.8 m/s^{-1}), ΔFMD (6.5 vs. 6.5%), both outcomes different vs. CG *P*<0.05. Likewise, the prevalence of responders and nonresponders was comparable ΔPWV (Rs: 78.5%; NRs 21.4%) and ΔFMD (Rs: 57.1%; NRs 42.8%). Despite significant superior ΔDBP decreases in WLNR$_{ET}$ (-5.5 mmHg vs. -1.3 mmHg, *P*<0.05), no other differences were detected for other outcomes.
Conclusions: Adult participants classified as weight loss nonresponders (WLNR$_{ET}$) also experienced reductions in ΔPWV and increased the ΔFMD in similar physiological adaptations to WLR$_{ET}$. These findings are supported by additional benefits observed in WLNR$_{ET}$, including reductions in blood pressure and improvements in vascular function.

Keywords

Arterial hypertension; arterial stiffness; blood pressure; endothelial dysfunction; flow-mediated dilation; obesity.

Resumen

Introducción: Aunque el ejercicio mejora los resultados vasculares asociados con la pérdida de peso, existe evidencia limitada sobre si beneficios similares ocurren en ausencia de cambios en el peso.
Objetivo: Examinar los efectos de un programa de 6 semanas de ejercicio concurrente de sobre la velocidad de onda de pulso (PWV) y la dilatación mediada por flujo (FMD) en adultos que respondieron o no respondieron a la pérdida de peso al ejercicio.
Metodología: Análisis secundario de un ensayo clínico aleatorizado y controlado en 60 adultos (IMC: 29.7 kg/m^2), distribuidos en tres grupos: respondedores (WLR$_{ET}$, *n*=14) y no respondedores a la pérdida de peso (WLNR$_{ET}$, *n*=14) y un grupo control (CG, *n*=30). La intervención consistió en tres sesiones semanales de ejercicio concurrente de intervalos de alta intensidad y fuerza. Se evaluaron los cambios (Δ) en PWV y FMD.
Resultados: Al comparar WLR$_{ET}$ y WLNR$_{ET}$ se observaron cambios significativos y similares en ΔPWV (-0.9 vs. -0.8 m·s^{-1}) y ΔFMD (6.5 vs. 6.5%), ambos diferentes frente al grupo control (*P*<0.05). La prevalencia de respondedores y no respondedores también fue comparable: ΔPWV (Rs: 78.5%; NRs: 21.4%) y ΔFMD (Rs: 57.1%; NRs: 42.8%). A pesar de una mayor reducción de la presión diastólica en WLNR$_{ET}$ (-5.5 vs. -1.3 mmHg; *P*<0.05), no se detectaron otras diferencias significativas.
Conclusiones: Los adultos no respondedores a la pérdida de peso también reducen la PWV y aumentan la FMD, con adaptaciones fisiológicas similares a los respondedores. Estos hallazgos se ven reforzados por beneficios adicionales en respondedores, incluyendo descensos en la presión arterial.

Palabras clave

Hipertensión arterial; rigidez arterial; presión arterial; disfunción endotelial; dilatación-mediada por flujo; obesidad.

Introduction

Hypertension (HTN) is a non-communicable disease of high prevalence that usually coexists with other morbidities such as diabetes, metabolic syndrome, dyslipidaemia or non-alcoholic fatty liver disease (Jones et al., 2025). It is well established that most cases of hypertension and its associated comorbidities are driven by modifiable risk factors including low physical activity, unhealthy diet, poor water consumption, excessive salt and coffee consumption, tobacco use, and alcohol intake, because all of these factors can contribute to increased blood pressure (Jones et al., 2025). Additionally, considering the common physical inactivity behaviour and their excess of body weight, it is commonly recommended by several health professionals to 'lose weight' in this profile of subjects.

Hypertension is a major risk factor for cardiovascular morbidity and mortality, where the American Heart Association (2018) reported a prevalence of 45.6% in the adult U.S. population (Wyss et al., 2020). In Chile, the prevalence of HTN is around 27.6% in adults aged 18 to 64 years (Petermann et al., 2017) however, in older adults aged 65 years and above the HTN prevalence is sharply increased to ~73% (Minsal, 2017). The relevance of treating early HTN or high blood pressure is their significant association with other major vascular abnormalities such as endothelial dysfunction (EDys). EDys is related to future plaque accumulation in the arterial wall, and atherosclerosis disease. Flow-mediated dilation (FMD) of the brachial artery is a non-invasive gold-standard method for assessing endothelial function and diagnosing EDys. On the other hand, subjects with a higher number of modifiable risk factors for HTN, they typically exhibit greater arterial stiffness (measured by aortic pulse wave velocity, PWV), that report the structural condition of the vascular wall in the endothelium (Kim et al., 2022). Thus, PWV values exceeding (>10 $m \cdot s^{-1}$) are associated with increased cardiovascular and cerebrovascular disease (Liu et al., 2025).

Exercise training is a physical therapy for HTN and is widely recommended by the American College of Sports Medicine, the American Diabetes Association, the American College of Cardiology, American Heart Association, and the European Society of Cardiology among other relevant institutions. Modalities such as moderate-intensity continuous (MICT), resistance (RT), high-intensity interval (HIIT), and concurrent training (CT, a combination of MICT and RT) can be applied to populations with HTN (Pedro Delgado-Floody et al., 2022). From here, it is frequently recommended in clinical guidelines to 'lose weight' as a strategy for improving cardiometabolic health. For example, under morbid obesity conditions where bariatric surgery strategy is the only way to improve health, HTN patients can associate the body weight loss with a better blood pressure level (Schiavon et al., 2024). Similarly, before bariatric surgery, these HTN patients are also encouraged to adopt lifestyle modifications and to lose 5 to 10% of body weight before bariatric surgery to ensure a more successful intervention (Delgado-Floody et al., 2020). However, novel evidence from exercise interventions in populations with diabetes mellitus for example, revealed that despite diabetes patients not losing body weight, they still showed improvement in their glucose control after exercise training. Early evidence have also shown that after 12 weeks of MICT without body weight loss, overweight/obese adults showed improved endothelium-dependent vasodilation capacity (tested by forearm blood flow in response to intra-arterial infusion of acetylcholine and sodium nitroprusside) (Mestek et al., 2010). Similarly, 12 weeks of MICT without weight loss effects, reported to decrease fasting glucose in type 2 diabetes mellitus (T2DM) patients (Dekker et al., 2007). However, there is poor information regarding short-term interventions (*i.e.*, ≤6 weeks) of concurrent training using HIIT plus RT ($CT_{HIIT+RT}$) at blood pressure, arterial stiffness and vascular dilatation in high blood pressure subjects. It could be probably that endothelial 'functional' improvements can be seen before 'structural' changes in vascular outcomes.

From here, as the weight loss response strategy can be easily reported by scales in clinical context, it is commonly thought that subjects that underwent exercise training regimes should look for weight loss, where all those who do not respond (*i.e.*, weight loss nonresponders) could apparently not perceive blood pressure or vascular benefits. Nonresponders to weight loss after exercise training ($WLNR_{ET}$) are all those participants who do not show a beneficial response (*e.g.*, weight loss in comparison with other peers) based on some statistical approaches proposed and previous literature reports in normotensive and hypertensive populations (Delgado-Floody et al., 2020). In this line, there is poor information regarding those $WLNR_{ET}$ and their response to blood pressure and EDys vascular outcomes, particularly by decreasing arterial stiffness by PWV or increasing FMD. For example, part of the physiological

mechanisms how? exercise training increases the glucose control in T2DM patients is promoting Glut-4 carrier translocation to the myocyte membrane and thus increase glucose uptake as insulin-independent and fat oxidation independent manner (Whytock & Goodpaster, 2025). On the other hand, there is scarcity of evidence regarding the effects and potential mechanisms on how exercise could improve vascular parameters in humans) without major weight changes. This study aimed to examine the effects of a 6-week $CT_{HIIT+RT}$ intervention on PWV and FMD in adults who were nonresponders to 'weight loss' after this exercise intervention period. We hypothesized that independent of body weight loss, those subjects reported as $WLNR_{ET}$ could also reduce arterial stiffness and increase vasodilation, similarly to WLR_{ET} peers.

Method

Participants

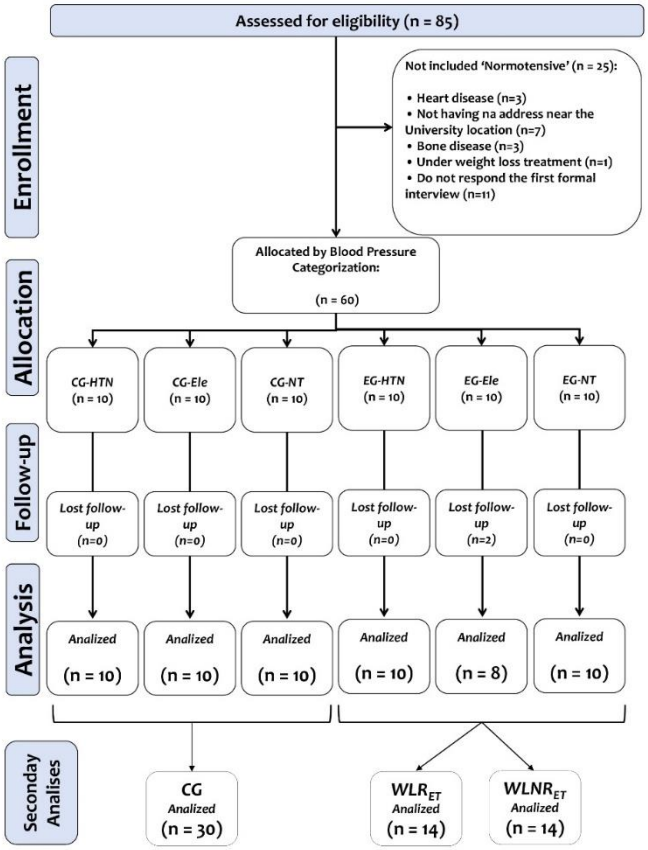
This study is a secondary analysis of our original 'VASCU-HEALTH' study, which is a randomized controlled clinical trial developed in sixty adult participants of a university community (Alvarez et al., 2023).

The sample included neighborhood, social group members, sports club affiliates, together with faculties, staff, and students part of the university. After a face-to-face interview, and screening, the participants took part in 6 weeks of $CT_{HIIT+RT}$. This study was supported by the Biological Science Research Unit (VRI) (DI-01-CBC/22) and approved by the of the Ethical Committee of the Universidad Andres Bello (N° 026/2022), following the Declaration of Helsinki for human studies. All participants signed an informed consent previous study participation. The study is registered at ClinicalTrials.gov ID: NCT05710653 (Register 02nd December 2023).

Criteria of inclusion were: *i*) hypertensive, elevated blood pressure both (*i.e.,* treated/untreated with pharmacotherapy), according with the AHA 2018 categorization (Whelton et al., 2018) or normotensive condition, *ii*) normal [*i.e.*, using body mass index BMI 18.5 to 24.9 kg/m^2], overweight ([BMI 25 to 29.9 kg/m^2], or obesity condition [BMI ≥30 and <40 kg/m^2]), *iii*) elevated fasting glucose or diabetes mellitus (T2DM, *i.e.*, treated with pharmacotherapy), and *iv*) living near the exercise laboratory (*i.e.,* to facilitate a good adherence). Criteria of exclusion were: *i*) history of cardiac rhythm (*i.e.*, by ECG) abnormalities, diagnosis of cardiovascular disease other than HTN, or vasculopathy, *ii*) uncontrolled stage 3 of hypertension, *iii*) diabetes complications such as varicose ulcers, nephropathies, *iv*) skeletal muscle abnormalities (*e.g.*, knee, or hip arthrosis, muscle pain), *v*) using weight loss treatment/pharmacotherapy or being active in exercise training programs (or within the past three months), and *vi*) use other pharmacotherapy that can influence body weight loss.

The sample size was calculated a priori by G*Power 3.1.9.7 software (Germany), with at least (n=10) participants per group, looking for a 'moderate' effect size, with 80% power, and ≤5% α error. In the enrolment stage of the original interventional study, (n=85) individuals were screened, and after that, (n=78) were considered eligible who were allocated 1:3 to a groups; control group hypertensive (CG-HTN), control group elevated blood pressure (CG-Ele), control group normotensive (CG-NT), exercise group hypertensive (EG-HTN), exercise group elevated blood pressure (EG-Ele), or exercise group normotensive (EG-NT). Thus, to the current study, we included participants under different blood pressure control, but also under 'normal weight', 'overweight', and 'obesity' condition, and after that, taking into account all participants we re-categorized the sample in those who were; weight loss responders to exercise training (WLR_{ET}, n=14), weight loss nonresponders to exercise training ($WLNR_{ET}$, n=14) and the control groups that we re-name as (CG, n=30). Additional details about the original intervention are available in (Alvarez et al., 2024). The study protocol to the current study can be seen from study design in (Figure 1).

Figure 1. CONSORT Study design. Original group distribution is shown as; (CG-HTN) Control group hypertensive, (CG-Ele) Control group elevated blood pressure, (CG-NT) Control group normotensive). (EG-HTN) Exercise group hypertensive, (EG-Ele) Exercise group elevated blood pressure, (EG-NT) Exercise group normotensive. Current secondary analyses groups are shown as; (CG) Control group, (WLR_{ET}) Weight loss responders to exercise training, and ($WLNR_{ET}$) Weight loss nonresponders to exercise training.



Blood pressure measurement

Blood pressure was measured following the AHA 2018 guidelines, defined as follows: systolic (SBP)/diastolic (DBP) blood pressure <120/80 mmHg, elevated blood pressure 120 to 129/80 mmHg, stage 1 of hypertension 130 to 139/80 to 89 mmHg, and finally, stage 2 hypertension ≥140/90 mmHg (Whelton et al., 2018). Two measurements were taken from the left arm using a cuff placed on the brachial artery (i.e., by a cuff in the brachial artery) and after a 10-minute rest position seated using an automatic monitor (OMRON™ model HEM 7114, Japan). From here, the delta of pulse pressure (ΔPP) and mean arterial pressure (ΔMAP) were calculated using both ΔSBP and ΔDBP data. We also measured the delta of systolic (ΔSBP_{ank}) and diastolic blood pressure of the left ankle (ΔDBP_{ank}). To do this, each patient remains in resting position for 5 minutes and using the same equipment for brachial blood pressure (OMRON™ model HEM 7114, Japan) SBP_{ank} and DBP_{ank} were obtained.

Anthropometric, body composition

The delta pre-post of body weight (ΔWeight, in kg), body fat percentage (ΔBF%), and skeletal muscle mass percentage (ΔSMM%) were measured by a bioimpedance analyzer (OMRON model HBF-514 Healthcare Inc., Lake Forest, IL, United States). Height (m) was measured with a stadiometer (HEALTH O METER™ model Professional, Sunbeam Products, Inc., Chicago, IL, United States). Waist circumference (cm) and their delta (ΔWC) was measured using an inextensible tape (SECA™, United States). Body mass index (kg/m^2) and its pre-post delta change (ΔBMI) was calculated using both weight divided by square of height (WHO, 2000) Antropometric and body composition data can be found in (**Table 1**).

Pulse wave velocity and vascular outcomes

Aortic PWV was measured using oscillometric pressure traces from the brachial artery in the upper left arm (measured in m/s) with an Arteriograph device after a 20-minute rest in a supine position (Arteriograph, TENSIOMED™, Budapest, Hungary). Data analysis was conducted with Arteriograph Software v.1.9.9.2. This equipment's blood pressure assessment algorithm is validated (Ring et al., 2014). PWV values exceeding 10 $m \cdot s^{-1}$ indicate elevated arterial stiffness, correlating with increased cardiovascular risk (Mancia et al., 2013). Other vascular outcomes were ejection duration (ED), diastolic reflection area (DRA), systolic area index (SAI), diastolic area index (DAI), return time (RT), using a non-invasive brachial cuff equipment (Arteriograph, TENSIOMED™, Budapest, Hungary) equipment (Morales et al., 2015).

Flow-mediated dilation of the brachial artery

For FMD, all participants remained in supine position for 20 minutes on a stretcher. Using an ultrasound equipment (GE™, Model LOGIQ-E PRO, Milwaukee, United States) with a 7–12 MHz linear-array transducer. The brachial artery was measured on the left side in a longitudinal plane 1–3 cm proximal to the antecubital fossa (pulsed Doppler) before the occlusion. The ultrasound transducer was supported with an adjustable mechanical metal arm precision holder (EDI™, Progetti e Sviluppo, Italy), to maintain stable the arm position, and avoid evaluator bias. A blood pressure cuff was positioned on the forearm and inflated at 50 mmHg over the baseline SBP during 5 minutes (RIESTER model ri-san™, Jungingen, Germany) (Thijssen et al., 2019). A baseline image before the occlusion, a 3-minute video (60 s before to stop the occlusion that was maintained until 2 minutes after cuff deflation), and finally, a last image (post occlusion), was recorded to compare with baseline measurements. The peak artery diameter after cuff deflation were recorded by storing each 10 s images. FMD is calculated as the percentage (%) rise of peak diameter from the preceding baseline diameter and the image after deflation (Atkinson, 2014) using the following formulae:

$$\text{FMD (\%)} = \frac{[(peak\ diameter\ -\ baseline\ diameter)] * 100}{baseline\ diameter}$$

A FMD >6.6 % proposed by the European Society of Hypertension, and European Society of Cardiology as the acceptable cut-off point for categorizing as a normal vasodilation. Reliability was estimated using intraclass correlation coefficients based on four baseline measurements of 0.91 for baseline diameter and of 0.83 for FMD (previously data) (Ramírez-Vélez et al., 2019). More details about the FMD procedure developed have been previously shown (Alvarez et al., 2024).

Concurrent training

The $CT_{HIIT+RT}$ rehabilitation program included five one-minute intervals at 80 to 100% of peak heart rate (HR_{peak}) of HIIT, each interval was followed by a rest period (*i.e.,* without movement) until heart rate returned to ≤70% of $HR_{peak.}$ The HIIT exercise was developed using vertical bikes (Impulse™, model PS 300, Sparta, Chile). For RT, participants completed three sets of three exercises such as biceps curl, shoulder press, and back exercises. Each RT exercise set was of 60 seconds, performed at 20-50% of one-repetition maximum (1RM), and was followed by a rest period until a subjective Borg scale rating of 1 to 3 was achieved (*i.e*., of the 1 to 10 points modified Borg scale).

Responders and nonresponders to weight changes

To the current study of secondary analysis, after the end of the 6-week $CT_{HIIT+RT}$ intervention, we used the technical error of measurement (TE) calculated from previous studies of TE: 0.5 kg to identify all those participants from the original groups EG-HTN, EG-Ele and EG-NT and participants were reclassified as weight loss responders (WLR_{ET}, n=14) and weight loss nonresponders ($WLNR_{ET}$, n=14) as was above explained. After both groups of interest were identified, we selected the control group (CG, n=30) to have balanced three groups for comparisons (**Figure 1**). Thus, we registered each delta changes (Δ) main and secondary outcomes from the WLR_{ET} and $WLNR_{ET}$ groups to be compared vs. the CG.

Responders and nonresponders to pulse wave velocity and flow-mediated dilation

After the results of the $CT_{HIIT+RT}$ intervention, all participants part of the CG, WLR_{ET} and $WLNR_{ET}$ group of our analyses were categorized according to those who reduced PWV ≤ 0.5 $m \cdot s^{-1}$ in favor of beneficial changes (i.e., negative values –0.5 $m \cdot s^{-1}$ or a major reduction), who were considered as responders (Rs)

for improving PWV. By contrast, all those with minor PWV decreases were considered as nonresponders (NRs). For FMD, were considered Rs all those who after exercise intervention showed a value of ≥ 0.9% of brachial vasodilation. All participants with FMD values <9% were considered as NRs.

Data analysis

Data are presented as the mean ± standard deviation (SD). Normality and homoscedasticity assumptions were tested using Shapiro-Wilk and Levene's (*F*) tests, respectively. One-way analysis of variance (ANOVA) was used to compare between groups variables at baseline (WLR_{ET} vs. $WLNR_{ET}$; WLR_{ET} vs. CG, and $WLNR_{ET}$ vs. CG). To the first original study, to test pre-post changes, a repeated measure 2-way ANOVA was applied to identify training-induced changes (group x time) in all outcomes (data not shown). To the present study, when an *F* value was significant, Tuckey's *post hoc* test was applied to establish group comparisons at pre- and post-test at $P<0.05$. One-way analysis of variance (ANOVA) was used to compare between groups at baseline. When significant results were detected in delta changes (Δ) of each group, the Tukey's *post hoc* was applied to identify differences among groups. Additionally, the Cohen's *d* effect size was applied with threshold values at 0.20, 0.60, 1.2, and 2.0 for small, moderate, large, and very large effects, respectively (Hopkins et al., 2009). Statistical analyses were developed using Prism 8.0 software (Graph Pad, San Diego, CA, United States). The alpha level was fixed at ($P\leq0.05$) for all statistical significance.

Results

Baseline characteristics

At baseline, and comparing WLR_{ET} *vs*. $WLNR_{ET}$, WLR_{ET} *vs*. CG and $WLNR_{ET}$ *vs*. CG, there were multiple differences among groups in outcomes ΔWeight, ΔWC, ΔBMI, ΔBody fat (%), ΔBody fat (kg), ΔMuscle mass (%), ΔLean mass (kg), ΔBMR, ΔBody age and ΔArterial age (**Table 1**).

Table 1. Anthropometric and body composition characteristics of participants.

Outcomes	Time	WLR_{ET}[a]	$WLNR_{ET}$[b]	CG[c]	$F_{()}$, *P*value, ES
Age (y)	Pre	44.6±15.1	44.0±12.1	41.6±12.6	*F*(0.19), *P*=0.825, 0.01
Height (cm)	Pre	164.0±0.08	164.0±0.09	163.0±0.09	*F*(0.08), *P*=0.914, 0.004
Weight (kg)	Pre	78.6±10.5	80.3±15.3	80.2±12.9	*F*(0.07), *P*=0.928, 0.003
	Δ	−1.36±0.78[bc]	0.71±0.90[c]	0.10±0.13	*F*(20.71), *P*<0.0001, 0.42
Body mass index (kg/m²)	Pre	28.8±2.3	30.2±3.8	30.2±3.4	*F*(0.81), *P*=0.448, 0.03
	Δ	−0.50±0.29[bc]	0.27±0.34[c]	0.04±0.24	*F*(23.20), *P*<0.0001, 0.53
Waist circumference (cm)	Pre	100.7±6.4	99.7±9.1	99.0±7.7	*F*(0.16), *P*=0.844, 0.009
	Δ	−3.64±2.06[bc]	−2.21±3.01[c]	0.10±1.93	*F*(14.33), *P*<0.0001, 0.34
Body composition					
Body fat (%)	Pre	39.9±5.7	40.3±6.9	38.7±8.6	*F*(0.14), *P*=0.867, 0.008
	Δ	−3.14±4.28[bc]	0.07±1.65[c]	0.70±1.42	*F*(12.02), *P*<0.0001, 0.30
Skeletal muscle mass (%)	Pre	26.2±3.7	25.7±3.9	30.0±7.4	*F*(0.28), *P*=0.117, 0.11
	Δ	1.97±2.08[bc]	0.27±1.26	0.20±0.87	*F*(8.99), *P*=0.004, 0.24
Fat-free mass (kg)	Pre	47.5±9.6	48.2±12.0	48.6±9.9	*F*(0.04), *P*=0.959, 0.002
	Δ	1.34±3.46[bc]	0.32±1.03	−0.53±0.89	*F*(4.89), *P*=0.011, 0.15

Data are shown as mean and ±SD. Groups are described as: (WLR_{ET}) Weight loss responders to exercise training. ($WLNR_{ET}$) Weight loss nonresponders to exercise training. (CG) Control group. (ES) Cohen *d* effect size. (Δ) Delta pre-post. (*F*) Levene test. (ES) Cohen *d* effect size. (a, b, c) Denotes significant differences among groups at $P<0.05$ by Tukey *post hoc*. Bold values denotes significant interactions.

At baseline, there were no statistical differences among main and secondary vascular outcomes (Table 2).

Table 2. Vascular and secondary vascular baseline characteristics of participants at baseline.

Outcomes	WLR_{ET}[a]	$WLNR_{ET}$[b]	CG[c]	$F_{()}$, *P*value, ES
Vascular	9	13	21	
Flow-mediated dilation (%)	7.0±2.3	12.0±6.3	7.4±5.7	*F*(3.08), *P*=0.056, 0.13
Pulse wave velocity (cm/s^{-1})	8.6±1.4	9.0±1.5	8.0±1.3	*F*(0.95), *P*=0.396, 0.05
Secondary vascular outcomes				
Systolic blood pressure (mmHg)	122.4±16.1	128.4±12.0	128.0±14.3	*F*(1.66), *P*=0.204, 0.08
Diastolic blood pressure (mmHg)	78.9±6.9	86.8±9.3	83.4±10.1	*F*(2.84), *P*=0.072, 0.14

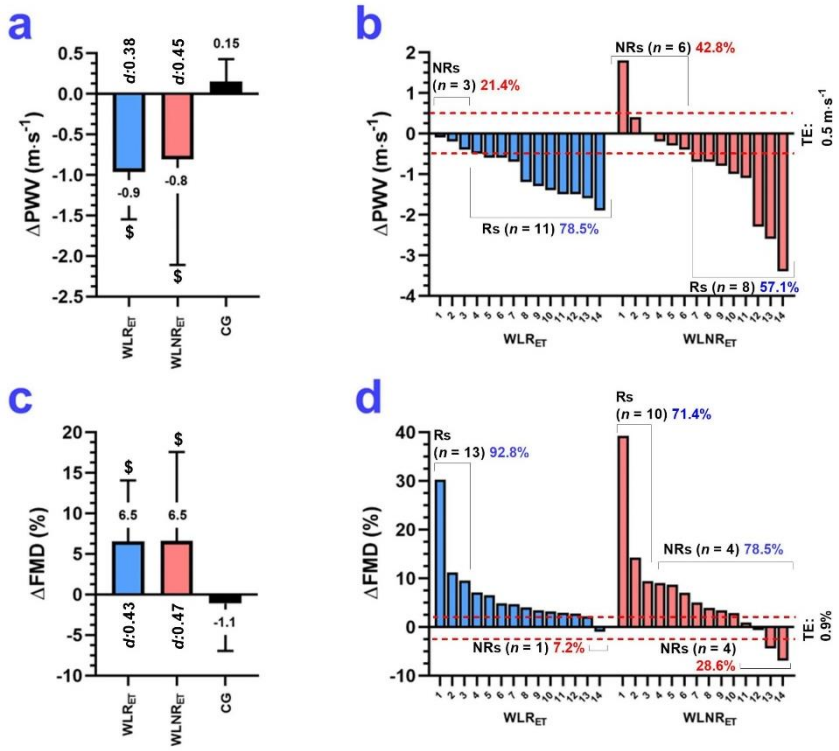
Pulse pressure (mmHg)	43.5±9.2	41.6±2.7	44.6±4.2	*F*(2.71), *P*=0.094, 0.14
Mean arterial pressure (mmHg)	93.4±9.0	100.7±9.1	98.3±10.1	*F*(2.85), *P*=0.071, 0.14
SBP_{ank} (mmHg)	134.2±21.0	136.3±11.1	141.9±16.4	*F*(2.11), *P*=0.136, 0.11
DBP_{ank} (mmHg)	84.6±22.0	81.8±11.8	81.1±6.1	*F*(0.26), *P*=0.768, 0.01
Ejection duration	308.7±13.7	313.9±12.9	304.5±21.0	*F*(0.91), *P*=0.410, 0.01
Systolic area index	46.3±4.6	46.9±3.1	45.6±6.1	*F*(0.26), *P*=0.768, 0.04
Return time	123.4±20.2	118.6±23.2	132.9±23.2	*F*(1.54), *P*=0.226, 0.07
Ankle brachial index	1.15±0.11	1.10±0.06	1.16±0.08	*F*(1.32), *P*=0.276, 0.06
Systolic blood pressure aortic	125.4±13.6	112.4±13.0	116.9±14.2	*F*(2.39), *P*=0.104, 0.10
Augmentation index	-17.3±23.0	-11.7±27.9	-21.1±18.2	*F*(0.58), *P*=0.563, 0.02

Data are shown as mean and ±SD. Groups are described as: (WLR_{ET}) Weight loss responders to exercise training. ($WLNR_{ET}$) Weight loss nonresponders to exercise training. (CG) Control group. (ES) Cohen *d* effect size. (Δ) Delta pre-post. (*F*) Levene test. (ES) Cohen *d* effect size. (a, b, c) Denotes significant differences among groups at *P*<0.05 by Tukey *post hoc*.

Pulse wave velocity and flow-mediated dilation among groups (Main outcomes)

At ΔPWV, there were significant differences between WLR_{ET} *vs.* CG (–0.9±0.6 *vs.* 0.1±0.2, *p*<0.0001 [*diff.* 10 m·s^{-1}]), and between $WLNR_{ET}$ *vs.* CG (–0.8±1.2 *vs.* 0.1±0.2, *p*<0.0001 [*diff.* 9.0 m·s^{-1}]) (Figure 2a). There were no significant differences comparing NRs between WLR_{ET} (*n*=3; 21.4%) *vs.* $WLNR_{ET}$ (*n*=6; 42.8%) (Figure 2a). At ΔFMD, there were significant differences between WLR_{ET} *vs.* CG (6.5±6.6 *vs.* –1.1±5.8, *p*<0.0001 [*diff.* 7.6 m·s^{-1}]), and between $WLNR_{ET}$ *vs.* CG (6.5±10.1 *vs.* –1.1±5.8, *p*<0.0001 [*diff.* 7.6 m·s^{-1}]) (Figure 2c). There were no significant differences comparing NRs between WLR_{ET} (*n*=3; 21.4%) *vs.* $WLNR_{ET}$ (*n*=6; 42.8%). There were no significant differences comparing NRs between WLR_{ET} (*n*=3; 21.4%) *vs.* $WLNR_{ET}$ (*n*=3; 21.4%) (Figure 2d).

Figure 2. Delta changes comparison in outcomes pulse wave velocity and flow-mediated dilation in adults' responders and nonresponders to body weight loss after 6-week concurrent training. Groups are described as; (WLR_{ET}) Weight loss responders to exercise training. ($WLNR_{ET}$) Weight loss nonresponders to exercise training. (CG) Control group of inactive adults. Outcomes are described as; (PWV) Pulse wave velocity. (FMD) Flow-mediated dilation. (Rs) Responders. (NRs) Nonresponders. ($) Denotes significant differences vs. CG at *P*<0.05. (*d*) Denotes Cohen *d* effect size at *P*<0.05.

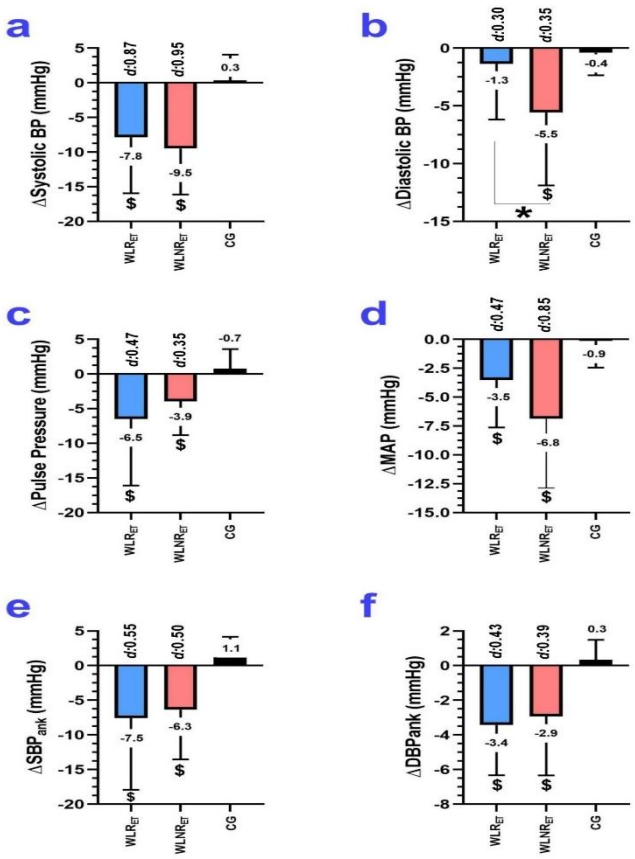


Blood pressure among groups (secondary outcomes)

At ΔSBP, there were no significant differences between WLRET vs. WLNRET (Figure 3a). In the same ΔSBP outcome, there were significant differences between WLRET vs. CG (–7.8±7.1 vs. 0.3±0.4, P<0.0001 [diff. 8.1 mmHg]), and between WLNRET vs. CG (–9.5±6.8 vs. 0.3±0.4, P<0.0001 [diff. 9.8 mmHg]) (Figure 3a). In ΔDBP, there were significant differences between WLNRET vs. CG (–5.5±5.8 vs.

–1.3±1.1, P<0.0001 [diff. 4.2 mmHg]) (Figure 3b). Significant differences were detected between WLRET vs. WLNRET (–1.3 vs. –5.5 mmHg) (Figure 3b). In ΔPP, there were significant differences between WLRET vs. CG (–6.5±9.9 vs. 0.7±2.1, P<0.0001 [diff. 7.2 mmHg]) (Figure 3c). In ΔMAP, there were significant differences between WLNRET vs. CG (–6.8±6.1 vs. –0.9±2.5, P<0.0001 [diff. 5.9 mmHg]), (Figure 3d). In ΔSBPank, there were significant differences between WLRET vs. CG (–7.5±9.9 vs. 1.1±2.2, P<0.0001 [diff. 8.6 mmHg]), and between WLNRET vs. CG (–6.3±6.2 vs. 1.1±2.2, P<0.0001 [diff. 7.4 mmHg]) (Figure 3e). At ΔDBP, there were significant differences between WLRET vs. CG (–3.4±3.0 vs. 0.3±1.2, P<0.0001 [diff. 3.7 mmHg]), and between WLNRET vs. CG (–2.9±3.3 vs. 0.3±0.4, P<0.0001 [diff. 3.2 mmHg]) (Figure 3f). No significant differences were observed comparing WLRET vs. WLNRET in outcomes ΔSBP, ΔPP, ΔMAP, ΔSBPank and ΔDBPank (Figure 3a-f).

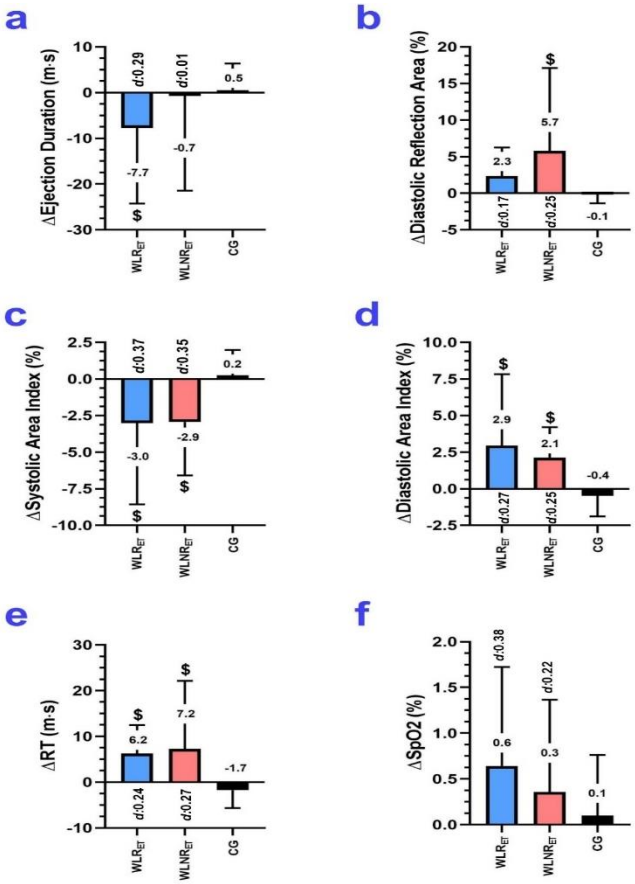
Figure 3. Delta changes comparison in outcomes pulse wave velocity and flow-mediated dilation in adult responders and nonresponders to body weight loss after 6-week concurrent training. Groups are described as; (WLR_{ET}) Weight loss responders to exercise training. ($WLNR_{ET}$) Weight loss nonresponders to exercise training. (CG) Control group of inactive adults. Outcomes are described as; (PWV) Pulse wave velocity. (FMD) Flow-mediated dilation. ($) Denotes significant differences vs. CG at *P*<0.05. (*) Denotes significant differences between WLR_{ET} vs. $WLNR_{ET}$ at *P*<0.05. (*d*) Denotes Cohen *d* effect size at *P*<0.05.



Secondary vascular outcomes among groups

At ΔEjection Duration, there were significant differences between WLR_{ET} *vs.* CG (–7.7±18.2 *vs.* 0.5±5.6, *P*<0.0001 [*diff.* 8.2 m·s]), (Figure 4a). There were significant differences in ΔDiastolic Reflection Area between $WLNR_{ET}$ *vs.* CG (–5.7±2.9 *vs.* –0.1±2.2, *P*<0.0001 [*diff.* 5.8 %]), (Figure 4b). In ΔSystolic Area Index, there were significant differences between WLR_{ET} *vs.* CG (3.0±5.3 *vs.* 0.2±2.4, *P*<0.0001 [*diff.* 3.2 %]), and between $WLNR_{ET}$ *vs.* CG (–2.9±4.3 *vs.* 0.2±2.4, *P*<0.0001 [*diff.* 3.1 %]) (Figure 4c). In ΔDiastolic Area Index, there were significant differences between WLR_{ET} *vs.* CG (2.9±5.1 *vs.* –0.4±2.4, *P*<0.0001 [*diff.* 3.3 %]), and between $WLNR_{ET}$ *vs.* CG (2.1±4.9 *vs.* –0.4±2.1, *P*<0.0001 [*diff.* 2.5 %]) (Figure 4d). At ΔReturn Time, there were significant differences between WLR_{ET} *vs.* CG (6.2±5.4 *vs.* –1.7±4.5, *P*<0.0001 [*diff.* 7.9 m·s]) and between $WLNR_{ET}$ *vs.* CG (7.2±17.1 *vs.* –1.7±4.5, *P*<0.0001 [*diff.* 8.9 m·s]) (Figure 4e). No significant differences were detected in ΔSpO_2 among groups (Figure 4f). No significant differences were detected in ΔEjection Duration, ΔDiastolic Reflection Area, ΔSystolic Area Index, ΔDiastolic Area Index, and ΔReturn Time comparing WLR_{ET} *vs.* $WLNR_{ET}$ group.

Figure 4. Delta changes after 6 weeks of $CT_{HIIT+RT}$ in secondary vascular outcomes of adult responders and nonresponders to weight loss. Groups are described as; (WLR_{ET}) Weight loss responders to exercise training. ($WLNR_{ET}$) Weight loss nonresponders to exercise training. (CG) Control group of inactive adults. Outcomes are described as; (RT) Return time ($) Denotes significant differences vs. CG at $P<0.05$. (*d*) Denotes Cohen *d* effect size at $P<0.05$.



Discussion

This study aimed to examine the effects of a 6 week of $CT_{HIIT+RT}$ on pulse wave velocity and flow-mediated dilation in adults who were nonresponders to 'weight loss' after this exercise intervention period. The main findings of this study showed *i*) 6 weeks of $CT_{HIIT+RT}$ exercise intervention show that $WLNR_{ET}$ participants can also decrease ΔPWV (*i.e.*, –0.8 $m \cdot s^{-1}$) and increase ΔFMD (*i.e.*, 6.5 %) in similar magnitude (*i.e.,* without significant between-group differences) in comparison with WLR_{ET} peers (Figure 2), and *ii*) similar benefits were observed in both WLR_{ET} and $WLNR_{ET}$ groups in outcomes ΔSBP, ΔPP, ΔMAP, ΔSBPank, ΔDBPank (Figure 3), where no significant differences were observed between $WLNR_{ET}$ vs. WLR_{ET} groups (Figure 4). Other secondary vascular parameters also improved in both $WLNR_{ET}$ and WLR_{ET} groups, including decreased ΔEjection Duration, increased ΔDiastolic Reflection Area, decreased ΔSystolic Area Index, increased ΔDiastolic Area Index, increased ΔReturn Time (Figure 4).

In recent years, research on inter-individual variability in response to exercise training or the exercise nonresponders known as 'nonresponders' referring to individuals who exhibit abnormal responses despite adherence to exercise training, have been increasing interest (Pedro Delgado-Floody et al., 2022; Ramírez-Vélez et al., 2020). However, there is a scarcity of studies reporting the vascular response after exercise therapy in subjects who do not show some 'weight loss' response as our $WLNR_{ET}$ group in comparisons with other peers who lose weight.

From here, our primary result revealed that those $WLNR_{ET}$ reduced ΔPWV (–0.8 $m \cdot s^{-1}$) and increased ΔFMD (6.5%) with similar adaptations to those observed in WLR_{ET}. A study from (Pedralli et al., 2020) reported that after 8 weeks of three different exercise modalities, there were similar ΔFMD increases from endurance training (ΔFMD3.2%), resistance training (ΔFMD4.0%), and concurrent training of endurance plus resistance training (ΔFMD6.8%). Interestingly, when considering all participants of the

three exercise groups of this study, there were similar weight loss among these (endurance ΔWeight –1.2 kg; resistance ΔWeight –0.4 kg; concurrent training ΔWeight –1.2 kg), most of whom would have been reclassified as 'weight loss responders' in our current secondary analysis. Additionally, part of these results have been also confirmed from relevant meta-analysis of (*n*=1865) subjects under exercise interventions (*n*=635) and control participants, where ΔFMD was reported to be increased (ΔFMD 9.2%) in exercise participation of a volume ≥150 min/week compared to those with lower exercise volume <150 min/week (ΔFMD 4.7%) (Early et al., 2017). Under this results from (Pedralli et al., 2020) including their blood pressure decreases (endurance ΔSBP –5.1 mmHg; resistance ΔSBP –4.0 mmHg; concurrent training ΔSBP –3.2 mmHg), our current study report similar results increasing ΔFMD using $CT_{HIIT+RT}$, superior results for decreasing ΔSBP –7.8/–9.5 mmHg in both Rs/NRs but in a minor volume of exercise intervention of 6 weeks.

About PWV, our results show decreases in both $WLNR_{ET}$ (*i.e.*, ΔPWV –0.8 $m \cdot s^{-1}$) and WLR_{ET} groups (ΔPWV –0.9 $m \cdot s^{-1}$) (Figure 2a). From recent studies, (Swift et al., 2023) after 10 weeks of a 'weight loss' program including diet control plus exercise training (endurance, 2-3 sessions/week), the participants of the 'weight maintenance' who developed exercise training of moderate intensity [200-300min/week, in similar protocol as previously (Donnelly et al., 2009)], they lose ≥7% of weight and decrease PWV (*i.e.*, ΔPWV –0.3 $m \cdot s^{-1}$). Considering this literature result, our results of decreasing arterial stiffness (*i.e.*, ΔPWV –0.8 to –0.9 $m \cdot s^{-1}$) are ~3 fold superior than (Swift et al., 2023). After 16 weeks of exercise training intervention, (Guimarães et al., 2010) reported that HIIT exercise was superior to decrease ΔPWV –0.4 $m \cdot s^{-1}$ vs. endurance exercise. Part of our significant PWV results have been alerted in recent studies of literature review from (Bakali et al., 2023), whom reported in (*n*=3729) subjects with/without hypertension that endurance exercise (≥3 weeks of volume interventions) decreased ΔPWV –0.6 $m \cdot s^{-1}$, and that by the present study we added more evidence about a concurrent training intervention using $CT_{HIIT+RT}$.

By contrast, a meta-analyses from (Ashor et al., 2014) have indicated that increases in ΔPWV 1 $m \cdot s^{-1}$ were associated with a 12 to 14 % increase in cardiovascular events and additionally with a 13-15 % increase in mortality due to cardiovascular disease. From here, our present results in ΔPWV decreases in both groups of WLR_{ET} and $WLNR_{ET}$ show to be clinically relevant as health strategy for cardiovascular risk reduction in physically inactive populations with risk factors for cardiovascular disease (i.e., all groups reported overweight/obesity and PWV >8.0 $m \cdot s^{-1}$ [Table 2]).

About clinical magnitude, it is possible to summarize two approaches, firstly that Cohen *d* ES for main outcomes PWV (WLR_{ET}: *d*=0.38, and $WLNR_{ET}$: *d*=0.45) and FMD (WLR_{ET}: *d*=043, and $WLNR_{ET}$: *d*=0.47) that are classified into the 'small' effect size, and secondly, considering also previous exercise literature review studies reporting clinical effects. For example, a current literature revie from (Xi et al., 2025) reported to arterial stiffness improvements after exercise intervention by decreases in ΔPWV –1.6 $m \cdot s^{-1}$ but with interventions ≥12 weeks. Similarly, FMD increases have been recently reported from literature review studies ranged from ΔFMD 0.4 to 5.1% but also after a superior volume of exercise intervention (8 to 24 weeks) (Paravlic & Drole, 2025). Thus, considering our short-term of 6-week $CT_{HIIT+RT}$ intervention our results are of clinical relevance decreasing ΔPWV –0.8 to -0.9 and increasing ΔFMD 6.5% of independent of subjects lose weight or not.

Regarding the proportion of non-responders (NRs) in $WLNR_{ET}$, we observed a prevalence of 42.8% (*n*=6) for reductions in ΔPWV and 28.6% (*n*=4) for increases in ΔFMD. However, there is still a scarcity of studies addressing this area. In our previous reports, we documented similar proportions of NRs for reductions in ΔPWV among individuals with hypertension (10%), elevated blood pressure (20%), and normotension (60%), respectively. (Ramírez-Vélez et al., 2019) reported that, after 12 weeks of endurance training (66% NRs) or HIIT (36% NRs), the prevalence of NRs for ΔFMD was comparable, while the prevalence of NRs for ΔPWV was 77% with endurance training and 45% with HIIT. In contrast to these findings, the prevalence of NRs in our study was lower. We hypothesize that, given our shorter intervention period (6 vs. 12 weeks) and differences in the exercise modality employed, the combined $CT_{HIIT+RT}$ protocol may have promoted vascular adaptations distinct from those elicited by endurance or HIIT training alone.

The improvement of vascular parameters as decreasing arterial stiffness and increase dilation by FMD without major weight loss changes are of high relevance to exercise prescription professionals, from several point of view, that includes; i) a decrease in the total volume (weeks) of exercise prescription

could be minor (6-weeks) with aims of decrease the cardiovascular risk, ii) patients with lower possibilities to adhere to some diet control could also receive benefits from exercise without other nutritional strategies, iii) the application of $CT_{HIIT+RT}$ is of interest in the additional body composition improvements because in long-term the RT exercise promote increases in muscle mass and thus other metabolic benefits such as increase glucose control and functional improvements in population with overweight/obesity that are more in risk of cardiometabolic diseases such as T2DM or metabolic syndrome as previous reports of concurrent training (P. Delgado-Floody et al., 2022). Thus, exercise programs under public or private health should consider short-term $CT_{HIIT+RT}$ with aims of promote a fast decrease in the cardiovascular risk of hypertensive or T2DM patients to avoid major cardiometabolic conditions.

The clinical relevance of improving FMD is well established, as a 1% increase in this parameter has been associated with a reduction in cardiovascular risk and all-cause mortality (You et al., 2022). On the other hand, in the present study, both WLR_{ET} and $WLNR_{ET}$ groups also elicited reductions in SBP of approximately –8 and –9 mmHg, respectively. Notably, SBP values of ≤120 mmHg has been linked to a reduction in major cardiovascular events and all-cause mortality (Bergmann et al., 2025).

A strength of our study includes *i*) the use of a standardized FMD protocol and validated equipment for PWV assessment, *ii*) we used the technical error of measurement to categorize those Rs and NRs to lose weight considering the original study. Part of the limitations include, we do not control strictly the diet of the subjects during the 6 weeks of intervention, however we recommended every week before exercise to all exercise participants to be hydrated (by water) and to consume carbohydrates to avoid symptoms of lack of energy during $CT_{HIIT+RT}$ exercise or dehydration. We check these diet patterns (x 2 times) by the 24ASA online questionnaire https://epi.grants.cancer.gov/asa24/ for macronutrients (protein 89.7 g, fat 81.3 g, carbohydrates 223.5 g, all in average considering both groups), and micronutrients (Folate: 463.3 mcg, Sodium 3473.2 mg, Potassium 2937.3 mg, Calcium 741 mg and Zinc 13.7 mg), all with an average Kcal consumption 1984.8 Kcal/day (data for internal control of the study). Among other approaches, recent literature from (Lobene et al., 2023) have discussed the diet and energy balance, on their impact in the vascular function of healthy subjects, where macronutrients and micronutrient are also considered before exercise to promote better endothelia function in responses to exercise, and future studies should consider the diet factor as potential factor for accelerating the exercise adaptations. Other limitations could include that the NRs were all those who decreased a X kg of body weight, more than recommended percentages in cases of obesity for example.

Conclusions

Adult participants of a six-week exercise training intervention who are nonresponders to body weight loss can also improve arterial stiffness by decreasing PWV and improve the arterial dilation capacity by increasing FMD in similar physiological adaptations than weight loss responders to exercise training. These results are displayed with other relevant results also in favor of weight loss nonresponders such as blood pressure decreases and additional vascular improvements. Overall, these findings could be of high relevance in the exercise prescription professionals and to individuals with overweight/obesity and high blood pressure with aims of improve vascular parameters and avoid major cardiovascular conditions as well as to decrease and control blood pressure. Future studies could add more mechanisms for explaining these results.

Acknowledgements

We like to recognize all those participants who accepted to participate in our research at the Universidad Andres Bello, and that additionally participated of all stages of this randomized controlled clinical study.

Financing

This research was funded by Dirección General de Investigación, Vicerrectoría de Investigación y Doctorado, Concurso de Ciencias Biomédicas y Clínicas, Universidad Andres Bello 2022 Código N ◦ DI-01-CBC/22.

References

Alvarez, C., Jara, C. A. C., Ciolac, E. G., Guimaraes, G. V., Mayorga, O. A., Montoya, J. C., Andrade, D. C., Floody, P. D., Martínez, A. M. A., & Izquierdo, M. (2023). Hypertensive patients show higher heart rate response during incremental exercise and elevated arterial age estimation than normotensive adult peers: VASCU-HEALTH PROJECT. *Retos: nuevas tendencias en educación física, deporte y recreación*(50), 25-32. https://doi.org/10.47197/retos.v50.99716

Alvarez, C., Peñailillo, L., Ibacache-Saavedra, P., Jerez-Mayorga, D., Campos-Jara, C., Andrade, D., Guimarães, G., Gomes-Ciolac, E., Delgado-Floody, P., & Izquierdo, M. (2024). Six weeks of a concurrent training therapy improves endothelial function and arterial stiffness in hypertensive adults with minimum non-responders. *Hipertensión y Riesgo Vascular*. https://doi.org/10.1016/j.hipert.2024.07.001

Ashor, A. W., Lara, J., Siervo, M., Celis-Morales, C., & Mathers, J. C. (2014). Effects of exercise modalities on arterial stiffness and wave reflection: a systematic review and meta-analysis of randomized controlled trials. *PloS one*, *9*(10), e110034. https://doi.org/10.1371/journal.pone.0110034

Atkinson, G. (2014). Shear rate normalization is not essential for removing the dependency of flow-mediated dilation on baseline artery diameter: past research revisited. *Physiological measurement*, *35*(9), 1825. https://doi.org/10.1088/0967-3334/35/9/1825

Bakali, M., Ward, T. C., Daynes, E., Jones, A. V., Hawthorne, G. M., Latimer, L., Divall, P., Graham-Brown, M., McCann, G. P., & Yates, T. (2023). Effect of aerobic exercise training on pulse wave velocity in adults with and without long-term conditions: a systematic review and meta-analysis. *Open Heart*, *10*(2), e002384. https://doi.org/10.1136/openhrt-2023-002384

Bergmann, F., Prager, M., Pracher, L., Sawodny, R., Steiner-Gager, G. M., Richter, B., Jilma, B., Zeitlinger, M., Gelbenegger, G., & Jorda, A. (2025). Systolic blood pressure targets below 120 mm Hg are associated with reduced mortality: A meta-analysis. *Journal of Internal Medicine*, *297*(5), 479-491. https://doi-org.recursosbiblioteca.unab.cl/10.1111/joim.20078

Dekker, M. J., Lee, S., Hudson, R., Kilpatrick, K., Graham, T. E., Ross, R., & Robinson, L. E. (2007). An exercise intervention without weight loss decreases circulating interleukin-6 in lean and obese men with and without type 2 diabetes mellitus. *Metabolism*, *56*(3), 332-338. https://doi.org/10.1016/j.metabol.2006.10.015

Delgado-Floody, P., Chirosa-Rios, L., Caamano-Navarrete, F., Valdes-Badilla, P., Herrera-Valenzuela, T., Monsalves-Alvarez, M., Nunez-Espinosa, C., Castro-Sepulveda, M., Guzman-Munoz, E., Andrade, D. C., & Alvarez, C. (2022). Concurrent training and interindividual response in women with a high number of metabolic syndrome risk factors. *Front Physiol*, *13*, 934038. https://doi.org/10.3389/fphys.2022.934038

Delgado-Floody, P., Chirosa-Ríos, L., Caamaño-Navarrete, F., Valdés-Badilla, P., Herrera-Valenzuela, T., Monsalves-Álvarez, M., Núñez-Espinosa, C., Castro-Sepulveda, M., Guzmán-Muñoz, E., & Andrade, D. C. (2022). Concurrent training and interindividual response in women with a high number of metabolic syndrome risk factors. *Front Physiol*, 1922. https://doi.org/10.3389/fphys.2022.934038

Delgado-Floody, P., Izquierdo, M., Ramírez-Vélez, R., Caamano-Navarrete, F., Moris, R., Jerez-Mayorga, D., Andrade, D. C., & Alvarez, C. (2020). Effect of high-intensity interval training on body composition, cardiorespiratory fitness, blood pressure, and substrate utilization during exercise among prehypertensive and hypertensive patients with excessive adiposity. *Front Physiol*, 1171. https://doi.org/10.3389/fphys.2020.558910

Donnelly, J. E., Blair, S. N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc*, *41*(2), 459-471. https://doi.org/10.1249/MSS.0b013e3181949333

Early, K. S., Stewart, A., Johannsen, N., Lavie, C. J., Thomas, J. R., & Welsch, M. (2017). The effects of exercise training on brachial artery flow-mediated dilation: a meta-analysis. *Journal of Cardiopulmonary Rehabilitation and Prevention*, *37*(2), 77-89. https://doi.org/10.1097/HCR.0000000000000206

Guimarães, G. V., Ciolac, E. G., Carvalho, V. O., D'Avila, V. M., Bortolotto, L. A., & Bocchi, E. A. (2010). Effects of continuous vs. interval exercise training on blood pressure and arterial stiffness in treated hypertension. *Hypertension Research*, *33*(6), 627-632. https://doi.org/10.1038/hr.2010.42

Hopkins, W. G., Marshall, S. W., Batterham, A. M., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Medicine and science in sports and exercise*, *41*(1), 3-13. https://doi.org/10.1249/mss.0b013e31818cb278

Jones, D. W., Ferdinand, K. C., Taler, S. J., Johnson, H. M., Shimbo, D., Abdalla, M., Altieri, M. M., Bansal, N., Bello, N. A., & Bress, A. P. (2025). 2025 AHA/ACC/AANP/AAPA/ABC/ACCP/ACPM/AGS/AMA/ASPC/NMA/PCNA/SGIM guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *JACC*. https://doi.org/10.1016/j.jacc.2025.05.007

Kim, H. M., Rhee, T.-M., & Kim, H.-L. (2022). Integrated approach of brachial-ankle pulse wave velocity and cardiovascular risk scores for predicting the risk of cardiovascular events. *PloS one*, *17*(4), e0267614. https://doi.org/10.1371/journal.pone.0267614

Liu, G., Sha, W., Wu, Y., Luo, J., Cai, Y., Zhang, T., & Yang, Y. (2025). The association between estimated pulse wave velocity and cardio-cerebrovascular disease risk: a cohort study. *European journal of medical research*, *30*(1), 16. https://doi.org/10.1186/s40001-024-02217-4

Lobene, A. J., Ragland, T. J., Lennon, S. L., & Malin, S. K. (2023). Nutrition Interactions With Exercise Training on Endothelial Function. *Exerc Sport Sci Rev*, *51*(2). https://doi.org/10.1249/JES.0000000000000312

Mancia, G., Fagard, R., Narkiewicz, K., Redón, J., Zanchetti, A., Böhm, M., Christiaens, T., Cifkova, R., De Backer, G., Dominiczak, A., Galderisi, M., Grobbee, D. E., Jaarsma, T., Kirchhof, P., Kjeldsen, S. E., Laurent, S., Manolis, A. J., Nilsson, P. M., Ruilope, L. M., . . . Members:, L. o. a. F. (2013). 2013 ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Journal of hypertension*, *31*(7), 1281-1357. https://doi.org/10.1097/01.hjh.0000431740.32696.cc

Mestek, M. L., Westby, C. M., Van Guilder, G. P., Greiner, J. J., Stauffer, B. L., & DeSouza, C. A. (2010). Regular Aerobic Exercise, Without Weight Loss, Improves Endothelium-dependent Vasodilation in Overweight and Obese Adults. *Obesity*, *18*(8), 1667-1669. https://doi.org/10.1038/oby.2009.467

Minsal. (2017). Departamento de Epidemiología. Encuesta Nacional de Salud 2016-2017. http://epi.minsal.cl/encuesta-ens/

Morales, M. S., Cuffaro, P. E., Barochiner, J., Rada, M. A., Alfie, J., Aparicio, L., Marin, M., Galarza, C. R., & Waisman, G. D. (2015). Validation of a new piezo-electronic device for non-invasive measurement of arterial pulse wave velocity according to the artery society guidelines. *Artery Research*, *10*, 32-37. https://doi.org/10.1016/j.artres.2015.03.001

Paravlic, A. H., & Drole, K. (2025). Effects of aerobic training on brachial artery flow-mediated dilation in healthy adults: a meta-analysis of inter-individual response differences in randomized controlled trials. *BMC Sports Sci Med Rehabil*, *17*(1), 72. https://doi.org/10.1186/s13102-025-01124-3

Pedralli, M. L., Marschner, R. A., Kollet, D. P., Neto, S. G., Eibel, B., Tanaka, H., & Lehnen, A. M. (2020). Different exercise training modalities produce similar endothelial function improvements in individuals with prehypertension or hypertension: a randomized clinical trial Exercise, endothelium and blood pressure. *Scientific reports*, *10*(1), 1-9. https://doi.org/10.1038/s41598-020-64365-x

Petermann, F., Durán, E., Labraña, A. M., Martínez, M. A., Leiva, A. M., Garrido-Méndez, A., Poblete-Valderrama, F., Díaz-Martínez, X., Salas, C., & Celis-Morales, C. (2017). Factores de riesgo asociados al desarrollo de hipertensión arterial en Chile. *Revista medica de Chile*, *145*(8), 996-1004. http://dx.doi.org/10.4067/s0034-98872017000800996

Ramírez-Vélez, R., Castro-Astudillo, K., Correa-Bautista, J. E., González-Ruíz, K., Izquierdo, M., García-Hermoso, A., Álvarez, C., Ramírez-Campillo, R., & Correa-Rodríguez, M. (2020). The effect of 12 Weeks of different exercise training modalities or nutritional guidance on cardiometabolic risk factors, vascular parameters, and physical fitness in overweight Adults: cardiometabolic high-intensity interval training-resistance training randomized controlled study. *The Journal of Strength & Conditioning Research*, *34*(8), 2178-2188. https://doi.org/10.1519/JSC.0000000000003533

Ramírez-Vélez, R., Hernández-Quiñones, P. A., Tordecilla-Sanders, A., Álvarez, C., Ramírez-Campillo, R., Izquierdo, M., Correa-Bautista, J. E., Garcia-Hermoso, A., & Garcia, R. G. (2019). Effectiveness of HIIT compared to moderate continuous training in improving vascular parameters in inactive adults. *Lipids in health and disease*, *18*(1), 42. https://doi.org/10.1186/s12944-019-0981-z

Ring, M., Eriksson, M. J., Zierath, J. R., & Caidahl, K. (2014). Arterial stiffness estimation in healthy subjects: a validation of oscillometric (Arteriograph) and tonometric (SphygmoCor) techniques. *Hypertension Research*, *37*(11), 999-1007. https://doi.org/10.1038/hr.2014.115

Schiavon, C. A., Cavalcanti, A. B., Oliveira, J. D., Machado, R. H. V., Santucci, E. V., Santos, R. N., Oliveira, J. S., Damiani, L. P., Junqueira, D., Halpern, H., Monteiro, F. d. L. J., Noujaim, P. M., Cohen, R. V., Sousa, M. G. d., Bortolotto, L. A., Berwanger, O., & Drager, L. F. (2024). Randomized Trial of Effect of Bariatric Surgery on Blood Pressure After 5 Years. *Journal of the American College of Cardiology*, *83*(6), 637-648. https://doi.org/doi:10.1016/j.jacc.2023.11.032

Swift, D. L., McGee, J. E., Grammer, E. E., Huff, A. C., Clunan, M. C., Hursey, N., Brown, T. T., Osborne, B. G., Houmard, J. A., & Carels, R. A. (2023). The effect of exercise training level on arterial stiffness after clinically significant weight loss. *Clinical obesity*, *13*(5), e12584. https://doi-org.recursosbiblioteca.unab.cl/10.1111/cob.12584

Thijssen, D. H., Bruno, R. M., van Mil, A. C., Holder, S. M., Faita, F., Greyling, A., Zock, P. L., Taddei, S., Deanfield, J. E., & Luscher, T. (2019). Expert consensus and evidence-based recommendations for the assessment of flow-mediated dilation in humans. *European Heart Journal*, *40*(30), 2534-2547. https://doi-org.recursosbiblioteca.unab.cl/10.1093/eurheartj/ehz350

Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Himmelfarb, C. D., DePalma, S. M., Gidding, S., Jamerson, K. A., Jones, D. W., MacLaughlin, E. J., Muntner, P., Ovbiagele, B., Smith, S. C., Spencer, C. C., Stafford, R. S., Taler, S. J., Thomas, R. J., Williams, K. A., . . . Wright, J. T. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, *71*(6), 1269-1324. https://doi.org/doi:10.1161/HYP.0000000000000066

WHO. (2000). Obesity: preventing and managing the global epidemic. Report of a WHO Consultation894:i–xii, 891–253. ISBN 92 4 120894 5; ISSN 0512-3054. https://iris.who.int/handle/10665/42330

Whytock, K. L., & Goodpaster, B. H. (2025). Unraveling Skeletal Muscle Insulin Resistance: Molecular Mechanisms and the Restorative Role of Exercise. *Circulation Research*, *137*(2), 184-204. https://doi.org/10.1161/CIRCRESAHA.125.3255

Wyss, F., Coca, A., Lopez-Jaramillo, P., Ponte-Negretti, C., Wyss, F. S., Restrepo, G., Ponte-Negretti, C. I., Lanas, F., Pérez, G., & Barroso, W. S. (2020). Position statement of the Interamerican Society of Cardiology (IASC) on the current guidelines for the prevention, diagnosis and treatment of arterial hypertension 2017–2020. *International Journal of Cardiology Hypertension*, *6*, 100041. https://doi.org/10.1016/j.ijchy.2020.100041

Xi, H., Du, L., Li, G., Zhang, S., Li, X., Lv, Y., Feng, L., & Yu, L. (2025). Effects of exercise on pulse wave velocity in hypertensive and prehypertensive patients: a systematic review and meta-analysis of randomized controlled trials. *Frontiers in Cardiovascular Medicine*, *12*, 1504632. https://doi.org/10.3389/fcvm.2025.1504632

You, Q., Yu, L., Li, G., He, H., & Lv, Y. (2022). Effects of Different Intensities and Durations of Aerobic Exercise on Vascular Endothelial Function in Middle-Aged and Elderly People: A Meta-analysis [Systematic Review]. *Front Physiol*, *Volume 12 - 2021*. https://doi.org/10.3389/fphys.2021.803102

Authors' and translators' details:

Cristian Alvarez	cristian.alvarez@unab.cl	Author
Pedro Delgado-Floody	pedro.delgado@ufrontera.cl	Author
Christian Campos-Jara	christian.campos@unab.cl	Author
Cristóbal Durán-Marín	c.durnmarn@uandresbello.edu	Author
Daniel Jerez-Mayorga	djerezmayorga@ugr.es	Author
David C. Andrade	david.andrade@uantof.cl	Author
Cristian Caparros-Manosalva	ccaparros@utalca.cl	Author
Gabriel Marzuca-Nassr	gabriel.marzuca@ufrontera.cl	Author
Omar Andrade-Mayorga	omar.andrade@ufrontera.cl	Author
Gabriel Rojas-Rojas	gabriel.rojas@unab.cl	Author