scientific reports



OPEN

Concurrent training based on an individual load-velocity ratio assessment as a better alternative to continuous endurance training to improve hypertension

Isabel López-Ruiz¹, Fernando Lozano Ruiz-Poveda², María Dolores Masía³, Juan Ramón Heredia-Elvar⁴ & Noelia González-Gálvez¹⊠

Hypertension remains a major global public health problem as the leading modifiable risk factor for cardiovascular death worldwide, responsible for more than 10 million deaths per year. The aim of the present study was to compare the effect of strength training, based on an individual load-velocity ratio assessment, concurrent with endurance training, and endurance training on blood and metabolic biomarkers, body composition and physical fitness in adults with hypertension. A randomised, single-blind, 12-week, prospective clinical trial was conducted. The study included 75 volunteers with an average age of 54 years, all of whom were hypertensive and sedentary. The sample was randomly assigned into three groups, strength training, based on an individual load-velocity ratio assessment, concurrent with endurance training, endurance training alone group, and a control group. The concurrent training group obtained greater reductions in SBP, DBP and MAP than the endurance training group, up to 12.8 mmHg, 6.8 mmHg and 8.6 mmHg respectively. The findings of the present study indicate that performing strength training, based on an individual load-velocity ratio assessment, concurrent with endurance training 2 days per week for 12 weeks, leads to improvements in metabolic and blood biomarkers, body composition, and physical fitness, of adults with hypertension, with these adaptations being superior to those produced by endurance training alone.

Keywords High blood pressure, Exercise, Combined training, Strength training, Velocity of execution, Endurance

Presently, hypertension remains the main modifiable risk factor worldwide for premature cardiovascular deaths¹. Large epidemiological studies have shown an inverse relationship between the incidence of hypertension and exercise^{2,3}, understood as the set of ordered, structured and systematised movements aimed at improving health or physical condition. The most important clinical guidelines recommend exercise for subjects with hypertension, giving priority to endurance training^{4–6}. The effects of endurance training on hypertension have been studied and are well described in the literature, showing reductions in the range of -4.9 to -12 millimetres of mercury (mmHg) for systolic blood pressure (SBP) and – 3.4 to -5.8 mmHg for diastolic blood pressure (DBP)^{7,8}. Some guidelines recommend incorporating strength training as well⁴, but few individuals comply with their prescription in depth⁸. Strength training has shown improvements ranging from – 3.0 to -4.7mmHg for SBP and – 3.2 to -3.8mmHg for DBP^{7,8}. In addition to these guidelines, the available evidence shows that concurrent training, understood as training that combines strength and endurance work in the same training session, has also shown great benefits on health, specifically on hypertension⁹⁻¹¹. Different controlled trials¹²⁻¹⁴ showed significant reductions in SBP and DBP levels with concurrent programme interventions^{9,15-17}.

However, there are large differences among the protocols applied (frequency, intensity, volume, recovery time and characteristics, selection, organisation and distribution of exercises) in the different studies conducted^{3,9}.

¹Facultad del Deporte, UCAM Universidad Católica de Murcia, Campus de los Jerónimos, 135 Guadalupe, Murcia 30107, Spain. ²Hospital General Universitario de Ciudad Real, Ciudad Real, Spain. ³Hospital Universitario San Juan de Alicante, Alicante, Spain. ⁴Facultad de Ciencias de la Salud, Universidad Alfonso X El Sabio (UAX), Madrid, Spain. [™]email: ngonzalez@ucam.edu

Despite scientific evidence showing improvements in blood pressure levels and other metabolic markers with combined or concurrent training, further research is needed to gain a more specific understanding of the doseresponse relationship of this type of training and to standardize such training programs^{9,18,19}. Recent studies indicate that due to the heterogeneity of research quality and heterogeneity in research protocols, further research is needed to determine the effect of these types of programs. This study is the first to apply a concurrent training program, considering the loss of running speed when prescribing repetitions^{9,20}. One of the main problems is the lack of precision in the quantification of the work stimulus in strength training, which makes it very difficult to establish a dose-response relationship, as it is not known whether the intensity with which the participants trained was the same as that programmed and applied, as well as not knowing the degree of fatigue reached by each participant in each set or training session. A major difficulty in designing strength training programmes is how to accurately quantify and control the workload in order to obtain the greatest health and fitness benefits²¹. In order to address this problem, the focus has been on the velocity of execution^{22,23}. This form of effort programming has brought about a paradigm shift in the control of strength training^{21,23,24}. Despite all of the research on strength training through speed of execution and its positive results as a tool for training programming in the young population and athletes^{24,25}, cancer survivors²⁶, multiple sclerosis and older adults²⁷, there is still a gap in the health field in relation to the design of exercise programmes with this methodology, and especially in subjects with hypertension. Training methods such as velocity-based training should be prioritized over the repetitions until failure method to minimize the risk of overexertion^{28,29}.

Therefore, the aim of the present study was to compare the effect of strength training, based on an individual load-velocity ratio assessment, combined with endurance training, and endurance training alone, on blood and metabolic biomarkers, body composition and physical fitness in adults with hypertension, by actually knowing the exercise dose applied in the training.

Results

Table 1 shows the training monitoring results (HR, %HRmax, and RPE) for each group in each week. Participants showed a HR average of 113.74 ± 11 , a %HR max of 145.14 ± 13.17 , and an RPE of 3.16 ± 0.92 ; there were no significant differences between the intervention groups. Tables 2 and 3 show the pre-post intervention results for EG1, EG2 and CG. The group * time interaction is also presented.

EG1 and EG2 showed significant improvements in SBP (mmHg), DBP (mmHg), mean arterial pressure (MAP) (mmHg), pulse pressure (PP) (mmHg), rate pressure product (RPP), resting heart rate (RHR), lean mass (kg), body fat (%), waist circumference (cm), glucose (mg/dl), right handgrip (kg), left handgrip (kg), upper limb MPV (m/s 50%), %RM UB, lower limb MPV (m/s 60%), and VO2peak (ml/kg/min). These changes were significantly different from the CG changes. Specifically, EG1 showed greater improvements of up to -12.8 mmHg in SBP, -6.8 mmHg in DBP and – 8.6 in MAP over EG2. Improvements in PP, waist circumference, upper limb PWV (m/s 50%), 1RM (LB), lower limb PWV (m/s 60%) and VO2peak (ml/kg/min) were also greater in EG1 than in EG2.

Discussion

The aim of this present study was to compare the effect of concurrent training, based on an individual load-velocity ratio assessment, and endurance training alone on blood and metabolic biomarkers, body composition and fitness of adults with hypertension. One of the main findings of this study was that both groups, concurrent training and endurance training, showed improvements in blood biomarkers, reducing SBP, DBP, MAP, PP, RPP and RHR. However, the concurrent training group obtained greater reductions in SBP, DBP and MAP than the endurance training group, up to -12.8 mmHg, -6.8 mmHg and - 8.6 mmHg respectively. The same dynamics was observed in PP. The results obtained in the present study, on the reduction of blood pressure levels, were significantly greater than those obtained in previous studies with this same training modality, despite the fact that the training frequency was lower. These improvements may be due to different mechanisms such as endothelium-

	EG1			EG2			
	HR average	%HR max	RPE	HR average	%HR max	RPE	
Week 1	118.88 ± 15.88	66.56	3.20 ± 1.41	117.75 ± 13.61	66.75	3.21 ± 1.22	
Week 2	116.32 ± 12.58	65.60	2.88 ± 1.33	118.25 ± 12.02	67.25	3.21 ± 0.98	
Week 3	118.12 ± 11.50	66.84	3.20 ± 1.08	118.21 ± 14.02	67.25	3.33 ± 1.09	
Week 4	115.60 ± 13.24	65.32	2.84 ± 1.18	118.21 ± 13.28	67.21	3.17 ± 1.20	
Week 5	115.92 ± 14.75	65.68	3.00 ± 1.26	118.83 ± 13.35	67.50	3.37 ± 1.06	
Week 6	115.32 ± 14.18	65.20	2.96 ± 0.93	117.21 ± 11.62	66.58	3.12±0.95	
Week 7	115.84 ± 12.72	65.12	2.84 ± 1.03	119.79 ± 13.38	68.13	3.46 ± 1.18	
Week 8	116.80 ± 15.57	65.72	3.16 ± 1.28	119.17 ± 11.37	67.75	3.21 ± 1.18	
Week 9	119.36 ± 13.39	67.16	3.16 ± 1.14	121.33 ± 11.62	68.75	3.38 ± 1.31	
Week 10	117.32 ± 12.56	66.36	3.08 ± 1.08	118.50 ± 10.47	67.29	3.08 ± 0.97	
Week 11	120.00 ± 16.03	67.80	3.52 ± 1.33	122.04 ± 11.72	69.42	3.54 ± 1.22	
Week 12	113.96 ± 13.25	64.56	2.84 ± 0.94	117.79 ± 10.89	67.00	3.21 ± 1.02	

Table 1. Training monitoring results.

							Group*time interaction		
Outcome	Group	Pre-test (M ± ED)	Post-test (M ± ED)	Difference post-pre (M±ED)	p	95% CI (Mpost-Mpre)	F	Sig	ES
	EG1	142.441 ± 7.53	121.76 ± 8.52	- 21.68 ± 1.43**##	< 0.001	- 24.52; - 18.84			
SBP (mmHg)	EG2	144.12 ± 8.81	135.24 ± 8.98	- 8.88 ± 1.43**	< 0.001	- 11.72; - 6.04	52.00	< 0.001	0.59
	CG	147.92 ± 11.84	146.56 ± 11.53	- 1.36 ± 1.43	0.343	- 4.20; 1.48			
	EG1	88.28 ± 5.73	74.36 ± 5.10	- 13.92 ± 1.08**##	< 0.001	- 16.067; - 11.773	26.92	< 0.001	0.43
DBP (mmHg)	EG2	89.68 ± 6.95	82.56 ± 6.26	- 7.12 ± 1.08*	< 0.001	- 9.267; - 4.973			
	CG	92.16 ± 7.28	89.32±6.95	- 2.84 ± 1.08	0.01	- 4.987; - 0.693			
	EG1	106.52 ± 5.28	90.12±5.34	- 16.40 ± 1.00**##	< 0.001	- 18.393; - 14.407	50.50	< 0.001	
MAP (mmHg)	EG2	107.84 ± 6.06	100.12 ± 6.37	- 7.72 ± 1.00**	< 0.001	- 9.713; - 5.727			0.58
	CG	110.60 ± 7.25	108.28 ± 7.80	- 2.32 ± 1.00	0.023	- 4.313; - 0.327			
	EG1	55.16 ± 7.49	47.40 ± 7.68	- 7.76 ± 1.45**#	< 0.001	- 10.642; - 4.878	10.43	< 0.001	0.23
PP (mmHg)	EG2	54.44 ± 10.08	52.68 ± 7.17	- 1.76 ± 1.45	0.227	- 4.642; 1.122			
	CG	55.76 ± 11.32	57.20 ± 8.52	1.44 ± 1.45	0.323	- 1.442; 4.322			
	EG1	11254.00 ± 1735.96	8507.64 ± 1309.88	- 2746.36 ± 251.20**##	< 0.001	- 3247.117; - 2245.603	30.94	< 0.001	0.46
RPP	EG2	11321.60 ± 1442.08	9957.36 ± 1358.10	- 1364.24 ± 251.20**	< 0.001	- 1864.997; - 863.483			
	CG	11827.56 ± 1474.97	11875.68 ± 1132.23	48.12 ± 251.20	0.849	- 452.637; 548.877			
	EG1	78.40 ± 11.06	69.72 ± 8.49	- 8.68 ± 1.68**	< 0.001	- 12.032; - 5.328	8.99	< 0.001	
RHR	EG2	78.80 ± 11.03	73.72±9.35	- 5.08 ± 1.68*	0.003	- 8.432; - 1728			0.20
	CG	80.00 ± 8.23	81.28 ± 8.07	1.28 ± 1.68	0.449	- 2.072; 4.632			
	EG1	27.43 ± 4.88	27.26 ± 4.57	- 0.18 ± 0.16	0.275	- 0.495; 0.143	0.27		
BMI (kg/m²)	EG2	32.75 ± 5.09	32.56 ± 5.06	- 0.19 ± 0.16	0.234	- 0.511; 0.127		0.76	0.01
	CG	31.91 ± 5.00	31.87 ± 5.10	- 0.04 ± 0.16	0.803	- 0.359; 0.279			
	EG1	28.99 ± 6.74	29.57 ± 6.77	0.58 ± 0.15**	< 0.001	0.279; 0.881	7.55	< 0.001	0.17
LBM (Kg)	EG2	30.84 ± 6.80	31.35 ± 6.81	0.51 ± 0.15**	< 0.001	0.207; 0.809			
	CG	30.56 ± 5.98	30.39 ± 5.92	- 0.17 ± 0.15	0.258	- 0.473; 0.129			
	EG1	32.63 ± 10.27	31.56±9.76	- 1.07 ± 0.34*	0.002	- 1.739; - 0.405	4.41	0.02	0.11
BFP (%)	EG2	39.43 ± 8.04	38.47 ± 8.14	- 0.96 ± 0.34*	0.005	- 1.631; - 0.297			
	CG	37.70 ± 8.45	37.89 ± 8.70	0.20 ± 0.34	0.56	- 0.471;0.863			
	EG1	125.19 ± 58.84	119.75 ± 54.33	- 5.44 ± 2.13*	0.013	- 9.68; - 1.208	2.78	0.07	0.07
VF (cm ²)	EG2	183.40 ± 58.34	178.28 ± 58.00	- 5.12 ± 2.13*	0.018	- 9.36; - 0.888			
	CG	165.26 ± 54.59	166.10 ± 55.82	0.85 ± 2.13	0.691	- 3.388; 5.084]		
	EG1	98.76 ± 10.54	92.16 ± 10.59	- 6.60 ± 0.84**#	< 0.001	- 8.265; - 4.935	19.25		
Waist circumference (cm)	EG2	110.48 ± 13.13	107.28 ± 15.12	- 3.20 ± 0.84**	< 0.001	- 4.865; - 1.535		< 0.001	0.35
	CG	105.80 ± 12.33	106.52 ± 12.65	0.72 ± 0.84	0.391	- 0.945; 2.385			

Table 2. Effect of combined training and endurance training on blood biomarker and body composition. SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Pressure; PP: Pulse Pressure: RPP: Rate Pressure Product; RHR: Resting Heart Rate; BMI: Body Mass Index; LBM: Lean Body Mass; BFP: Body Fat Percentage; VF: Visceral Fat. *Significantly different from CG value at the p < 0.05 level. **Significantly different from other EG p < 0.05 level; **Significantly different from other EG value at the p < 0.01 level.

dependent vascular function and structure and sympathetic nervous system regulation, which are altered and may contribute to blood pressure elevation^{7,30,31}. Endothelial dysfunction is a determinant factor in vascular function, and it is largely dependent on the bioavailability of nitric oxide (NO), which induces vessel relaxation and regulates blood pressure^{32,33}. Strength training may act as a regulator of NO bioavailability by increasing vascular AMPK/PPAR δ and suppressing endoplasmic reticulum stress, thereby improving and maintaining endothelial function^{34,35}. These results may be in line with previous studies that showed how strength training significantly improved the endothelial function in different population groups^{36,37}. In addition, this training modality may also improve vascular structure by increasing artery diameter and reducing vascular intimamedia wall thickness³⁸. On the other hand, in patients with hypertension, it is common to find an overactivation of the sympathetic nervous system and a reduction in parasympathetic control^{30,39}. This physiological imbalance can also be normalised and regularised by strength training as previous research has shown^{40,41}.

In terms of body composition variables, lean mass, body fat, and waist circumference, both groups showed significant improvements as compared to the CG. Physical exercise induces changes in body composition through skeletal muscles, which are able to release hundreds of myokines into the bloodstream during muscle contraction that are able to interact with the muscle itself and with the rest of the body's organs, resulting in great health benefits 42,43. These benefits include fat loss and increased muscle mass. The secretion of some of these

		Pre-test (M ± ED)	Post-test (M ± ED)			95% CI (Mpost-Mpre)	Group*time interaction		
Outcome	Group			Difference post-pre (M ± ED)	p		F	Sig	ES
	EG1	199.72 ± 30.11	192.08 ± 29.21	- 7.64 ± 5.52	0.17	- 18.636; 3.356	6.08		0.15
CT (mg/dl)	EG2	190.12 ± 41.09	183.12 ± 24.57	-7.00 ± 5.52	0.209	- 17.996; 3.996		< 0.001	
	CG	191.76 ± 28.51	208.00 ± 29.89	16.24±5.52	0.004	5.244; 27.236			
	EG1	54.44 ± 9.93	55.32 ± 11.29	0.88 ± 1.07	0.413	- 1.25; 3.01	0.85	0.43	0.02
HDL (mg/dl)	EG2	48.04 ± 7.06	49.72 ± 10.25	1.68 ± 1.07	0.12	- 0.45; 3.81			
	CG	48.76±11.10	48.48 ± 12.49	- 0.28 ± 1.07	0.794	- 2.41; 1.85			
	EG1	120.62 ± 24.78	114.14±24.55	- 6.47 ± 5.01	0.201	- 16.461; 3.517	6.34	< 0.001	0.15
LDL (mg/dl)	EG2	116.32 ± 36.92	108.22 ± 23.88	- 8.10 ± 5.01	0.111	- 18.085; 1.893			
	CG	116.82 ± 23.25	131.34±31.29	14.52±5.01	0.005	4.531; 24.505			
	EG1	122.32 ± 59.16	113.72 ± 43.21	- 8.60 ± 8.08	0.291	- 24.726; 7.526			
TG (mg/dl)	EG2	126.16 ± 51.06	124.88 ± 39.08	- 1.280 ± 8.089	0.875	- 17.406; 14.846	2.134	0.126	0.06
-	CG	133.44 ± 64.33	147.96 ± 79.97	14.520 ± 8.089	0.077	- 1.606; 30.646			
	EG1	98.16 ± 14.69	91.36 ± 10.77	- 6.800 ± 1.366**	< 0.001	- 9.524; - 4.076	15.99	< 0.001	0.31
Glucose (mg/dl)	EG2	106.84 ± 13.44	99.84 ± 13.77	- 7.000 ± 1.366**	< 0.001	- 9.724; - 4.276			
	CG	97.24 ± 22.48	99.80 ± 19.94	2.560 ± 1.366	0.65	- 0.164; 5.284			
	EG1	32.36 ± 10.20	34.52 ± 11.45	2.16±0.59**	< 0.001	0.983; 3.337	4.50	0.01	0.11
Handgrip RA (Kg)	EG2	34.40 ± 8.36	36.80 ± 10.30	2.40±0.59**	< 0.001	1.223; 3.577			
0 1 0	CG	32.64 ± 9.63	32.76±9.76	0.12±0.59	0.84	- 1.057; 1.297			
	EG1	30.00 ± 9.61	31.96±11.10	1.96±0.59**	0.001	0.787; 3.133	6.43	< 0.001	
Handgrip LA (Kg)	EG2	32.44 ± 8.06	35.56±11.04	3.12±0.59**	< 0.001	1.947; 4.293			0.15
	CG	30.20±8.18	30.36 ± 8.22	0.16±0.59	0.786	- 1.013; 1.333			
	EG1	46.24±23.68	65.12±30.34	- 23.12 ± 2.31**##	< 0.001	- 27.715; - 18.525	0.81	0.45	0.02
1RM (UB)	EG2	52.08 ± 21.08	61.16±25.30	- 26.04 ± 2.31**	< 0.001	- 30.635; - 21.445			
, ,	CG	54.24 ± 24.27	52.20 ± 23.20	- 27.12 ± 2.31	< 0.001	- 31.715; - 22.525			
	EG1	0.96 ± 0.03	1.20 ± 0.06	0.237 ± 0.013**##	< 0.001	0.211; 0.263			
Upper limb MPV (m/s 50%)	EG2	0.97 ± 0.02	1.08 ± 0.07	0.112±0.013**	< 0.001	0.086; 0.139	0.26	<0.001	0.77
	CG	0.97 ± 0.02	0.92 ± 0.04	- 0.050**±0.013	< 0.001	- 0.077; - 0.024			
	EG1	73.08 ± 35.12	98.32±41.42	- 29.23 ± 2.82**#	< 0.001	- 34.856; - 23.608			
1RM (LB)	EG2	74.48 ± 38.23	91.80 ± 42.49	- 29.79 ± 2.82**	< 0.001	- 35.416; - 24.168			
. ,	CG	79.92 ± 32.18	76.44±30.73	- 31.97 ± 2.82	< 0.001	- 37.592; - 26.344			
	EG1	0.99±0.02	1.21 ± 0.05	0.225 ± 0.014**#	< 0.001	0.197; 0.253	98.01	< 0.001	0.73
Lower limb MPV (m/s 60%)	EG2	0.99±0.02	1.15±0.07	0.161 ± 0.014**	< 0.001	0.133; 0.189			
Lower mile iii (m, o oo /o)	CG	0.99±0.02	0.95 ± 0.05	-0.039 ± 0.014	0.006	- 0.067; - 0.011			
	EG1	25.09 ± 3.82	30.87 ± 4.41	5.78 ± 0.34**#	< 0.001	5.108; 6.452	87.68	+	0.71
VO2p (ml/kg/min)	EG2	22.18±4.33	26.78±5.29	4.60 ± 0.34**	< 0.001	3.929; 5.274		< 0.001	
2p (, ng,)	CG	21.62±3.58	21.44±3.58	- 0.18 ± 0.34	0.586	- 0.857; 0.488	107.00	.0.001	
	EG1	127±30.55	167 ± 34.40	40±2.36**	< 0.001	35.29; 44.71			
WATT	EG2	125±35.36	162 ± 40.26	37 ± 2.36**	< 0.001	32.29; 41.71	98.33	< 0.001	0.73
	CG	117±32.05	115±29.76	-2±2,36	0.4	- 6.71; 2.71	- 70.33		0.73
	CG	11/ ±34.03	113 ± 47./0	- 4 ± 4,30	0.4	- 0./1; 2./1			

Table 3. Effect of combined training and endurance training on metabolic biomarkers, and physical fitness. CT: Total Cholesterol; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein; TG: Triglycerides; RA: right arm; LA: left arm; 1RM: Maximum Repetition; UL: Upper Limb; LL: Lower Limb; MPV: mean propulsive velocity; VO2p: Peak oxygen consumption. *Significantly different from CG value at the p < 0.05 level. **Significantly different from OCG value at the p < 0.05 level. #Significantly different from other EG value at the p < 0.01 level.

myokines promotes lipolysis and fatty acid oxidation, and induces the darkening of white adipose tissue^{43,44}. It is important to note that the participants who performed the concurrent training obtained greater reductions in abdominal perimeter than the participants who performed the endurance training. These results may be due to the fact that strength training favours visceral fat loss by promoting metabolic adaptations in the abdominal subcutaneous adipose tissue, with a significant increase in lipolysis in this area both during and after the training ^{45,46}.

Glucose values in both experimental groups were significantly reduced after the intervention versus the CG. This improvement could be due to the fact that exercise is able to improve insulin sensitivity starting from the first training session, as well as overall glucose homeostasis⁴⁷. Exercise can increase glucose-6-phosphate levels

accompanying the increase in GLUT4, hexokinase, and glycogen synthase activity, leading to an increase in glucose tolerance and a decrease in blood glucose levels^{34,47,48}.

With respect to the physical condition variables, the participants in both experimental groups showed improvements in both their strength and cardiorespiratory capacity. With regard to strength gains, these were due to the fact that training, regardless of the modality, is able to provoke structural (peripheral) and neural (central) physiological adaptations, given that in all the exercises, muscle action is required to overcome resistance^{21,22}. It is important to note that the concurrent training group obtained greater improvements in all the tests than the endurance training group. These results may be due to the specificity of the strength training, which provoked greater adaptations of the central nervous system thanks to a greater recruitment of motor units, their better synchronisation, and the frequency of the stimulus, as already shown in much of the literature^{49–52}.

Finally, the cardiorespiratory capacity also improved significantly after completion of the exercise programmes in both groups. Exercise promotes adaptations in skeletal muscle such as increased mitochondrial biogenesis and capillary density, thereby improving the ability to transport and use oxygen to generate energy^{53–55}. Numerous studies have attributed a key role to peroxisome proliferator-activated receptor gamma coactivator 1a (PGC-1a) in these cardiovascular exercise-induced muscle adaptations^{53,55-57}. Although there is a controversy in the literature that concurrent training may interfere with the adaptations induced by the different metabolic pathways, AMPK was activated by endurance training and m-TOR was activated by strength training. However, in this present study, it can be observed that the participants who performed the combined training showed greater improvements in VO₂peak than the participants who performed the endurance training. These results of concurrent training could be justified by the large adaptive reserve of sedentary subjects to any training programme. Previous work has shown that when a previously sedentary subject begins a concurrent training programme, the response to the two exercise modalities is positive and additive, promoting a generic adaptation in the absence of a true specificity of the training effect⁵⁸. Another important factor to consider is the low degree of effort involved in strength training, which would explain why the possible interference would also be low, resulting in greater responses with respect to the maintenance of MHC IIX fibres⁵⁹, which could be translated into an improved ability to transport and use oxygen, an increased ability to apply force and a significant improvement in physical fitness.

This study has some limitations that should be taken into account for future research, such as the total intervention time, possibly with longer periods of time more significant results could have been obtained in some of the variables. Another point to consider is the lack of a strength group in which the intensity and volume were not quantified through the speed of execution, but in the traditional way, through the percentage of 1RM and a determined number of repetitions. This would have allowed us to know the effects of the different training programs on blood pressure levels and to continue advancing in the improvement of cardiovascular health. Finally, another limitation was the daily intake of the participants, the diet could have been controlled by making a daily dietary record. Despite the limitations, this would be the first study conducted in an adult population with hypertension, in which the intensity and volume of strength training had been programmed through the velocity of execution, which allowed individualising the work stimulus for each participant with the greatest possible precision, something unknown to date in this population group. In addition, this quantification of the training load allowed the degree of fatigue reached by the participants to be similar throughout the intervention period. This study will provide new insights into the scientific community's multicomponent impact on adults with hypertension. This is the first study to apply a strength-speed training protocol based on training in this population. It also compares it with aerobic training. Controlling this variable is vital for injury prevention^{28,29}, and the present study shows improvements in hemodynamic parameters, demonstrating reduced fatigue. This improves training control, enhances safety, and reduces the risk of injury.

The findings of the present study indicate that following a strength training programme, based on an individual load-velocity ratio assessment, combined with endurance training at a frequency of 2 days per week for 12 weeks, produces improvements in metabolic and blood biomarkers, body composition, and physical fitness of adults with hypertension, with these adaptations being superior to those produced by endurance training alone, even though the latter showed improvements, but to a lesser extent.

Methods Study design

A randomised control trial was conducted. A simple randomization method with the software Microsoft Excel (version 2016) was used to distribute subjects into the Experimental Group 1 (EG1) (n=25), Experimental Group 2 (EG2) (n=25), and Control Group (CG) (n=25). Based on blood pressure, a randomised sequence was generated. The group assignment was blinded to the examiner and staff who performed the statistical analysis. The trial design was recorded (22/06/2023) in ClinicalTrial.gov (Code: NCT05914870) and followed the Consolidated Standards of Reporting Trials (CONSORT) guidelines and the Template for Intervention Description and Replication (TIDIER) checklist.

This randomized control trial obtained the approval by the Research Ethics Committee of the Universidad Católica de Murcia (CE062107), in accordance with the Declaration of Helsinki. The Informed Consent document was signed by all participants prior to the start of the study.

Participants

The study included 75 volunteers, 36 women and 39 men (54.45 ± 6.72 years). Figure 1 shows a flowchart representing the Consolidated Standards of Reporting Trials.

The inclusion criteria were: (a) adults with diagnosed hypertension and with follow-up by their primary care physician; (b) being within the age range of 40–65 years old and (c) not having exercised during the last year. The exclusion criteria were: (a) subjects with osteoarticular or musculoskeletal problems that could interfere with

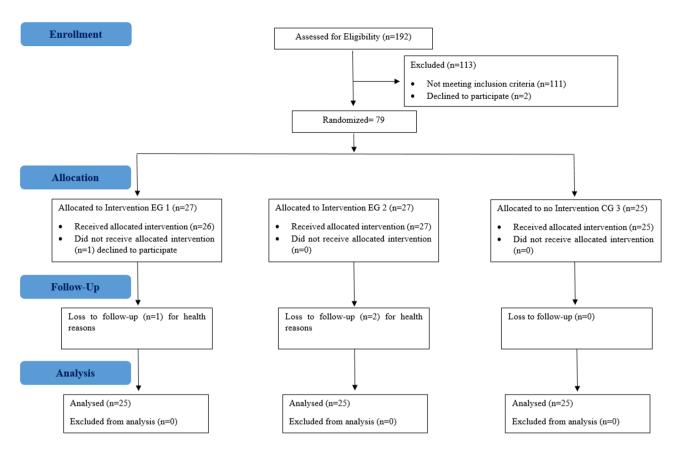


Fig. 1. Consolidated standards of reporting trials flow diagram.

the performance of daily activities; (b) acute or chronic disease that could compromise the subject's autonomy and functional independence; or (c) neurological problems. The participants were instructed not to modify their lifestyle: eating habits and physical activity. The sample size and power calculations were performed using Rstudio 3.15.0 software. The significance level was set at $\alpha=0.05$ and the power to 95% (1- $\beta=0.95$). The standard deviation was used according to the standard deviation for blood pressure from previous studies 60 . A total of 10 mmHg in SBP and 5 mmHg in DBP were set as a minimum clinically significant change 61 . Considering a dropout rate of up to $9.57\%^{62}$, 27 participants in each group needed to be enrolled. The final sample provided a power of 95% if found between and within a variance of 4.9 mmHg of SBP and 3.39 mmHg in DBP.

Assessment

Two researches conducted all pre-post intervention measurements under the same conditions. Two weeks before the initial evaluations, all participants were informed of the prerequisites for each of the tests.

Two assessment sessions were necessary, 48–72 h apart from one another. The assessments were carried out in a laboratory with a standardised temperature of 24 °C. The measurements were taken in the morning, between 8:00 and 13:00 h from Monday to Saturday. Height was measured with a SECA 217 stadiometer (SECA, Germany). The Body Composition Analysis was performed with the Body Composition Analyser InBody 770 through the measurement method DSM-BIA (Bioelectric Impedance Multifrequency and Direct Segmental)^{63,64}. Prior to the assessment, the participant was informed that they should come to the appointment fasting or at least 3 h after the last meal; 30 min before the assessment, they should not have ingested any liquids; they should not have been to the toilet; they should not have drunk alcohol 48 h before; and they should not wear any metal elements in contact with the skin (rings, bracelets or watches). For the measurement the participant completely cleaned his hands and feet completely with the InBody tissue and stood in front of the equipment; they climbed onto the InBody stepping barefoot on the foot plates and holding the hand electrodes, with arms outstretched without touching the sides of the body and without being allowed to speak or move until the end of the test.

Waist circumference was measured according to the ISAK protocol⁶⁵ with a non-elastic tape measure (SECA 200; SECA). The participant stood with their feet together, arms crossed over the chest and relaxed abdomen; the measurement was taken at the narrowest point of the waist at the end of a normal exhalation.

Haemodynamic parameters were assessed using the BPBIO-750 sphygmomanometer, clinically validated according to the ESH protocol⁶⁶. The measurement was performed on the left arm, supported and relaxed on the blood pressure monitor, with the cuff at the level of the heart. Three measurements were taken within 3 min of each other. The mean values of the measurements were taken as a reference⁶⁷.

Blood samples were taken by puncturing the cubital vein. The samples were stored in sterile blood tubes in refrigerated conditions (4–8° degrees Celsius (C)) for no longer than 4 h and then sent to testing according

to standardized procedures. Blood glucose was measured using an ultraviolet assay with hexokinase; total cholesterol (TC), high density lipoprotein (HD), and low-density lipoprotein (LDL) were measured using a homogeneous enzymatic colorimetric test, and triglycerides (TG) were measured using enzymatic assay (Cobas 8000 C702, Roche, Mannheim, Germany).

The manual grip force was measured with the Baseline manual hydraulic dynamometer. For the measurement, the participants had to remain seated on the chair with their back against it, feet on the floor, right elbow flexed at 90°, and the wrist in a neutral position. Then, they had to grip the dynamometer with their thumb upwards; once in the correct position, the subject squeezed the grip as tightly as possible for 3 s. The highest peak strength (kilograms (kg)) recorded between the two attempts was considered for analysis 68.

The assessment of lower and upper body strength was carried out through the velocity of execution in the concentric action by means of the Velowin optoelectronic encoder (Velowin v.1.7.232, Instruments and Sports Technology; Murcia, Spain)^{24,25}. In addition, the velocity of execution allowed for high precision programming of training intensity and volume. Two tests per body region were carried out, one of progressive loads, to estime 1RM and determine the training load, and the other of loss of velocity in the set (VL%), to establish the number of repetitions, on a Multipower BH TR machine, and the exercises performed were the squat and the bench press^{24,25,69}. For reasons of standardization and safety, the concentric phase of each exercise was performed at the maximum possible velocity and the eccentric phase at a slow and controlled velocity, between 3 and 4 s in duration. The squat and bench press tests were performed in different sessions and their order was randomised. All participants performed 2–3 pre-test repetitions in the same evaluation session, they were instructed to move the barbell at the maximum possible speed in the concentric phase and in a slow and controlled manner in the eccentric phase.

For the progressive load test with the squat exercise, the participant stood with knees and hips extended, legs approximately shoulder-width apart and the Multipower bar resting on the upper back. The subject performed the parallel squat downwards in a continuous motion until reaching 80-90° of knee flexion, touching the reference bench, and immediately climbed back up to the starting position at the maximum possible velocity keeping the feet in contact with the ground (without jumping), although the heels could be lifted slightly^{70,71}. The initial external load for the squat exercise was set at 15 kg for women and progressively increased individually with 5 kg, 2.5 kg and 1.25 kg discs. For men, it was set at 25 kg and progressively increased individually with 10 kg, 5 kg and 2.5 kg discs. Both, males and females executed the exercise until reaching a mean propulsive velocity (MPV) between 0.76 – 0.68 m·s⁻¹, which corresponds to 75%-80% 1 repetition maximum (1RM) in this exercise. In the test, 5 sets were performed, in the first set the participant performed 3 reps with a very light load which allowed them to move the bar at a lower velocity of 1.28 m·s⁻¹ (less than 40% 1RM); in the second set, they performed 3 reps with a light load that allowed them to move the bar between 1.28 and 1.21 m·s⁻¹ (40%-45% 1RM); In the third set, he performed 3 reps with a medium load that allowed him to move the barbell between 1.14 and 1.00 m·s⁻¹ (50%-60% 1RM); In the fourth set, they performed 2 reps with a medium-high load that allowed them to move the barbell between 0.92 -0.84 m·s⁻¹ (65%-70% 1RM); and in the fifth set he performed 1 rep with a high load that allowed them to shift the bar between 0.76 and 0.68 m·s⁻¹ (75%-80% 1RM). The velocity loss (VL) test was performed 24 h after the progressive load test. For this test, a medium workload was defined, the participant was allowed to move the barbell to $0.99 \pm 0.02 \text{ m} \cdot \text{s}^{-1}$, assuming 60% of 1RM, and a low VL magnitude of 10% was set for the squat exercise. The test ended when the subject reached the prescribed VL% limit, regardless of the number of repetitions performed in the set^{70,72,73}. For the progressive load test with the bench press exercise, the participant was positioned lying on a horizontal bench with feet resting on the back bar of the Multipower to avoid lumbar arching; hands were placed on the non-slip area of the bar 5-7 cm wider than shoulder width apart. The position of the bench was adjusted so that the bar was aligned with the chest line of each participant. During execution, the eccentric phase of each repetition was performed with controlled speed until the bar was 1-2 cm above the chest, they stopped for 1s, to minimise the contribution of the stretchshortening cycle, and then pushed the bar at maximum velocity. The initial external load for bench press exercise was set at 10 kg for women and progressively increased individually with 5 kg, 2.5 kg and 1.25 kg discs. For men, it was set at 20 kg and progressively increased individually with 10 kg, 5 kg and 2.5 kg discs. Both, males and females executed the exercise until reaching an MPV between 0.55 and 0.47 m·s⁻¹, which corresponds to 75%-80% 1RM in this exercise. For the test, 5 sets were performed, in the first set the participant performed 3 reps with a very light load which allowed them to move the bar at a lower speed of 1.13 m·s⁻¹ (less than 40% 1RM); in the second set, they performed 3 reps with a light load that allowed them to move the bar between 1.13 and 1.04 m·s⁻¹ (40%-45% 1RM); In the third set, they performed 3 reps with a medium load that allowed him to move the barbell between $0.95 - 0.87 \text{ m} \cdot \text{s}^{-1}$ (50%-60% 1RM); In the fourth set, they performed 2 reps with a mediumhigh load that allowed them to move the barbell between 0.70 -0.62 m·s⁻¹ (65%-70% 1RM); and in the fifth set they performed 1 rep with a high load that allowed him to shift the bar between 0.55 and 0.47 m·s⁻¹ (75%-80% 1RM). The velocity loss test was performed 24 h after the progressive load test. For this test, a medium workload was defined, which allowed the participant to move the barbell to $0.97 \pm 0.03 \text{ m} \cdot \text{s}^{-1}$, assuming 50% 1RM, and a low velocity loss magnitude of 15% was set. The test ended when the subject reached the prescribed VL% limit, regardless of the number of repetitions performed in the set^{24,72,74}.

Cardiorespiratory capacity was assessed using a maximal effort test on the IC7 high-precision cycloergometer with a direct WattRate power meter and an exact magnetic resistance of 300 degrees. The steps lasted 2 min and the pedalling cadence was 70-80ppm. In the first 2 min, the subject pedalled at 50w, after which the load was increased by 25w every 2 min; the test ended when the participants voluntarily decided to stop due to fatigue that prevented them from continuing with the test⁷⁵. The test lasted between 10 and 15 min. The maximum working power was determined as the last 2 min step that the subject was able to complete without interruption, and the ACSM formula was used for the estimation of VO_2max^{76} . The following formula was used: $VO_2max = (1.8 * workload (kg.m.min-1))/body weight (kg) + 3.5 + 3.5 (where one watt = 6.12 kg.m.min-1). For intensity control$

Set	Load	Repetitions	1RM	Squat exercise speed	Bench press speed
1	Very light	3	< 40%	<1.28 m·s ⁻¹	<1.13 m·s ⁻¹
2	Light	3	40%-45%	1.28-1.21 m·s ⁻	1.13−1.04 m·s ⁻¹
3	Medium	3	50%-60%	1.14-1.00 m·s ⁻¹	0.95 −0.87 m·s ⁻¹
4	Medium-high	2	65%-70%	0.92 -0.84 m·s ⁻¹	0.70 −0.62 m·s ⁻¹
5	High	1	75%-80%	0.76-0.68 m·s ⁻¹	0.55-0.47 m·s ⁻¹

Table 4. Description of the 5-set protocol.

the heart rate (HR) was monitored at all times using the MyZone MZ-3 Heart Rate Monitor⁷⁷. To address the potential limitations of this method of HR monitoring, we simultaneously recorded the subjective perception of exertion (RPE)^{78,79} every 10 min during the session, and monitored and recorded the training in watts at all times with the WattRate direct measurement potentiometer⁸⁰ via a computer installed in the cycloergometer, helping participants to achieve the prescribed work intensity for each training protocol. In addition, blood pressure and oxygen saturation were monitored during the stress test.

Procedure

The intervention period lasted 12 weeks, 2 days/week, with the sessions lasting 60 min each.

EG1 performed strength training, based on an individual load-velocity ratio assessment, combined with endurance training. The first phase was a warmup of 5 min of Nordic walking on an elliptical bike, joint mobility exercises, and dynamic stretching; and 2 sets of squat and bench press with a light weight. The main phase consisted of 20 min of strength work with a selection of 2 exercises: squat (60% 1RM) and bench press (50% 1RM). For both exercises, 3 sets were performed, with a 3 min rest between sets and the number of repetitions individualised for each participant by the initial speed loss test; the average number of repetitions performed for the squat exercise was 8 repetitions and for the bench press exercise 7 repetitions (Table 4).

The initial absolute loads corresponding to 50% of 1RM in bench press and 60% of 1RM in squat were not modified throughout the intervention period, nor were the number of repetitions set for each participant. Regarding the selection of volume for each of the exercises used, and following a minimum stimulus criterion, we considered the scientific literature regarding the control of this variable by means of the loss of velocity in the sets (VL%), as a more precise way of establishing the degree of effort^{24,72,81}. In this way, it was determined that the same loss of velocity in the sets allows for a similar degree of effort or fatigue to be reached between subjects who performed a training protocol with the same relative intensity, although they performed different numbers of repetitions^{69,74}. The analysis of the studies already carried out allowed establishing a VL of 25–30% in squat and 35-40% in bench press, as moderate-high degrees of effort beyond which the degree of fatigue begins to have important repercussions at a metabolic and mechanical level, causing a significant reduction in physical capacity and performance^{24,69,74}. In strength training, speed losses in the series of less than 20-30% in exercises such as squats or bench presses are associated with better physical fitness results. A speed loss in the set between 10 and 20% is sufficient to achieve significant physical condotion benefits, while losses greater than 30% are closer to muscle failure, which may not be optimal for a health improvement goal. To guarantee the use of a low training volume, for the intensity rates proposed in the present study, VL% was set at 10% for the squat exercise and 15% for the bench press in order to ensure improvements in physical condition and reduce potencial risks, given that the study population is sedentary.

In the last 20 min of the main phase, the participants performed continuous endurance training on a cycloergometer. The training intensity was programmed using the Heart Rate Reserve (HRR) using Karvonen's formula (HRR = HRmax - HRrest); once the HRR was known, the prescribed intensity percentage was calculated (% HR= (HRR \times % intensity) + HRrest)⁸². As this was a sedentary population, and the exercise tolerance of each subject was unknown, a moderate intensity range was prescribed for the entire intervention, between 55% and 70% of their heart rate reserve (HRR), in order to achieve the metabolic adaptations already described in the scientific literature⁸³. Finally, the post-training phase included 10 min of cool down and static stretching.

As this was a sedentary population, and the exercise tolerance of each subject was unknown, a moderate intensity range was prescribed for the entire intervention, between 55% and 70% of their heart rate reserve (HRR), in order to achieve the metabolic adaptations already described in the scientific literature.

EG2 performed continuous endurance training. The warm-up was the same as EG1 except for the squat and bench press sets. In the main phase, they performed 40 min of continuous endurance training on a cycloergometer, taking into account the same protocol as in EG1. The post-workout phase was the same as EG1.

Statistical analysis

The Kolmogorov–Smirnov test and Mauchly's W-test were used to evaluate the normality and the sphericity of the data. The mean and standard deviation were calculated from the quantitative variables, and frequency and percentage were used for the qualitative variables. A two-way ANOVA analysis of variance with repeated measurements and Bonferroni's correction were used to compare the changes from the baseline between groups, evaluation time interaction, and evaluation time. To protect against a Type I error, Bonferroni's correction was used to achieve a p = 0.005 for statistical significance. The effect size was calculated using partial eta-squared (η 2p) for analysis of variance, and was defined as small: ES \geq 0.10; moderate: ES \geq 0.30, large: ES \geq 1.2; or very large: ES \geq 2.0; an error of $p \leq$ 0.05 is established⁸⁴. All analyses were based on intention-to-treat with an error of $p \leq$ 0.05. The statistical analysis was performed using the statistical package SPSS 24.0 for Windows.

Data availability

Data will be available upon request to the corresponding author.

Received: 9 February 2025; Accepted: 19 September 2025

Published online: 13 October 2025

References

- Vaduganathan, M., Mensah, G. A., Turco, J. V., Fuster, V. & Roth, G. A. The global burden of cardiovascular diseases and risk: A compass for future health. J. Am. Coll. Cardiol. 80, 2361–2371. https://doi.org/10.1016/j.jacc.2022.11.005 (2022).
- Liu, X. et al. Dose-Response association between physical activity and incident hypertension: A systematic review and Meta-Analysis of cohort studies. Hypertension 69, 813–820. https://doi.org/10.1161/hypertensionaha.116.08994 (2017).
- 3. Diaz, K. M. & Shimbo, D. Physical activity and the prevention of hypertension. Curr. Hypertens. Rep. 15, 659–668. https://doi.org/10.1007/s11906-013-0386-8 (2013).
- 5. Pelliccia, A. et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease: The Task Force on sports cardiology and exercise in patients with cardiovascular disease of the European Society of Cardiology (ESC). *Eur. Heart J.* 42, 17–96. https://doi.org/10.1093/eurheartj/ehaa605 (2020).
- 6. Rabi, D. M. et al. Hypertension canada's 2020 comprehensive guidelines for the Prevention, Diagnosis, risk Assessment, and treatment of hypertension in adults and children. Can. J. Cardiol. 36, 596–624. https://doi.org/10.1016/j.cjca.2020.02.086 (2020).
- 7. Mancia, G. et al. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). *J. Hypertens.* 41, 1874–2071. https://doi.org/10.1097/hjh.000000000003480 (2023).
- 8. Hanssen, H. et al. Personalized exercise prescription in the prevention and treatment of arterial hypertension: a consensus document from the European association of preventive cardiology (EAPC) and the ESC Council on hypertension. *Eur. J. Prev. Cardiol.* https://doi.org/10.1093/eurjpc/zwaa141 (2021).
- 9. López-Ruiz, I., Lozano, F. & Masia, M. D. González-Gálvez, N. Multicomponent training and optimal dosing strategies for adults with hypertension: A systematic review and meta-analysis of randomized controlled trials. *Sports (Basel)* 11. https://doi.org/10.33 90/sports11060115 (2023).
- Bavaresco Gambassi, B. et al. Short-duration dynamic power training with elastic bands combined with endurance training: a promising approach to hypertension management in older adults. J. Hypertens. 42, 735–742. https://doi.org/10.1097/hjh.0000000 000003681 (2024).
- 11. Gambassi, B. B. & Schwingel, P. A. Dynamic power training combined with endurance training reduces arterial stiffness and improves hemodynamic parameters in older adults diagnosed with grade 1 hypertension? *J. Hypertens.* 42, 1461–1462 (2024).
- 12. Masroor, S., Bhati, P., Verma, S., Khan, M. & Hussain, M. E. Heart rate variability following combined aerobic and resistance training in sedentary hypertensive women: A randomised control trial. *Indian Heart J.* 70 (Suppl 3), S28-s35. https://doi.org/10.1016/j.ihj.2018.03.005 (2018).
- 13. Dos Santos, E. S. et al. Acute and chronic cardiovascular response to 16 weeks of combined eccentric or traditional resistance and aerobic training in elderly hypertensive women: a randomized controlled trial. *J. Strength. Cond Res.* 28, 3073–3084. https://doi.org/10.1519/jsc.0000000000000537 (2014).
- Sousa, N., Mendes, R., Abrantes, C., Sampaio, J. & Oliveira, J. A randomized 9-month study of blood pressure and body fat responses to aerobic training versus combined aerobic and resistance training in older men. *Exp. Gerontol.* 48, 727–733. https://doi.org/10.1016/j.exger.2013.04.008 (2013).
- 15. Herrod, P. J. J. et al. Exercise and other nonpharmacological strategies to reduce blood pressure in older adults: a systematic review and meta-analysis. *J. Am. Soc. Hypertens.* 12, 248–267. https://doi.org/10.1016/j.jash.2018.01.008 (2018).
- Pescatello, L. S. et al. Physical activity to prevent and treat hypertension: A systematic review. Med. Sci. Sports Exerc. 51, 1314–1323. https://doi.org/10.1249/mss.000000000001943 (2019).
- 17. Costa Chaves, L. F. et al. Power exercises with elastic bands combined with endurance training improve pulse pressure, systolic blood pressure, and functional parameters in older adults. *Blood Press. Monit.* 30, 49–56. https://doi.org/10.1097/mbp.00000000000000033
- 18. Hejazi, K. et al. Differential effects of exercise training protocols on blood pressures and lipid profiles in older adults patients with hypertension: A systematic review and meta-analysis. *Arch. Gerontol. Geriatr.* 131, 105737. https://doi.org/10.1016/j.archger.2024 .105737 (2025).
- 19. Amare, F., Kiflu, A. & Taddese, A. Effects of concurrent continuous aerobic and short rest resistance exercise training on metabolic biomarkers in type 2 diabetes patients: a systematic review and meta-analysis. *Diabetol. Metab. Syndr.* 17, 290. https://doi.org/10.1 186/s13098-025-01838-x (2025).
- 20. Poli, L. et al. The effects of multicomponent training on Clinical, Functional, and psychological outcomes in cardiovascular disease: A narrative review. *Medicina (Kaunas)* 61. https://doi.org/10.3390/medicina61050822 (2025).
- González-Badillo, J. J., Sánchez-Medina, L., Ribas-Serna, J. & Rodríguez-Rosell, D. Toward a new paradigm in resistance training by means of velocity monitoring: A critical and challenging narrative. Sports Med. Open. 8, 118. https://doi.org/10.1186/s40798-0 22-00513-z (2022).
- 22. Badillo, J. J. G. & Serna, J. R. Bases De La programación Del Entrenamiento De Fuerza, Vol. 308 (Inde, 2002).
- 23. González-Badillo, J. J. Sánchez-Medina, L. Movement velocity as a measure of loading intensity in resistance training. *Int. J. Sports Med.* 31, 347–352. https://doi.org/10.1055/s-0030-1248333 (2010).
- SÁNCHEZ-MEDINA, L. & GONZÁLEZ-BADILLO, J. J. Velocity loss as an indicator of neuromuscular fatigue during resistance training. Med. Sci. Sports Exerc. 43, 1725–1734. https://doi.org/10.1249/MSS.0b013e318213f880 (2011).
- 25. Conceição, F., Fernandes, J., Lewis, M. & Gonzaléz-Badillo, J. J. Jimenéz-Reyes, P. Movement velocity as a measure of exercise intensity in three lower limb exercises. J. Sports Sci. 34, 1099–1106. https://doi.org/10.1080/02640414.2015.1090010 (2016).
- Díez-Fernández, D. M. et al. Estimating the one-repetition maximum on the leg-press exercise in female breast cancer survivors. PeerJ 11, e16175. https://doi.org/10.7717/peerj.16175 (2023).
- 27. Andreu-Caravaca, L., Ramos-Campo, D. J., Abellán-Aynés, O. & Rubio-Arias, J. Movement velocity as A measure of exercise intensity in persons with multiple sclerosis: A validity study. *J. Clin. Med.* **9**. https://doi.org/10.3390/jcm9082458 (2020).
- 28. McClean, Z. J. et al. A biopsychosocial model for Understanding training Load, Fatigue, and musculoskeletal sport injury in university athletes: A scoping review. J. Strength. Cond Res. 38, 1177–1188. https://doi.org/10.1519/jsc.00000000000004789 (2024).
- Motlagh, J. G. & Lipps, D. B. The contribution of muscular fatigue and shoulder biomechanics to shoulder injury incidence during the bench press exercise: A narrative review. J. Strength. Cond Res. 38, 2147–2163. https://doi.org/10.1519/jsc.00000000000004973 (2024).
- 30. Mancia, G. & Grassi, G. The autonomic nervous system and hypertension. Circul. Res. 114, 1804–1814 (2014).

- 31. Gallo, G., Volpe, M. & Savoia, C. Endothelial dysfunction in hypertension: current concepts and clinical implications. *Front. Med. (Lausanne)*. **8**, 798958, https://doi.org/10.3389/fmed.2021.798958 (2021).
- Gamboa, A. et al. Contribution of endothelial nitric oxide to blood pressure in humans. Hypertension 49, 170–177. https://doi.org/10.1161/01.hyp.0000252425.06216.26 (2007).
- 33. Node, K., Kitakaze, M., Yoshikawa, H., Kosaka, H. & Hori, M. Reduced plasma concentrations of nitrogen oxide in individuals with essential hypertension. *Hypertension* 30, 405–408. https://doi.org/10.1161/01.hyp.30.3.405 (1997).
- Tian, D. & Meng, J. Exercise for prevention and relief of cardiovascular disease: Prognoses, Mechanisms, and approaches. Oxid. Med. Cell. Longev. 2019, 3756750. https://doi.org/10.1155/2019/3756750 (2019).
- 35. Cheang, W. S. et al. PPARδ is required for exercise to attenuate Endoplasmic reticulum stress and endothelial dysfunction in diabetic mice. *Diabetes* **66**, 519–528. https://doi.org/10.2337/db15-1657 (2017).
- 36. Ashor, A. W. et al. Exercise modalities and endothelial function: a systematic review and dose-response meta-analysis of randomized controlled trials. Sports Med. 45, 279–296. https://doi.org/10.1007/s40279-014-0272-9 (2015).
- 37. Zhang, Y., Zhang, Y. J., Zhang, H. W., Ye, W. B. & Korivi, M. Low-to-Moderate-Intensity resistance exercise is more effective than High-Intensity at improving endothelial function in adults: A systematic review and Meta-Analysis. *Int. J. Environ. Res. Public. Health* 18. https://doi.org/10.3390/ijerph18136723 (2021).
- 38. Spence, A. L., Carter, H. H., Naylor, L. H. & Green, D. J. A prospective randomized longitudinal study involving 6 months of endurance or resistance exercise. Conduit artery adaptation in humans. *J. Physiol.* **591**, 1265–1275. https://doi.org/10.1113/jphysiol.2012.247387 (2013).
- 39. Fisher, J. P. & Paton, J. F. The sympathetic nervous system and blood pressure in humans: implications for hypertension. *J. Hum. Hypertens.* 26, 463–475. https://doi.org/10.1038/jhh.2011.66 (2012).
- Laterza, M. C. et al. Exercise training restores baroreflex sensitivity in never-treated hypertensive patients. Hypertension 49, 1298–1306. https://doi.org/10.1161/hypertensionaha.106.085548 (2007).
- 41. Trevizani, G. A. et al. Effect of resistance training on blood pressure and autonomic responses in treated hypertensives. *J. Strength. Cond Res.* 32, 1462–1470. https://doi.org/10.1519/jsc.0000000000001995 (2018).
- Pedersen, B. K. & Saltin, B. Exercise as medicine evidence for prescribing exercise as therapy in 26 different chronic diseases. Scand. J. Med. Sci. Sports. 25 (Suppl 3), 1–72. https://doi.org/10.1111/sms.12581 (2015).
- 43. Pedersen, B. K. The anti-inflammatory effect of exercise: its role in diabetes and cardiovascular disease control. *Essays Biochem.* 42, 105–117. https://doi.org/10.1042/bse0420105 (2006).
- Brandt, C. & Pedersen, B. K. The role of exercise-induced myokines in muscle homeostasis and the defense against chronic diseases. J. Biomed. Biotechnol. 2010, 520258. https://doi.org/10.1155/2010/520258 (2010).
- 45. Khalafi, M., Malandish, A., Rosenkranz, S. K. & Ravasi, A. A. Effect of resistance training with and without caloric restriction on visceral fat: A systemic review and meta-analysis. *Obes. Rev.* 22, e13275. https://doi.org/10.1111/obr.13275 (2021).
- 46. Wewege, M. A. et al. Sports Med. 52, 287-300 (2021).
- Mul, J. D., Stanford, K. I., Hirshman, M. F. & Goodyear, L. J. Exercise and regulation of carbohydrate metabolism. *Prog Mol. Biol. Transl Sci.* 135, 17–37. https://doi.org/10.1016/bs.pmbts.2015.07.020 (2015).
- 48. Way, K. L., Hackett, D. A., Baker, M. K. & Johnson, N. A. The effect of regular exercise on insulin sensitivity in type 2 diabetes mellitus: A systematic review and Meta-Analysis. *Diabetes Metab. J.* 40, 253–271. https://doi.org/10.4093/dmj.2016.40.4.253 (2016).
- 49. Sale, D. G. Neural adaptation to resistance training. *Med. Sci. Sports Exerc.* **20**, 135–145. https://doi.org/10.1249/00005768-198810 001-00009 (1988).
- 50. Enoka, R. M. Neural adaptations with chronic physical activity. *J. Biomech.* **30**, 447–455. https://doi.org/10.1016/s0021-9290(96)0 0170-4 (1997).
- 51. Del Vecchio, A. et al. The increase in muscle force after 4 weeks of strength training is mediated by adaptations in motor unit recruitment and rate coding. *J. Physiol.* **597**, 1873–1887. https://doi.org/10.1113/jp277250 (2019).
- 52. Folland, J. P. & Williams, A. G. Morphological and neurological contributions to increased strength. *Sports Med.* 37, 145–168 (2007).
- 53. Akimoto, T. et al. Exercise stimulates Pgc-1alpha transcription in skeletal muscle through activation of the p38 MAPK pathway. *J. Biol. Chem.* 280, 19587–19593. https://doi.org/10.1074/jbc.M408862200 (2005).
- 54. Booth, F. W., Ruegsegger, G. N., Toedebusch, R. G. & Yan, Z. Endurance exercise and the regulation of skeletal muscle metabolism. Prog Mol. Biol. Transl Sci. 135, 129–151. https://doi.org/10.1016/bs.pmbts.2015.07.016 (2015).
- 55. Jung, S. & Kim, K. Exercise-induced PGC-1α transcriptional factors in skeletal muscle. Integr. Med. Res. 3, 155-160 (2014)
- 56. Baar, K. et al. Adaptations of skeletal muscle to exercise: rapid increase in the transcriptional coactivator PGC-1. Faseb j. 16, 1879–1886. https://doi.org/10.1096/fj.02-0367com (2002).
- Halling, J. F. & Pilegaard, H. PGC-1α-mediated regulation of mitochondrial function and physiological implications. Appl. Physiol. Nutr. Metab. 45, 927–936. https://doi.org/10.1139/apnm-2020-0005 (2020).
- 58. Coffey, V. G. & Hawley, J. A. Concurrent exercise training: do opposites distract? *J. Physiol.* 595, 2883–2896. https://doi.org/10.11 13/jp272270 (2017).
- 59. Pareja-Blanco, F. et al. Effects of velocity loss during resistance training on athletic performance, strength gains and muscle adaptations. *Scand. J. Med. Sci. Sports.* 27, 724–735. https://doi.org/10.1111/sms.12678 (2017).
- Schroeder, E. C., Franke, W. D., Sharp, R. L. & Lee, D. C. Comparative effectiveness of aerobic, resistance, and combined training on cardiovascular disease risk factors: A randomized controlled trial. *PLoS One.* 14, e0210292. https://doi.org/10.1371/journal.po ne.0210292 (2019).
- 61. Gorostidi, M. et al. Guía práctica sobre El diagnóstico y Tratamiento de La hipertensión arterial En España, 2022. Sociedad Española de Hipertensión-Liga Española Para La Lucha contra La Hipertensión arterial (SEH-LELHA). Hipertensión Y Riesgo Vascular. 39, 174–194 (2022).
- 62. Stewart, K. J. et al. Effect of exercise on blood pressure in older persons: a randomized controlled trial. *Arch. Intern. Med.* 165, 756–762. https://doi.org/10.1001/archinte.165.7.756 (2005).
- 63. Buch, A. et al. Validation of a multi-frequency bioelectrical impedance analysis device for the assessment of body composition in older adults with type 2 diabetes. *Nutr. Diabetes.* 12, 45. https://doi.org/10.1038/s41387-022-00223-1 (2022).
- 64. Lahav, Y., Goldstein, N. & Gepner, Y. Comparison of body composition assessment across body mass index categories by two multifrequency bioelectrical impedance analysis devices and dual-energy X-ray absorptiometry in clinical settings. *Eur. J. Clin. Nutr.* 75, 1275–1282. https://doi.org/10.1038/s41430-020-00839-5 (2021).
- 65. Marfell Jones, M., Olds, T., Stewart, A. & Carter, L. Sociedad Internacional para el Avance de la Cineantropometría (ISAK). *Manual ISAK* (2006).
- 66. Hypertension, E. S. o. (2010).
- 67. Stergiou, G. S. et al. European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement. *J. Hypertens.* 39, 1293–1302. https://doi.org/10.1097/hjh.0000000000002843 (2021).
- 68. Mathiowetz, V. et al. Grip and pinch strength: normative data for adults. Arch. Phys. Med. Rehabil. 66, 69-74 (1985).
- 69. Rodríguez-Rosell, D., Yáñez-García, J. M., Sánchez-Medina, L., Mora-Custodio, Ř. & González-Badillo, J. J. Relationship between velocity loss and repetitions in reserve in the bench press and back squat exercises. *J. Strength. Conditioning Res.* 34, 2537–2547 (2020).

- 70. Sánchez-Medina, L., Pallarés, J. G., Pérez, C. E., Morán-Navarro, R. & González-Badillo, J. J. Estimation of relative load from bar velocity in the full back squat exercise. *Sports Med. Int. open.* 1, E80–E88 (2017).
- Escamilla, R. F. Knee biomechanics of the dynamic squat exercise. Med. Sci. Sports Exerc. 33, 127–141. https://doi.org/10.1097/00 005768-200101000-00020 (2001).
- 72. Rodríguez-Rosell, D. et al. Velocity-based resistance training: impact of velocity loss in the set on neuromuscular performance and hormonal response. *Appl. Physiol. Nutr. Metab.* **45**, 817–828. https://doi.org/10.1139/apnm-2019-0829 (2020).
- 73. Morán-Navarro, R. et al. Movement velocity as a measure of level of effort during resistance exercise. J. Strength. Cond Res. 33, 1496–1504. https://doi.org/10.1519/jsc.0000000000002017 (2019).
- González-Badillo, J. J., Yañez-García, J. M. & Mora-Custodio, R. Rodríguez-Rosell, D. Velocity loss as a variable for monitoring resistance exercise. Int. J. Sports Med. 38, 217–225. https://doi.org/10.1055/s-0042-120324 (2017).
- 75. Marqueta, P. M. et al. Pruebas de esfuerzo en medicina del deporte. Documento de consenso de la Sociedad Española de Medicina del Deporte (SEMED-FEMEDE). *Arch. Med. Deporte* 33, 5–83 (2016).
- 76. Ferguson, B. J. Can. Chiropr. Assoc. 58, 328 (2014).
- Cuadros, L., Ismail, H. & Ho, K. Evaluation of reliability of MYZONE MZ-3 heart rate monitor: A study for the future of telephysiotherapy for preoperative prehabilitation in cancer patients. *Telemed J. E Health.* 23, 334–338. https://doi.org/10.1089/tm j.2016.0138 (2017).
- 78. Borg, G. Borg's Perceived Exertion and Pain Scales (Human Kinetics, 1998).
- 79. Lea, J. W. D., O'Driscoll, J. M., Hulbert, S., Scales, J. & Wiles, J. D. Sports Med. Open 82 (2022).
- 80. Allen, H., Coggan, A. R. & McGregor, S. Training and Racing with a Power Meter (VeloPress, 2019).
- Pareja-Blanco, F., Sánchez-Medina, L., Suárez-Arrones, L. & González-Badillo, J. J. Effects of velocity loss during resistance training on performance in professional soccer players. *Int. J. Sports Physiol. Perform.* 12, 512–519. https://doi.org/10.1123/ijspp.2016-0170 (2017)
- 82. Karvonen, M. J., Kentala, E. & Mustala, O. The effects of training on heart rate; a longitudinal study. Ann. Med. Exp. Biol. Fenn. 35, 307–315 (1957).
- 83. Mølmen, K. S., Almquist, N. W. & Skattebo, Ø. Effects of exercise training on mitochondrial and capillary growth in human skeletal muscle: A systematic review and Meta-Regression. Sports Med. 55, 115–144. https://doi.org/10.1007/s40279-024-02120-2 (2025).
- 84. Hopkins, W. G., Marshall, S. W., Batterham, A. M. & Hanin, J. Progressive statistics for studies in sports medicine and exercise science. *Med. Sci. Sports Exerc.* 41, 3–13. https://doi.org/10.1249/MSS.0b013e31818cb278 (2009).

Acknowledgements

The authors would like to thank you for the indispensable collaboration of Centro Oxigeno Sport & Wellness.

Author contributions

I.L.-R. and N.G.-G: conceived and designed the experiments; I.L.-R., F.L.R.-P., M.D.M., J.R.H.-E. and N.G.-G.: performed the experiments; I.L.-R. and N.G.-G: analysed the data, I.L.-R. and N.G.-G: wrote the manuscript; I.L.-R., F.L.R.-P., M.D.M., J.R.H.-E. and N.G.-G.: proofed the manuscript. All authors have proofed the article.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to N.G.-G.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Open Access This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

© The Author(s) 2025