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Training, detraining and retraining effects of moderate vs. high intensity exercise training programme on cardiovascular risk factors

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Background: The aim of the present study was to analyse the effect of 12 weeks of training, 7 weeks of detraining and 16 weeks of retraining using a moderate or high intensity training programme on cardiovascular risk factors in hypertensive patients.

Method: Thirty-four patients took part in the study. The intensity training was 80–90% of maximum heart rate for the high-intensity training (HIT) group ($n = 15$) and at 50–70% of maximum heart rate for the moderate training (MT) group ($n = 19$). Blood pressure, body composition, lipid profile, fasting glucose, strength and cardiovascular fitness were analysed.

Results: The first training period did not decrease blood pressure, but the second training period saw significant decreases in blood pressures in HIT group. Moreover, 12 weeks of MT or HIT did not decrease body mass, body mass index or fat mass. However, after 7 weeks of detraining, the inclusion of a second training period using HIT saw decreases in these body composition variables. Both training periods and intensities improved high-density lipoprotein and low-density lipoprotein, but only HIT decreased total cholesterol. In addition, after 7 weeks of detraining, the lipid profile variables returned to baseline values. Additionally, 16 weeks of retraining with HIT or MT decreased blood glucose significantly. Moreover, MT and HIT training programmes in both periods improved cardiorespiratory fitness, but with 7 weeks of detraining, it returned to baseline values.

Conclusion: Our data demonstrated the effectiveness of the inclusion of a MT or HIT programme as adjuvant therapy in hypertensive patients.

Keywords: cardiovascular risk, community program, exercise is medicine, hypertension

Abbreviations: [Lac], blood lactate concentration; CVDs, cardiovascular disease; DBP, diastolic blood pressure; HIT, high-intensity training; MT, moderate training; RPE, rating of perceived exertion; SBP, systolic blood pressure

INTRODUCTION

Cardiovascular disease (CVD) remains the most common cause of death worldwide, accounting for nearly half of all noncommunicable disease deaths [1]. Almost half of deaths are due to acute myocardial infarction and one-third of them are due to cerebrovascular accident worldwide [1]. Therefore, reducing the prevalence of cardiovascular risk factors implies reducing the number of deaths due to CVD, and this fact is of great importance for public health. Especially interesting is the reduction of modifiable lifestyle factors, such as smoking, physical inactivity, unhealthy diet and high alcohol consumption [2]. In this way, a sedentary lifestyle is prevalent in most industrialized countries [3]. Over the last six decades, accumulating epidemiological evidence has shown that being physically active is beneficial for health, particularly for the cardiovascular system [3,4]. Physical inactivity ranks fourth among the leading risk factors for mortality worldwide [5]. The role of physical activity in the primary prevention of CVD appears to be established.

Continuous moderate aerobic training is the most common type of training programme to treat cardiovascular risk factors. This type of exercise increases cardiorespiratory fitness and enhances endothelial function, insulin signalling and skeletal muscle biogenesis, as well as decreases blood pressure and reduces body weight and fat [6]. However, during the last years, the application of training programmes that include high-intensity aerobic interval training has grown. Accordingly, high-intensity aerobic interval training programmes reduce blood pressure [7], improve cardiorespiratory fitness [8] and heart function [9], induce mitochondrial biogenesis [10] and improve insulin sensitivity [11]. Although the two training programmes (moderate

training [MT] and high-intensity training [HIT]) are effective to reduce cardiovascular risk factors, previous studies have demonstrated that HIT is superior to MT in reversing risk factors [6]. Therefore, exercise intensity is an important factor in the optimization of aerobic capacity improvements and reversing cardiovascular risk factors.

Some previous studies have analysed the effects of exercise discontinuity, inserting training and detraining periods [12–14], in patients with cardiovascular risk factors. In this way, some training adaptations rapidly return to basal values (fat oxidation, cardiorespiratory fitness and triglycerides), whereas others are retained during 1 month of detraining (body composition and high-density lipoprotein [HDL] cholesterol). In addition, a previous study has analysed the effect of consecutive years of 4-month aerobic interval training and concluded that, to chronically improve cardiovascular risk factors, at least two training periods are required. In this way, the blood pressure does not fully return to pretraining values, allowing a cumulative improvement [12]. However, the effects of detraining and retraining according to the training intensity (MT or HIT) are not fully understood. Therefore, the aim of the present study was to analyse the effect of 12 weeks of training, 7 weeks of detraining and 16 weeks of a second period of training using a MT or HIT programme on body composition, blood pressure, strength, cardiorespiratory fitness and fasting blood glucose and lipid profile variables in hypertensive patients treated with antihypertensive therapy. Based on the previous research, our hypothesis was that HIT and MT would significantly improve cardiorespiratory fitness, strength and biochemical and body composition markers in hypertensive patients. In addition, it was hypothesized that HIT would induce greater adaptations and would reduce the detraining effect in these variables when compared to MT.

METHODS

Design

We developed a prospective observational study to analyse the effect of 12 weeks of training, 7 weeks of detraining and 16 weeks of a second period of retraining using a MT or HIT programme on body composition, blood pressure, cardiorespiratory fitness, strength and fasting blood glucose and lipid profile variables in hypertensive patients following antihypertensive therapy (Fig. 1). Prior to the study,

participants read and signed a form to provide informed consent. In addition, the study conforms to the Declaration of Helsinki and was approved by the Catholic University of Murcia's Science Ethics Committee (C231111).

Participants

All participants included in the study were referred by their primary care physicians who prescribed physical exercise as a healthy lifestyle intervention added to pharmacological treatment in the framework of a behavioural and healthy lifestyle programme (i.e. 'ACTIVA-Murcia Programme'). Patients went to the sports centre closest to their home. The inclusion criteria were: men or women aged from 40 to 65 years; hypertensive patients receiving antihypertensive therapy (one or more drugs) for at least 1 year; and patients without experience in regular physical exercise. On the other hand, the exclusion criteria were: patients with terminal disease, ischaemic cardiopathy, cerebrovascular disease or cardiovascular pathology (i.e. peripherical arterial disease); participants with a pathology which limited aerobic or resistance training (i.e. muscle disorders, pulmonary obstructive disease, arrhythmia...); or participants were excluded from the analysis if they did not fulfil a minimum compliance of 66% of attendance of the training sessions.

Testing protocol

As Fig. 1 shows, testing sessions were carried out four times: at baseline (T1), at the end of the first training period of 12 weeks duration (T2), after 7 weeks of detraining (T3) and at the end of the study after 16 weeks of a second retraining period (T4). All the tests were measured in one visit to the laboratory. Moreover, a familiarization session was included 48–72 h before the training session where participants performed the isokinetic tests. During the testing session, blood pressure, biochemical blood analysis, body composition tests, isokinetic tests and cardiorespiratory tests were performed.

Two days after the last training session, in all of the testing moments, the blood pressure profile was measured using an oscillometric device (SpaceLabs 90217; Spacelabs Healthcare, Snoqualmie, Washington, USA). Blood pressure was assessed every 30 min during the day and every 60 min during the night. Mean daytime, awake and sleep values of the systolic blood pressure (SBP) and diastolic blood pressure (DBP) were extracted.
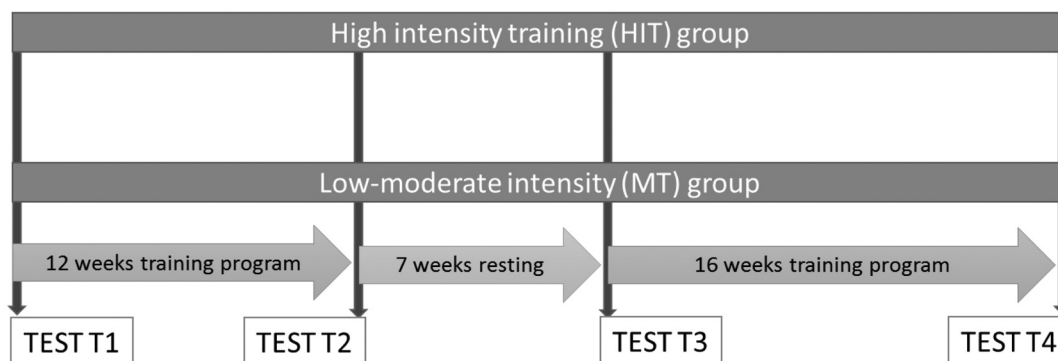

FIGURE 1 Experimental design.

A blood sample (2.5 ml) was withdrawn from the antecubital vein using a sterile technique to analyse haematological variables. Blood samples were taken before breakfast after an overnight fast. Blood extraction was performed with the participant seated. The haematological variables of total cholesterol (mg/dl), LDH (mg/dl), HDL (mg/dl), blood glucose (mg/dl), glycosylated haemoglobin (%) and triglycerides (mg/dl) were analysed using an automatic haematology analyser (Pentra 80-HORIBA ABX; Horiba Medical, Northampton, UK).

Body composition was assessed with a segmental multifrequency bioimpedance analyser (Tanita BC-601; Tanita-Corp., Tokyo, Japan). We conducted the test at the same time, in the same participant order, and in the same place, with a constant temperature and humidity. To carry out the tests, the participants stood upright on foot electrodes on the instrument platform, with legs and thighs apart and arms not touching the torso. They were barefoot and without excess clothing. Body height was measured using a stadiometer (Seca 700; Seca Ltd, Germany). Body mass (kg), body mass index (kg/m^2), fat mass (%) and fat free mass (kg) were analysed. In addition, waist circumference (cm) was assessed.

Regarding isokinetic tests, following a standardized warm-up consisting of 10 min of submaximal stationary cycling and dynamic stretching, an isokinetic dynamometer (Biodex 3; Biodex Corporation, Shirley, New York, USA) was used to measure peak torque values and the torque angle of right leg during knee flexion and extension. The motor axis was visually aligned with the axis of the knee. The participant was seated and stabilized by straps so that only the knee to be tested was moving with a single degree of freedom. The dynamometer was calibrated, using the protocol from the Biodex 6000 manual, at the beginning of the test session. All participants performed five continuous maximum effort concentric contractions of the knee flexors and extensors at the angular velocities of 60°/s and 270°/s. Before the trial set, a specific warm-up consisting of two series at 50 and 80% of the perceived maximum effort of the participant were carried out. The test started 5 min after the warm-up trials had been completed to prevent fatigue. The first and last repetitions were excluded from the data analysis. The second, third and fourth contractions were averaged for the determination of the optimum angle by fitting a fourth order polynomial curve. Only the highest peak torque values of the fitted curve of the flexors and extensors of each velocity were used in the analysis.

After 15 min of rest, aerobic exercise testing on a treadmill following a modification of the Balke–Ware protocol [15], but using the same protocol for men and women, was carried out. Warm-up exercises were developed previous to the test during 2 min: the first minute at a speed of 3 km/h and 1.5% slope and the second minute at 4 km/h and 4% slope. The test was divided into 15 phases of 1 min each with increments of speed (0.2 km/h) and slope (1%), starting at a speed of 5 km/h and 5% slope. All participants were monitored by a gas analyser (Jaeger Oxicom Pro; Jaeger, Wuerzburg, Germany). Maximal oxygen uptake (VO$_2$ max) and time to exhaustion were analysed. Based on previous studies [16], criteria to assume that maximal effort was reached were at least two of the following: a levelling-off of oxygen uptake, maximal respiratory exchange ratio ≥1.10, age predicted HRmax ≥90% and maximal rate of perceived exertion ≥19. Moreover, capillary blood samples (5 μl) for blood lactate concentration ([Lac]) analysis were collected from a finger pick 2 min after the end of the test and analysed using a Lactate Pro analyser (Arkay, Inc., Kyoto, Japan). After the test, second ventilatory threshold was determined in the second increase in ventilation (VE) with a concomitant rapid increase in VE/VO$_2$ and VE/VCO$_2$ and decrease of end-tidal CO$_2$ tension. Maximum oxygen consumption (min/kg per min), second ventilatory threshold (min/kg per min), time to exhaustion (min), time to second ventilatory threshold (min) and blood lactate concentration were analysed.

Training programme

The training programme characteristics were similar in both groups, but the intensity was moderate or high in each group (MT vs. HIT). The training programme included two training periods separated by a 7-week detraining period, which was coincident with a vacation period: 12 weeks duration (from September to December) and 16 weeks duration (from February to June). The frequency of training was 3 days per week in sessions of 60 min duration, with at least 48 h of rest between sessions. The physical training programme was focused on basic exercises of endurance (global bodily activities), strength (specific muscle regions) and flexibility. Specifically, sessions were developed using circuit-based training that provided a dynamic combination of exercises and included the following phases: a warm-up with muscular activation, articular mobility and dynamic exercises; main part that included an aerobic interval exercise at 80–90% of the maximum heart rate for the HIT group and at 50–70% of the maximum heart rate for the MT group; and a cool down based on stretching exercises. Heart rate (HR) was monitored using a pulsometer (Polar RS400; Polar Electro Oy, Kempele, Finland) and recorded. Later, HR data were analysed to determine if the participants fulfilled the intensity programmed into each training programme. Moreover, the rating of perceived exertion (RPE) was also used for intensity monitoring. During the training programme, training intensity was progressively increased according to HR and RPE. All sessions were monitored and supervised by the same researcher, who graduated in Sports Sciences and specialized in strength and conditioning training.

Statistical analysis

Data collection, treatment and analysis were performed using the SPSS for Windows statistical package (version 25.0; IBM, Armonk, New York, USA). Descriptive statistics with measures of central tendency and dispersion were used. The assumption of normality and homoscedasticity were verified using the Shapiro–Wilk W and Levene test. In addition, a two-way analysis (training group × time) of variance with repeated measures and Bonferroni post hoc test were used to investigate differences between study variables. The effect size was calculated using eta-squared (η^2). For all procedures, a level of significance of $P \leq 0.05$ was established.

RESULTS

Based on previous studies [17], a total of 18 participants per group would be needed to be able of establishing statistical

differences between programs (MT vs. HIT) in systolic blood pressure (80% statistical power; $\alpha = 0.05$). Physicians prescribed physical exercise to 41 individuals. On the first day, 37 (90.2%) patients showed up at the sports centre. The study population included 37 hypertensive individuals: 17 started the HIT programme and 20 started the MT programme. However, 34 (91.9%) participants (15 men, 19 women; mean age 56.0 ± 5.5 years) completed the programme (minimum compliance of 66% of training sessions during the programmed sessions): the HIT group ($n = 15$; 8 (53.3%) women; 55.1 ± 5.9 years; 77.0 ± 12.8 kg; 163.0 ± 8.0 cm) and MT group ($n = 19$; 11 (57.9%) women; 56.7 ± 5.1 years; 81.5 ± 14.4 kg; 162.7 ± 7.0 cm). Three participants did not finish the program and they were excluded for the analysis (MT group: $n = 1$; man; 50.0 years; 81.0 kg; 162.9 cm; HIT group: $n = 2$; 1 (50%) woman; 49.5 ± 9.2 years; 90.6 ± 13.2 kg; 171.6 ± 4.0 cm).

There were no statistically significant differences in age ($T = 0.421$; $P = 0.421$), BMI ($T = 1.229$; $P = 0.228$), fat body mass ($T = 1.229$; $P = 0.217$), SBP ($T = 0.275$; $P = 0.785$) or DBP ($T = 0.032$; $P = 0.974$) when baseline values were compared (between-group differences). Antihypertensive pharmacological treatment included angiotensin receptor blockers in 52.9% of patients, diuretics in 23.5% of patients, beta-blockers in 17.6% of patients, calcium antagonists in 11.8% of patients, angiotensin-converting enzyme inhibitors in 26.5% of patients and alpha-blockers in 2.9% of patients. In one patient assigned to the HIT group, the dose of beta-blocker was reduced by their primary care physician during the study; 26% of the patients were on lipid-lowering drugs and none of them changed their dosage during the programme.

With respect to the ambulatory blood pressure variables (Table 1), a significant effect was observed on the mean daytime SBP ($F = 6.664$; $P = 0.001$; $\eta^2 = 0.172$) and on the awake SBP ($F = 4.268$; $P = 0.007$; $\eta^2 = 0.118$), showing a decrease after the second retraining period of the HIT programme in comparison with the other testing sessions (baseline, after the first training period and after 7 weeks of detraining). Similarly, a significant effect was observed on the mean daytime DBP ($F = 26.352$; $P < 0.001$; $\eta^2 = 0.279$), awake DBP ($F = 3.139$; $P = 0.033$; $\eta^2 = 0.089$) and sleep DBP ($F = 3.304$; $P = 0.024$; $\eta^2 = 0.094$), showing the same behaviour of the HIT programme at the end of the programme in comparison with the other three testing sessions and detecting a significant difference between the two types of exercise (MT vs. HIT) at the end of the second retraining period ($P = 0.011$). No significant main effect was observed in sleep SBP.

No main effect was observed in body composition variables (i.e. body mass, BMI, fat mass and fat free mass) (Table 2). However, a significant decrease of fat mass was observed from baseline to the end of the second period of retraining in the HIT programme. Conversely, a significant main effect was found on waist circumference ($F = 3.029$; $P = 0.038$; $\eta^2 = 0.151$) seeing a significant decrease at the end of the retraining period in HIT group. On the other hand, a significant effect was observed on total cholesterol ($F = 3.031$; $P = 0.033$; $\eta^2 = 0.087$), showing a significant difference between groups after the first period of training ($P = 0.02$) and decreasing significantly at the end of the second training programme in the HIT group ($P = 0.006$). In this way, a significant effect was found in HDL ($F = 2.879$; $P = 0.04$; $\eta^2 = 0.083$), LDL ($F = 4.583$; $P = 0.005$; $\eta^2 = 0.125$). Both the HIT and MT programmes significantly increased HDL after the first and second training periods, without differences between training programmes. Furthermore, LDL decreased significantly after the second training period in the HIT programme, but no change was observed in the MT programme at this point. Finally, no main effect was

TABLE 1. Ambulatory blood pressure variables in the four testing points after HIT and MT programmes

Variable		Baseline [1]		After 1st training period (12 weeks) [2]		After 7 weeks of detraining [3]		After 2nd training period (16 weeks) [4]					
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	F	P	η^2	Pairwise comparison
Mean daytime systolic blood pressure in 24 h (mmHg)	MT	130.3	9.7	130.3	9.0	131.6	11.0	127.8	9.6	6.664	0.001	0.172	HIT: 1 vs. 4 ($P < 0.001$); 2 vs. 4 ($P < 0.001$); 3 vs. 4 ($P < 0.001$)
	HIT	131.3	11.9	132.1	11.1	134.5	12.3	121.9	11.9				
Mean daytime diastolic blood pressure in 24 h (mmHg)	MT	80.3	6.2	81.1	5.5	81.8	6.2	79.6	6.6	26.352	<0.001	0.279	MT vs. HIT: 4 ($P = 0.011$); HIT: 1 vs. 4 ($P < 0.001$); 1 vs. 3 ($P = 0.044$); 2 vs. 4 ($P < 0.001$); 3 vs. 4 ($P < 0.001$)
	HIT	80.4	9.0	81.3	8.1	83.7	9.2	73.1	7.6				
Awake systolic blood pressure (mmHg)	MT	136.3	10.2	135.6	12.1	137.4	12.5	135.0	11.2	4.268	0.007	0.118	MT vs. HIT: 4 ($P = 0.02$); HIT: 1 vs. 4 ($P = 0.001$); 2 vs. 4 ($P = 0.004$); 3 vs. 4 ($P < 0.001$)
	HIT	131.9	10.6	132.8	9.4	134.6	11.6	125.5	11.1				
Awake diastolic blood pressure (mmHg)	MT	83.3	1.8	83.1	1.6	84.6	1.9	82.5	1.7	3.139	0.033	0.089	HIT: 1 vs. 4 ($P = 0.016$); 2 vs. 4 ($P = 0.006$); 3 vs. 4 ($P < 0.001$)
	HIT	84.3	2.0	85.0	1.8	86.7	2.1	80.2	1.9				
Sleep Systolic blood pressure (mmHg)	MT	127.4	12.6	127.5	14.0	128.9	10.4	126.3	14.2	1.892	0.136	0.056	HIT: 3 vs. 4 ($P < 0.001$)
	HIT	121.9	10.7	122.6	11.2	125.7	11.1	117.8	11.3				
Sleep diastolic blood pressure (mmHg)	MT	73.8	10.1	74.1	12.1	75.0	9.8	73.4	9.7	3.304	0.024	0.094	HIT: 1 vs. 4 ($P = 0.006$); 2 vs. 4 ($P = 0.003$); 3 vs. 4 ($P < 0.001$)
	HIT	73.6	6.3	75.1	5.7	75.3	5.4	68.8	6.6				

HIT, high-intensity training; MT, moderate training; SD, standard deviation.

TABLE 2. Body composition and biochemical variables in the four testing points after HIT and MT programmes

Variable		Baseline [1] Mean	SD	After 1st training period (12 weeks) [2] Mean	SD	After 7 weeks of detraining [3] Mean	SD	After 2nd training period (16 weeks) [4] Mean	SD	F	P	η^2	Pairwise comparison
Body mass (kg)	MT	81.5	14.4	81.1	14.7	81.4	14.3	80.9	13.8	2.480	0.066	0.072	HIT: 1 vs. 4 (P = 0.008);
	HIT	77.0	12.8	76.9	13.2	76.4	13.3	75.3	13.3				2 vs. 4 (P = 0.017)
BMI (kg/m²)	MT	30.6	3.8	30.4	3.9	30.6	3.8	30.4	3.5	2.645	0.054	0.076	HIT: 1 vs. 4 (P = 0.007);
	HIT	29.0	4.1	28.9	4.2	28.7	4.2	28.3	4.1				2 vs. 4 (P = 0.017)
Fat mass (%)	MT	32.7	9.3	31.9	9.7	31.6	9.6	31.4	8.5	0.442	0.723	0.014	HIT: 1 vs. 2 (P = 0.041);
	HIT	28.9	8.0	27.8	7.4	27.6	7.6	26.8	7.1				1 vs. 4 (P = 0.006)
Fat free mass (kg)	MT	48.8	10.4	49.2	10.5	49.8	10.3	49.6	10.7	1.231	0.303	0.037	
	HIT	48.1	11.3	49.1	11.1	48.9	12.0	48.5	11.9				
Waist circumference (cm)	MT	87.8	5.3	87.8	5.3	87.7	5.0	87.3	4.5	3.029	0.038	0.151	HIT: 1 vs. 4 (P = 0.001);
	HIT	86.0	6.0	85.7	5.8	85.5	5.7	84.2	5.7				2 vs. 4 (P = 0.004); 3 vs. 4 (P = 0.004)
Total cholesterol (mg/dl)	MT	198.2	25.8	190.3	22.2	200.8	30.3	191.5	20.1	3.031	0.033	0.087	MT vs. HIT: 2 (P = 0.02); HIT: 1 vs. 4 (P = 0.006); 2 vs. 4 (P = 0.002); 3 vs. 4 (P = 0.003)
	HIT	208.9	29.7	208.8	21.7	213.2	26.9	191.1	25.8				
HDL (mg/dl)	MT	60.8	11.6	66.2	11.9	54.8	9.5	59.4	8.7	2.879	0.040	0.083	HIT: 1 vs. 2 (P < 0.001); 1 vs. 3 (P = 0.009); 2 vs. 3 (P < 0.001); 2 vs. 4 (P < 0.001); 3 vs. 4 (P < 0.001); MT: 1 vs. 2 (P < 0.001); 1 vs. 3 (P = 0.003); 2 vs. 3 (P < 0.001); 2 vs. 4 (P < 0.001); 3 vs. 4 (P = 0.011)
	HIT	58.7	9.0	65.1	8.3	52.6	6.9	62.3	5.9				
LDL (mg/dL)	MT	109.3	22.6	98.5	22.9	118.3	22.4	107.3	17.3	4.583	0.005	0.125	HIT: 1 vs. 4 (P = 0.009); 2 vs. 3 (P = 0.011); 2 vs. 4 (P = 0.039); 3 vs. 4 (P < 0.001); MT: 2 vs. 3 (P < 0.001)
	HIT	117.6	28.7	113.1	21.0	127.1	25.8	100.2	24.2				
Triglycerides (mg/dl)	MT	140.6	71.9	127.7	53.3	138.7	68.6	123.9	53.1	0.406	0.749	0.013	HIT: 3 vs. 4 (P = 0.009)
	HIT	162.9	69.0	153.3	66.9	167.4	67.4	142.7	61.0				
Blood glucose (mg/dl)	MT	102.1	17.4	104.1	17.3	106.5	17.6	99.8	16.38.7	2.194	0.094	0.219	HIT: 1 vs. 4 (P < 0.001); 2 vs. 4 (P = 0.012); 3 vs. 4 (P < 0.001); MT: 3 vs. 4 (P = 0.045)
	HIT	106.5	10.9	106.4	14.2	110.8	14.3	97.3					
Glycosylated haemoglobin (%)	MT	6.0	0.3	5.8	0.5	6.0	0.5	6.0	0.4	1.760	0.160	0.052	HIT: 1 vs. 2 (P = 0.026); MT: 1 vs. 2 (P = 0.002); 2 vs. 3 (P = 0.017)
	HIT	6.2	0.6	6.0	0.6	6.2	0.7	5.9	0.5				

BMI, body mass index; HDL, high-density lipoprotein; HIT, high-intensity training; LDL, low-density lipoprotein; MT, moderate training; SD, standard deviation.

observed in blood glucose, glycosylated haemoglobin and triglycerides.

Regarding cardiorespiratory fitness variables, no main effect was observed in time to exhaustion and time to second ventilatory threshold, VO_2max or lactate variables (Table 2). However, a significant effect was observed on the second ventilatory threshold ($F = 3.085$; $P = 0.031$; $\eta^2 = 0.088$), showing a significant difference between MT and HIT at the end of the retraining period ($P = 0.007$). In this way, both the HIT and MT programmes improved second ventilatory threshold in the first training period and in the second retraining period but HIT produced greater improvements. On the other hand, no main effect was observed in isokinetic strength variables (Table 3).

DISCUSSION

The aim of the present study was to analyse the effect of 12 weeks of training, 7 weeks of detraining and 16 weeks of a second period of training using a MT or HIT programme on body composition and waist circumference, ambulatory blood pressure outcomes, strength, cardiorespiratory fitness and blood sugar and lipid profile variables in hypertensive patients. The main findings were: the first 12 weeks of the training programme did not decrease blood pressure but the second training period (16 weeks) significantly decreased SBP and DBP in HIT programme; 12 weeks of MT or HIT did not decrease body mass, BMI or fat mass; however, after 7 weeks of detraining, the inclusion of a second training period of 16 weeks using HIT significantly decreased these body composition variables and waist circumference; both training periods and intensities improved HDL and LDL, but only HIT decreased total cholesterol (in addition, with only 7 weeks of detraining, the lipid profile variables returned to baseline values; the first period training period did not decrease fasting blood glucose but a second 16-week training period of HIT or MT decreased it significantly; both MT and HIT programmes and both

TABLE 3. Cardiorespiratory fitness and isokinetic strength variables in the four testing points after HIT and MT programmes

Variable		Baseline [1]		After 1st training period (12 weeks) [2]		After 7 weeks of detraining [3]		After 2nd training period (16 weeks) [4]					
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	F	P	η^2	Pairwise comparison
VO$_2$max (ml/kg per min)	MT	27.7	4.3	29.7	4.5	27.3	4.3	30.2	4.2	2.118	0.103	0.062	HIT: 1 vs. 2 ($P=0.002$); 1 vs. 4 ($P<0.001$); 2 vs. 3 ($P<0.001$); 3 vs. 4 ($P<0.001$); MT: 1 vs. 2 ($P=0.002$); 1 vs. 4 ($P<0.001$); 2 vs. 3 ($P=0.001$); 3 vs. 4 ($P<0.001$)
	HIT	29.3	5.1	31.6	4.7	28.7	4.5	33.3	5.5				
Second ventilatory threshold (ml/kg per min)	MT	23.3	3.7	25.6	4.2	23.9	3.7	26.4	4.0	3.085	0.031	0.088	MT vs. HIT: 4 ($P=0.007$); HIT: 1 vs. 2 ($P=0.004$); 1 vs. 4 ($P<0.001$); 2 vs. 3 ($P=0.007$); 2 vs. 4 ($P<0.001$); 3 vs. 4 ($P<0.001$); MT: 1 vs. 2 ($P=0.011$); 1 vs. 4 ($P=0.006$); 2 vs. 3 ($P=0.006$); 3 vs. 4 ($P=0.006$)
	HIT	24.6	5.1	27.5	3.9	25.5	4.1	30.4	4.1				
Lactate (mmol/l)	MT	7.2	1.8	8.4	2.5	8.1	2.1	9.0	2.5	0.507	0.679	0.016	HIT: 1 vs. 4 ($P=0.001$); 3 vs. 4 ($P=0.016$); MT: 1 vs. 2 ($P=0.014$); 1 vs. 4 ($P<0.001$); 3 vs. 4 ($P=0.001$)
	HIT	8.0	1.7	8.8	2.3	8.9	1.9	9.8	1.7				
Time to second ventilatory threshold (min)	MT	5.6	2.7	7.9	1.4	6.3	1.9	7.7	1.9	2.474	0.066	0.072	HIT: 1 vs. 4 ($P<0.001$); 2 vs. 4 ($P=0.019$); 3 vs. 4 ($P<0.001$); MT: 1 vs. 2 ($P<0.001$); 1 vs. 4 ($P<0.001$); 2 vs. 3 ($P=0.001$); 3 vs. 4 ($P=0.001$)
	HIT	6.1	2.5	7.6	2.1	6.8	2.2	9.0	2.2				
Time to exhaustion (min)	MT	7.7	2.8	9.0	2.6	8.9	2.5	10.11	1.8	1.433	0.238	0.043	HIT: 1 vs. 2 ($P=0.001$); 1 vs. 3 ($P<0.001$); 1 vs. 4 ($P<0.001$); 2 vs. 4 ($P<0.001$); 3 vs. 4 ($P<0.001$); MT: 1 vs. 2 ($P=0.003$); 1 vs. 3 ($P=0.006$); 1 vs. 4 ($P<0.001$); 2 vs. 4 ($P=0.001$); 3 vs. 4 ($P<0.001$)
	HIT	8.4	2.6	10.0	2.6	10.3	2.4	11.7	2.8				
Peak torque knee extension 60°/s (N/m)	MT	113.4	38.9	119.8	40.3	125.5	47.3	133.1	52.2	0.233	0.874	0.007	HIT: 1 vs. 2 ($P=0.006$); 3 vs. 4 ($P=0.006$); MT: 1 vs. 2 ($P=0.04$)
	HIT	127.6	42.6	136.7	41.7	149.3	133.4	165.6	140.8				
Peak torque knee flexion 60°/s (N/m)	MT	53.2	18.2	57.8	22.1	56.0	20.2	58.3	22.4	2.741	0.047	0.079	HIT: 1 vs. 4 ($P=0.002$); 3 vs. 4 ($P=0.001$); MT: 1 vs. 2 ($P=0.012$)
	HIT	57.8	18.4	61.8	16.4	58.6	17.6	68.1	17.9				
Peak torque knee extension 270°/s (N/m)	MT	74.7	25.8	85.6	30.0	84.6	30.7	86.6	29.1	0.819	0.487	0.025	HIT: 1 vs. 2 ($P<0.001$); 1 vs. 4 ($P<0.001$); 3 vs. 4 ($P=0.011$); MT: 1 vs. 2 ($P<0.001$); 1 vs. 3 ($P=0.016$); 1 vs. 4 ($P=0.001$)
	HIT	83.0	28.3	94.2	29.8	90.2	29.8	98.2	25.9				
Peak torque knee flexion 270°/s (N/m)	MT	46.4	14.8	53.3	16.6	50.9	16.0	54.0	17.7	1.016	0.389	0.031	HIT: 1 vs. 2 ($P=0.003$); 1 vs. 4 ($P<0.001$); 3 vs. 4 ($P=0.002$); MT: 1 vs. 2 ($P<0.001$); 1 vs. 3 ($P=0.024$); 1 vs. 4 ($P<0.001$)
	HIT	53.8	12.0	59.2	13.8	57.0	13.1	63.0	13.1				

HIT, high-intensity training; MT, moderate training; SD, standard deviation; VO$_2$max, maximum oxygen uptake.

periods (12 and 16 weeks) improved cardiorespiratory fitness, but only after 7 weeks of detraining, it returned to baseline values; and isokinetic strength increased using HIT or MT in the first and second training periods.

Interestingly, the first 12 weeks of training of both programmes (MT and HIT) did not lower blood pressure. However, after the retraining period (16 weeks), the mean daytime and awake SBP and mean daytime, awake and sleep DBP decreased significantly in HIT programme. In this way, the training programme duration seems to be a key factor to obtain a consistent benefit in blood pressure values. A previous review suggests that at least 3 months of HIT or MT are required to improve blood pressure parameters, and these effects are only visible in participants not taking antihypertensive medication [18]. This fact could explain our results because all the participants of the

present study were hypertensive and they were treated with antihypertensive pharmacological therapy during the study. Therefore, our data suggest that only 3 months of MT or HIT were not enough duration to lower blood pressure, but a second retraining period of 16 weeks of HIT effectively improved SBP and DBP. Thus, the training programmes proposed to decrease blood pressure must include high intensity training and a longer duration, and they should be repeated along the time in a longitudinal way and with retraining periods, specifically in patients with pharmacological antihypertensive therapy.

Postexercise oxygen consumption and energy expenditure are factors that affect the lipid profile and body composition. It would seem that HIT promotes both higher postexercise oxygen consumption and energy expenditure than low-intensity exercise [19]. Moreover, HIT inhibits energy intake to a greater extent than low-intensity exercise [20]. In addition, HIT produces a greater mobilization of lipids than a low-intensity training programme and increases the potential of skeletal muscle to utilize lipids [21]. Therefore, evidence suggests that HIT has the potential to chronically influence lipid balance and body composition, and it would explain the data reported in the present study. In this way, our results showed a significant decrease in body mass, BMI, fat mass and waist circumference after the second 16 weeks of the HIT programme. MT did not produce any change. On the other hand, we found that the retraining period using HIT decreased total cholesterol, LDL and triglycerides. Furthermore, HDL increased significantly after the training (12 weeks) and retraining (16 weeks) periods using HIT or MT, but these changes were inconsistent and the values relapsed to baseline during the 7 weeks of detraining. Thus, when the training target is focused on the optimization of body composition and/or lipid profile, the programme must use HIT for at least 3 months duration.

With regard to fasting blood glucose variables, the first training period (12 weeks) did not improve these outcomes. However, after the retraining phase (16 weeks), a significant decrease of fasting blood glucose was observed in both groups (HIT and MT). Previous studies [22,23] found an improvement in this cardiovascular risk factor after 12 weeks of MT or HIT. However, a previous review [18] suggests that HIT studies of less than 12-weeks' duration did not show a change in fasting blood glucose and studies of at least 12-weeks' duration were inconsistent. According to our results, both retraining programmes were valid to decrease fasting blood glucose to values <100 mg/dl but shorter duration programmes (i.e. the first training period of both programmes) did not produce any change. Therefore, duration seems to be a key factor to optimize glucose metabolism outcomes in this population.

Regarding cardiorespiratory fitness, previous studies have found an inverse association between cardiorespiratory fitness and blood pressure [24]. In addition, several cross-sectional studies in the adult population suggest that cardiovascular fitness is inversely associated with the metabolic syndrome [25,26]. In this way, we found that both MT and HIT programmes and both training periods (12 and 16 weeks) improved cardiorespiratory fitness. In addition, both HIT and MT programmes improved second ventilatory threshold in the first training period and in the second

retraining period but HIT produced greater improvements. High intensity training stimulus has been established to regulate mitochondrial biogenesis and exercise tolerance [27]. In addition, HIT increases enzyme activity leads to improved muscle buffering of hydrogen ions [28]. This adaptation improves anaerobic metabolism and tolerance of exercise [29] and could explain the main findings of the present study regarding second ventilatory threshold. Furthermore, the improvement in VO_2max is linked to the improvement of maximal stroke volume, cardiac output (Q) and peripheral factors (i.e. higher capillarization, improvement in muscle buffering or increases in activities of metabolic enzymes) [30]. However, in the present study Q and peripheral factors were not assessed to corroborate this hypothesis. Nevertheless, previous research has reported that the main responses of the cardiovascular system to moderate or high-intensity circuit training, the same type of training applied in the present study, are a significant increase in VO_2max, with a concomitant improvement in maximal stroke volume and cardiac output [31]. These adaptations could help to explain the results obtained in the present study. In the same way, as this parameter is directly linked to the participant's body mass, the body mass loss observed in the present study (specifically in HIT group) would also affect cardiorespiratory fitness and explain, at least in part, the findings obtained. Furthermore, one of the findings of the present study was that only 7 weeks of detraining returned VO_2max and second ventilatory threshold adaptations to baseline values. This fact could be explained by the previously reported decrement in oxygen delivery to the muscle after training cessation due to a rapid decrease in Q_{max} associated with an important drop in maximal stroke volume, which was partly compensated by an increase in the maximal heart rate [32]. In addition, a rapid decrease in blood volume after the first days of training cessation could play a key role in the decrease in Q_{max}. Moreover, the decrease of av-DO_2max (likely due to the decrease in mitochondrial density and the reduction in muscle blood flow or capillary transit time) could also explain the decrease of VO_2max [33]. Therefore, both types of training programmes are effective at improving cardiorespiratory fitness in hypertensive patients, but training cessation periods must be avoided because some training adaptations rapidly return to basal values.

Another factor improved by both training programmes is strength, which is inversely linked with the prevalence of metabolic syndrome [34]. One of the main physiological explanations of this improvement in strength may be due to an increase in muscle mass; however, no change in fat free mass was observed along the study. Therefore, participants will be able to further increase their strength in this type of training with 3–4 months of duration through neural adaptations (i.e. inter- and intramuscular coordination and increased recruitment of motor units), regardless of changes in body composition or structural and morphological adaptations (e.g. increase in the size and number of myofibrils or changes in fibre type, muscle architecture and myofilament density) [35]. Furthermore, our data showed no return of strength values to baseline during the 7 weeks of detraining. This finding is in accordance with previous studies that have analysed the effect of resistance and/or endurance

training in some populations (i.e. older [36] or endurance athletes [37]). Therefore, strength adaptations are consistent and relatively permanent after the cessation of training.

Several strengths and limitations of this study warrant discussion. One of the strengths is the practical application of the results to the real field, the duration of the study and the compressive picture given by all the variables analysed and the clinical relevance of the results in the use of exercise programmes as adjuvant therapy to chronic pathologies related to cardiovascular risk factors. Moreover, we acknowledge some limitations that must be taken into consideration in data interpretation. First, an observational study design was used in the present study and no control group was included in the research. Another limitation was the small number of participants that took part in the research study. On the other hand, future studies would corroborate the results obtained in the present study using a randomized controlled trial design. In addition, longer studies to analyse the chronic effects of the different training programmes must be developed. Accordingly, to achieve a more comprehensive picture, future studies should include a better quality of design and analyse the effect of interventions of longer duration. Additionally, future studies should identify the mechanism involved in the improvements in cardiometabolic markers after HIT or MT. From a practical application point of view, if physicians or patients want to obtain greater improvements in cardiometabolic markers, the programme should include three sessions per week of HIT of 60 min duration and with a longer duration (>4 months). However, the effects of detraining or exercise discontinuity negatively affect some training adaptations and some of them rapidly return to basal values. Therefore, one of the training programme targets is training continuity.

In conclusion, the data of the present study demonstrated the effectiveness of the inclusion of MT or HIT programmes as adjuvant therapy in hypertensive patients. Only HIT is effective at decreasing blood pressure and losing body mass and fat, but only 12 weeks is not enough time. Also, the HIT programme generates greater decrements. Moreover, both MT and HIT improve the lipid profile and cardiorespiratory fitness, but only after 7 weeks of detraining, these variables return to baseline values.

ACKNOWLEDGEMENTS

Conflicts of interest

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