

Long-Term Impact of Physical Activity Levels After High-Speed Resistance Training on Cardiac Autonomic Control in Independent Older Adults

Sage Open Aging
Volume 11: 1–11
© The Author(s) 2025
DOI: 10.1177/30495334251369446
journals.sagepub.com/home/ggm


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Abstract

This study investigated the influence of physical activity (PA) levels on the long-term retention of the effects on cardiac autonomic control, assessed by heart rate variability (HRV), following a 16-week high-speed resistance training (HSRT) program over a 12-month follow-up period. At 12-month follow-up period, 36 participants who completed the measurements were categorized into light activity group (LAG) and moderate-to-vigorous activity group (MVAG) based on their PA levels. HRV data were recorded over a 6-min period. Significant within-group differences were observed over time. In MVAG, systolic blood pressure was significantly lower at the 6-month follow-up compared to pre-intervention ($d_{\text{unb}} = -0.87$), while in LAG, diastolic blood pressure was lower at the 12-month follow-up compared to post-intervention ($d_{\text{unb}} = -0.66$). Additionally, MVAG demonstrated significantly higher mean RR values at the 12-month compared to the 6-month follow-up ($d_{\text{unb}} = 0.49$). Moreover, minimum and mean heart rate values were significantly higher at the 6-month compared to the 12-month follow-up ($d_{\text{unb}} = -0.39$ and $d_{\text{unb}} = -0.42$, respectively) in MVAG. Lastly, Sample Entropy was significantly higher in LAG compared to MVAG at 12-month follow-up ($d_{\text{unb}} = -0.89$). In conclusion, participants who maintained moderate-to-vigorous PA during the 12-month follow-up demonstrated better retention of parasympathetic-related indices effects, as well as overall autonomic nervous system markers.

Keywords

aging, physical activity, exercise, heart rate variability, cardiovascular system

Received: April 15, 2025; revised: June 26, 2025; accepted: July 29, 2025

Introduction

Cardiovascular diseases (CVD), including heart failure and stroke, remain the leading cause of mortality worldwide (Roth et al., 2020). According to Kannel (2000), major risk factors for CVD include increased blood pressure, glucose intolerance, dyslipidaemia, abdominal obesity, and left ventricular hypertrophy, factors that persist as key contributors to CVD risk (Paluch et al., 2024). Additionally, aging has been linked to a heightened risk of CVD (Díez-Villanueva et al., 2022), largely due to macrovascular and microvascular impairments (Nowak et al., 2018).

Heart rate variability (HRV), a non-invasive method to assess the autonomic nervous system (ANS) function (Shaffer & Ginsberg, 2017; Task Force of the European

Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), has been linked to CVD risk factors (Fang et al., 2020). It also exhibits dynamic balance between the sympathetic and parasympathetic branches of the ANS, as assessed by variations in beat-to-beat intervals through both linear and non-linear analytical methods (Shaffer & Ginsberg, 2017).

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Impairments in ANS regulation have been associated with adverse health outcomes in older adults, including functional decline (Ogliari et al., 2015), and sarcopenia (Freitas et al., 2018).

The *American Heart Association* (AHA) has also emphasized physical inactivity as a well-established risk factor for CVD (Paluch et al., 2024), urging greater focus on promoting physical activity (PA) through both aerobic and resistance training (RT) interventions (Lloyd-Jones et al., 2022). Data from a 20-year longitudinal study (Hyde et al., 2021) reveals that individuals engaging in aerobic programs are more likely to participate in RT (43.5%) compared to those who do not participate in aerobic programs (3.6%). These findings, together with results from a recent study by Cancela-Carral et al. (2025), underscore the pivotal role of maintaining high levels of PA, not only as a means to support participation in multimodal exercise routines like RT, but also as a comprehensive strategy for improving cardiovascular health, preserving functional independence, extending healthy lifespan, and enhancing overall quality of life in aging populations (Izquierdo et al., 2025; Lloyd-Jones et al., 2022).

Structured exercise programs have demonstrated effectiveness in mitigating age-related declines in ANS function, particularly in parasympathetic-related indices (Bhati et al., 2019; Grässler et al., 2021). However, in older populations, interruptions or discontinuation of community-based exercise programs are often unavoidable due to health, logistical, or seasonal factors (Douda et al., 2015). These disruptions may lead to a regression or complete loss of previously achieved autonomic benefits (Dias Reis et al., 2017; Douda et al., 2015; Heffernan et al., 2009). Despite this, research has largely overlooked PA behaviors following structured exercise programs, instead focusing on the detraining effects on cardiac autonomic control in various populations, including middle-aged and older adults (Ammar et al., 2021; Rodrigues et al., 2021), individuals with heart failure (Adamopoulos et al., 1995), cancer survivors (Dias Reis et al., 2017), young adults (Gamelin et al., 2007; Heffernan et al., 2007, 2009), and obese individuals (Park et al., 2019).

While these studies have provided valuable insights into detraining-induced changes in HRV, they may fall short in addressing an ethical challenge, specifically the lack of encouragement for participants to maintain PA levels or initiate new exercise programs after the exercise program cessation (Esmonde, 2023). Particularly, findings from Soares-Miranda et al. (2014) underscore that older adults should maintain at least modest PA levels as they age, highlighting the critical role of sustained lifestyle changes in promoting long-term cardiovascular and autonomic health. Therefore, to address this gap, the present study analyzed the influence of PA levels on the long-term retention of the effects on cardiac autonomic control, assessed by HRV, following a 16-week high-speed resistance training (HSRT) program over a 12-month follow-up period.

Materials and Methods

Study Design

This exploratory and longitudinal study belongs to the “*Active Aging*” research project, previously registered on *clinicaltrials.gov* (ID: NCT05586087). The project was applied in accordance with the Declaration of Helsinki and received ethical approval from the University of Evora Ethics Committee (clearance number: 22030). All participants were thoroughly informed of the study’s objectives, potential benefits, and risks, providing written informed consent before participation.

The present study exclusively included participants from the intervention group (IG) since participants from the control group (CG) were placed on a waiting list at pre-intervention in order to start new research projects after the 16-week intervention, making them ineligible for inclusion in this analysis. The results of the clinical trial design comparing the IG and CG over the 16-week HSRT program was published elsewhere.

The research committee overseeing the study did not approve the use of the term *detraining*, which traditionally involves a restriction on participants’ PA over a given period. Instead, the committee proposed encouraging IG participants to maintain their PA or start new exercise programs over the 12-month follow-up period.

Participants

Following the intervention phase, a total of 37 older adults were available for the 12-month follow-up. However, one participant was lost to follow-up due to loss of contact, resulting in a final sample of 36 participants (mean age: 69.33 ± 3.12 years) included in the present analysis.

Based on their PA levels at 12-month follow-up, assessed using the *International Physical Activity Questionnaire-Short Form* (IPAQ-SF), participants were categorized into two groups: a light activity group (LAG; $N=20$, mean age: 70.00 ± 3.66 years) and a moderate-to-vigorous activity group (MVAG; $N=16$, mean age: 68.50 ± 2.09 years). Additionally, attrition was minimal (2.7%) and evenly distributed, with no evidence of differential dropout that could bias the results.

To be included in the study, participants had to meet the following inclusion criteria: (a) be at least 65 years old; (b) be able to walk independently; and (c) be capable of performing daily living tasks. Lastly, exclusion criteria included a diagnosis of diabetes or cardiac disease, recent surgery within the past 6 months, or an active oncological condition.

Procedures

All assessments were scheduled in the morning between 8:30 a.m. and 10:30 a.m. Participants were instructed to arrive in a fast state, with an empty bladder, and have refrained from exercise, alcohol, and coffee consumption

in the preceding 24 hr. The assessment order was as follows: anthropometric, blood pressure, and HRV. A single researcher performed all measurements in a consistent sequence for each participant.

Measurements

Heart Rate Variability. The study adhered to the international guidelines for HRV measurements (Shaffer & Ginsberg, 2017; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The participants underwent 10-min dorsal decubitus stabilization before a 6-min recording assessment.

Sinus-origin heartbeat interval variation was gaged via a Polar® H10 HR chest strap connected to the Elite HRV® app (version 5.5.5, Elite® HRV Inc., Asheville, USA) for recording and computation of HRV indices (Speer et al., 2020). The raw RR data, which were exported in a .txt file, were analyzed using the Kubios HRV software (version 3.5.0, University of Kuopio, Finland). Files were corrected for ectopic beats and artifacts using medium-level artifact correction (Moya-Ramon et al., 2022), with detailed algorithm descriptions in the Kubios software manual (Lipponen & Tarvainen, 2019).

For the time domain, following indices were computed: (i) mean RR intervals (mean RR) in milliseconds (ms); (ii) standard deviation of RR intervals (SDNN) in ms; (iii) root mean square of successive RR interval differences (RMSSD) in ms; (iv) percentage of successive RR intervals differing by >50 ms (pNN50); and (v) the stress index, representing the square root of Baevsky's stress index (Baevsky & Berseneva, 2008).

For the frequency domain, the computed indices included: (i) LF, the absolute power of the low-frequency band (0.04–0.15 Hz) in ms^2 ; (ii) HF, the absolute power of the high-frequency band (0.15–0.4 Hz) in ms^2 ; (iii) the LF/HF ratio; and (iv) total power (TP) in ms^2 .

Lastly, the non-linear domain indices were: (i) SD1, the Poincaré plot standard deviation perpendicular to the line of identity in ms; (ii) SD2, the Poincaré plot standard deviation along the line of identity in ms; (iii) sample entropy (SampEn), indicating the probability of finding specific patterns in a short time series; and (iv) detrended fluctuation analysis (DFA α_1), which describes short-term fluctuations.

Anthropometric and Blood Pressure. Anthropometric assessments included measuring weight and height by an electronic scale (TANITA®, MC 780MA, Amsterdam, Netherlands) and a stadiometer (SECA® 220, Hamburg, Germany), respectively. Body mass index (BMI) was subsequently calculated via the standard formula: $\text{BMI} = \text{body mass (kg)} / \text{height}^2 (\text{m}^2)$.

Blood pressure was assessed using the Omron HEM-907 (Omron Healthcare Co. Ltd., Kyoto, Japan), with appropriately sized automated cuffs on the left arm. Two

measurements, taken 1 min apart, were averaged after a 5-min rest period to ensure participant tranquility. The device provided readings for systolic blood pressure (SBP) and diastolic blood pressure (DBP). Afterward, pulse pressure (PP) was calculated by subtracting the DBP values from the SBP values.

Physical Activity. Participants' PA levels were assessed using the IPAQ-SF (Craig et al., 2003; Grimm et al., 2012). This questionnaire gathered data on several metrics, including the number of activity days and minutes per week, as well as total PA, walking, moderate, and vigorous activity levels expressed in MET-minutes/week. The sitting time during weekdays and weekends were also recorded.

Based on the participants' answers, they were then categorized into light, moderate, or vigorous activity levels (Supplemental Table A in the Supplemental File). The data were analyzed using a scoring spreadsheet developed by Cheng (2016). Lastly, Supplemental Table B in the Supplemental File summarizes the PA reported by participants at the 12-month follow-up.

High-Speed Resistance Training Protocol

The HSRT program was performed three times weekly for 16 weeks under supervision. The prescription was adjusted every 2 weeks. Each session consisted of an initial warm-up (10–15 min), the main HSRT exercises (45–55 min), and a concluding cool-down phase (5–10 min). The main phase included the following upper- and lower-body exercises: squats on smith machine or with dumbbells (depending on each participant's ability); leg press, leg extension; calf raise; seated row; peck fly; lat pull down; and incline bench press (Technogym, SPA, Cesena, Italy).

This present training protocol employs progressively increasing loads, tailored to the participants' mean concentric phase velocity for each set across all exercises (B. Mann, 2016; J. B. Mann et al., 2015): 1st to 4th weeks, an average speed over 1.3 m/s was required (*starting strength*); from the 5th to 10th weeks, speeds were adjusted to between 1.3 and 1.0 m/s (*speed/strength*); and in the 11th to 16th weeks, speeds ranged from 1.0 to 0.75 m/s (*strength/speed*). The mean concentric phase velocity for each set and exercise was monitored using a BEAST™ sensor (Beast Technologies, Brescia, Italy) (Vallejo et al., 2020). This device provided real-time feedback on instantaneous velocity to both participants and supervisors and displayed the mean velocity at the end of each set. Each session used six accelerometers connected via Bluetooth to six separate cell phones. Participants were also actively encouraged to execute each repetition swiftly and explosively, while maintaining a controlled pace of 2 to 3 s during the eccentric phase.

The intensity of the exercises was evaluated using the rating of perceived exertion (RPE) scale, developed by

Borg (1982), and heart rate monitoring (Polar M200, Polar Electro Ltd). The training period passed without any adverse events, and participant attendance was diligently recorded.

Statistical Analysis

Prior to the study, a sample size calculation was performed via G-power software (University of Dusseldorf, Germany; Faul et al., 2009) for F tests through ANOVA, repeated measured, within factors: $f=0.25$, α error probability=.05, power ($1-\beta$ err prob)=.80, number of groups=1, and number of measurements=4. The actual power output indicated that this clinical trial should include at least 24 participants to achieve an 82% chance of successfully rejecting the null hypothesis. All statistical analyses were conducted using SPSS for Windows, version 26 (IBM Corp., Armonk, NY, USA), with the significance level set at $p \leq .050$ (two-tailed). To complement traditional null hypothesis significance testing, an estimation approach was employed (Cumming & Calin-Jageman, 2017; Ho et al., 2019).

To evaluate the time effect across the four measurement points for each outcome, repeated measures ANOVA were applied. Pairwise comparisons between time points and groups were conducted using the Bonferroni post-hoc test. The effect sizes (ESs) were then calculated according to Cohen (1988), using Cohen's $d_{unbiased}$ (d_{unb}) through a specific spreadsheet (Cumming & Calin-Jageman, 2017). While the ESs for ANOVA were expressed as partial eta-squared values (η_p^2) and interpreted using the following thresholds: 0.010 to 0.059 (small), 0.060 to 0.140 (medium), and greater than 0.140 (large), the pairwise comparisons' ESs, expressed as d_{unb} , were categorized as: less than 0.20 (trivial), 0.20 to 0.49 (small), 0.50 to 0.80 (medium), and greater than 0.80 (large). Finally, graphical representations were created using the RStudio software.

Results

Participants

The participants' general characteristics included in the present study are shown in the Supplemental File as Supplemental Table C. Several significant differences were observed in age, weight, and BMI over the study period.

Heart Rate Variability

Table 1 shows the changes in HRV indices across the four measurement points. Among these, only DFA $\alpha 1$ demonstrated a significant time effect, with a moderate ES, throughout the study period.

Table 1 also exhibits the interaction effects within and between groups. In MVAG, the SBP was significantly higher at pre-intervention compared to the

6-month follow-up ($p=.014$, $d_{unb}=-0.87$ [-1.43 to -0.38]), while in LAG, the DBP was significantly higher at post-intervention than 12-month follow-up ($p=.037$, $d_{unb}=-0.66$ [-1.14 to -0.22]). Moreover, Min and Mean HR values were significantly higher at the 6-month compared to the 12-month follow-up ($p=.045$, $d_{unb}=-0.39$ [-0.80 to -0.02] and $p=.037$, $d_{unb}=-0.42$ [-0.83 to -0.03], respectively) in the MVAG. For Max HR, the LAG exhibited significantly lower values than the MVAG at pre- ($p=.002$, $d_{unb}=1.09$ [0.41–1.83]) and post-intervention ($p=.021$, $d_{unb}=0.79$ [0.12–1.49]). In addition, the MVAG demonstrated significantly higher Mean RR values at the 12-month compared to the 6-month follow-up ($p=.043$, $d_{unb}=0.49$ [0.01–1.01]).

Furthermore, SampEn was significantly higher in the LAG compared to the MVAG at pre-intervention ($p=.003$, $d_{unb}=-1.03$ [-1.75 to -0.35]) and 12-month follow-up ($p=.010$, $d_{unb}=-0.89$ [-1.61 to -0.22]). Lastly, the LAG also showed significantly lower DFA $\alpha 1$ values compared to the MVAG at 6-month follow-up ($p=.025$, $d_{unb}=0.77$ [0.09–1.46]). Variations in HRV indices over the study period are further illustrated in Supplemental Figure S1 of the Supplemental File.

Discussion

This study investigated whether different levels of PA (light vs. moderate-to-vigorous activity) influenced the effects on cardiac autonomic control, assessed by HRV, following a 16-week HSRT program over a 12-month follow-up period. The main study's findings were: (i) MVAG demonstrated better values in parasympathetic-related indices, including RMSSD, HF (ms^2), and SD1; (ii) the MVAG also exhibited higher values in indices representing overall ANS function, such as SDNN, TP, and SD2; (iii) the LAG showed poorer results for stress index and SampEn at 12-month follow-up period; and (iv) higher PA levels appear to play an important role to blood pressure regulation. In summary, the ANS modulation achieved during the intervention was mostly and gradually reversed over the follow-up among LAG participants.

Unlike previous studies on HRV detraining effects after structured exercise programs, including RT (Heffernan et al., 2007, 2009), aerobic (Adamopoulos et al., 1995; Ammar et al., 2021; Gamelin et al., 2007; Park et al., 2019), combined (Dias Reis et al., 2017), respiratory (Rodrigues et al., 2021), and yoga (Uthiravelu et al., 2023), the present study encouraged participants to maintain or increase PA levels post-intervention. This approach shifts the focus from detraining effects to importance of sustained lifestyle habits for long-term cardiovascular and ANS health (Soares-Miranda et al., 2014).

The study findings revealed that MVAG participants exhibited higher values in parasympathetic-related indices, including RMSSD, pNN50%, HF (ms^2), and SD1. In contrast, the LAG participants showed declines in these indices over time (Table 1). Although no significant

Table 1. Changes in Heart Rate Variability Indices Over the Study Period.

Measures	Groups	Intervention		Follow-up		M3 - 12-month	Time effect	Interaction effect within groups	Interaction effect between groups
		M0 - Pre	M1 - Post	M2 - 6-month	M3 - 12-month				
General									
SBP (mmHg)	LAG	134.50 ± 18.24	130.00 ± 11.04	128.10 ± 17.96	128.05 ± 13.25	F=4.988	F=0.564	F=1.349	
	MVAG ^a	134.38 ± 11.46	124.13 ± 13.85	121.25 ± 16.75	123.38 ± 16.82	p=.003	p=.640	p=.254	
DBP (mmHg)	LAG ^b	79.85 ± 10.86	82.65 ± 8.92	77.90 ± 10.94	77.15 ± 7.09	$\eta^2=0.128^*$	$\eta^2=0.016^{\#}$	$\eta^2=0.038^{\#}$	
	MVAG	82.38 ± 10.24	77.81 ± 8.29	77.00 ± 7.72	76.00 ± 8.63	F=3.444	F=1.650	F=0.217	
PP (mmHg)	LAG	54.65 ± 13.99	47.35 ± 7.14	50.20 ± 10.57	50.90 ± 10.65	p=.020	p=.182	p=.644	
	MVAG	52.00 ± 10.67	46.31 ± 10.45	44.25 ± 11.40	47.38 ± 14.25	$\eta^2=0.092^*$	$\eta^2=0.046^{\#}$	$\eta^2=0.006$	
Mean HR (bpm)	LAG	61.25 ± 11.63	58.60 ± 6.75	60.15 ± 6.72	60.65 ± 6.54	F=3.204 [*]	F=0.381 [*]	F=1.791	
	MVAG ^c	66.38 ± 11.11	62.75 ± 7.91	63.38 ± 8.33	60.19 ± 6.04	p=.037	p=.721	p=.190	
Min HR (bpm)	LAG	55.70 ± 7.85	54.70 ± 7.09	55.85 ± 6.15	56.70 ± 6.59	$\eta^2=0.086^*$	$\eta^2=0.011^{\#}$	$\eta^2=0.050^{\#}$	
	MVAG ^c	60.75 ± 11.08	57.31 ± 8.31	58.44 ± 8.94	55.38 ± 5.28	F=2.126 [*]	F=1.328 [*]	F=2.021	
Max HR (bpm)	LAG	64.35 ± 7.01 [†]	63.40 ± 6.69 [†]	66.00 ± 7.83	66.10 ± 7.48	p=.143	p=.268	p=.164	
	MVAG	76.13 ± 13.68	69.56 ± 8.64	71.00 ± 7.76	67.56 ± 8.66	$\eta^2=0.059^{\#}$	$\eta^2=0.038^{\#}$	$\eta^2=0.056^{\#}$	
Time-domain									
Mean RR (ms)	LAG	1004.20 ± 146.13	1036.30 ± 125.75	1009.95 ± 111.96	999.75 ± 105.32	F=1.575 [*]	F=1.623 [*]	F=2.271	
	MVAG ^c	928.00 ± 161.82	970.44 ± 109.88	944.19 ± 142.83	1005.94 ± 91.85	p=.216	p=.206	p=.141	
SDNN (ms)	LAG	28.63 ± 26.24	27.55 ± 14.67	24.84 ± 8.55	21.45 ± 8.16	$\eta^2=0.044^{\#}$	$\eta^2=0.046^{\#}$	$\eta^2=0.063^*$	
	MVAG	38.26 ± 42.66	35.79 ± 52.58	36.91 ± 58.78	36.41 ± 41.63	F=0.600 [*]	F=0.372 [*]	F=1.049	
RMSSD (ms)	LAG	30.65 ± 20.59	32.96 ± 22.58	28.13 ± 12.28	22.82 ± 8.33	p=.551	p=.690	p=.313	
	MVAG	31.25 ± 17.21	26.68 ± 20.09	24.54 ± 18.13	34.05 ± 31.80	$\eta^2=0.017^{\#}$	$\eta^2=0.011^{\#}$	$\eta^2=0.030^{\#}$	
pNN50 (%)	LAG	7.50 ± 15.46	9.09 ± 12.32	5.55 ± 6.99	4.35 ± 4.70	F=0.573 [*]	F=2.145 [*]	F=0.011	
	MVAG	9.74 ± 19.18	9.14 ± 22.49	3.64 ± 6.37	5.61 ± 10.23	p=.590	p=.117	p=.918	
Stress index	LAG	14.75 ± 5.74	13.57 ± 5.86	13.98 ± 4.45	16.51 ± 5.18	$\eta^2=0.017^{\#}$	$\eta^2=0.059^{\#}$	$\eta^2=0.001$	
	MVAG	12.08 ± 5.25	16.00 ± 8.62	16.05 ± 7.35	13.72 ± 5.50	F=2.298 [*]	F=0.328 [*]	F=0.013	
Frequency domain	LAG	294.50 ± 282.01	316.05 ± 266.23	321.90 ± 275.57	225.40 ± 225.98	p=.101	p=.747	p=.909	
	MVAG	323.56 ± 280.65	292.13 ± 267.02	333.38 ± 352.09	354.63 ± 301.11	$\eta^2=0.063^*$	$\eta^2=0.010^{\#}$	$\eta^2=0.001$	
LF (ms ²)	LAG	48.02 ± 15.19	48.33 ± 18.20	53.65 ± 20.66	53.89 ± 17.26	F=0.985	F=3.277	F=0.026	
	MVAG	54.62 ± 19.34	53.26 ± 26.73	61.28 ± 18.51	56.95 ± 19.10	p=.403	p=.024	p=.873	
LF (nu)	LAG					$\eta^2=0.028^{\#}$	$\eta^2=0.088^*$	$\eta^2=0.001$	
	MVAG					F=0.169	F=0.755	F=0.289	
					p=.917	p=.522	p=.595		
					$\eta^2=0.005$	$\eta^2=0.022^{\#}$	$\eta^2=0.008$		
					F=1.693	F=0.163	F=1.284		
					p=.173	p=.921	p=.265		
					$\eta^2=0.047^{\#}$	$\eta^2=0.005$	$\eta^2=0.036^{\#}$		

(continued)

Table 1. (continued)

Measures	Groups	Intervention		Follow-up		Time effect	Interaction effect within groups	Interaction effect between groups
		M0 - Pre	M1 - Post	M2 - 6-month	M3 - 12-month			
HF (ms ²)	LAG	221.35 ± 195.42	307.85 ± 264.07	259.40 ± 220.83	165.30 ± 124.58	F=0.763*	F=2.030*	F=0.945
	MVAG	269.38 ± 256.00	327.44 ± 334.37	244.44 ± 307.09	390.50 ± 512.09	p=.495 η ² =0.022#	p=.127 η ² =0.056#	p=.338 η ² =0.027#
HF (nu)	LAG	51.93 ± 15.18	51.57 ± 18.11	46.26 ± 20.58	46.04 ± 17.16	F=1.686	F=0.167	F=1.294
	MVAG	45.22 ± 19.10	46.69 ± 26.71	38.67 ± 18.50	43.01 ± 19.07	p=.175 η ² =0.047#	p=.918 η ² =0.005	p=.263 η ² =0.037#
Ratio LF/HF	LAG	1.14 ± 0.85	1.25 ± 1.01	1.61 ± 1.39	1.46 ± 1.05	F=1.243	F=0.389	F=2.319
	MVAG	1.59 ± 1.06	2.02 ± 1.91	1.95 ± 1.23	1.83 ± 1.31	p=.298 η ² =0.035#	p=.761 η ² =0.011#	p=.137 η ² =0.064*
TP (ms ²)	LAG	408.15 ± 273.00	541.90 ± 364.53	630.45 ± 424.52	449.60 ± 344.81	F=2.050*	F=2.305*	F=0.932
	MVAG	495.06 ± 355.19	530.19 ± 358.95	618.00 ± 635.04	824.88 ± 785.39	p=.125 η ² =0.057#	p=.095 η ² =0.064*	p=.341 η ² =0.027#
Non-linear domain								
SDI (ms)	LAG	23.16 ± 20.15	24.46 ± 16.09	20.54 ± 9.45	16.11 ± 5.89	F=0.631*	F=2.021*	F=0.001
	MVAG	22.37 ± 12.06	19.47 ± 15.73	18.24 ± 13.43	24.11 ± 22.53	p=.547 η ² =0.018#	p=.136 η ² =0.056#	p=.996 η ² =0.001
SD2 (ms)	LAG	29.55 ± 18.24	29.73 ± 14.60	28.61 ± 10.47	25.37 ± 10.79	F=0.272*	F=0.412*	F=1.780
	MVAG	35.17 ± 18.85	31.66 ± 17.98	32.13 ± 18.07	33.88 ± 16.98	p=.768 η ² =0.008	p=.669 η ² =0.012#	p=.191 η ² =0.050#
SampEn	LAG	1.80 ± 0.31†	1.72 ± 0.30	1.69 ± 0.31	1.82 ± 0.21†	F=0.560	F=1.887	F=7.527
	MVAG	1.43 ± 0.40	1.59 ± 0.34	1.54 ± 0.37	1.54 ± 0.38	p=.623 η ² =0.016#	p=.137 η ² =0.053#	p=.010 η ² =0.181§
DFA α1	LAG	0.85 ± 0.16	0.84 ± 0.29	0.89 ± 0.25†	0.95 ± 0.25	F=3.546	F=1.164	F=2.689
	MVAG	0.89 ± 0.30	0.99 ± 0.37	1.10 ± 0.26	1.02 ± 0.26	p=.017 η ² =0.094*	p=.327 η ² =0.033#	p=.110 η ² =0.073*

Note. Significant differences between periods are highlighted in bold (p ≤ .050). LAG=light activity group; MVAG = moderate-to-vigorous activity group; SBP = systolic blood pressure; DBP = diastolic blood pressure; PP = pulse pressure; mmHg = millimeter of mercury; HR = heart rate; bpm = beats per minute; ms = milliseconds; SDNN = standard deviation of RR; RMSSD; root mean square of successive RR interval differences; pNIN50 = percentage of successive RR intervals differing by >50ms; LF = low frequency; HF = high frequency; nu = normalized units; TP = total power; SampEn = sample entropy; DFA = detrended fluctuation analysis.

†Greenhouse-Geisser correction.

‡Significant differences.

§Pre-intervention versus 6-month follow-up.

¶Post-intervention versus 12-month follow-up.

‡Six-month follow-up versus 12-month follow-up.

†Between groups at that assessment point.

η² values thresholds:

#Small effect: 0.010 to 0.059;

*Medium effect: 0.060 to 0.140;

§Large effect: >0.140.

differences were observed between groups or time points, LAG demonstrated a 31% downward trend in RMSSD (*medium ES*), while the MVAG revealed a 28% upward trend (*small ES*) from post-intervention to 12-month follow-up. Similar patterns were observed for pNN50, HF (ms^2), and SD2. Prior studies also reported declines in RMSSD after intervention cessation. For instance, a 6-week RT intervention in young men followed by 4-week detraining period showed RMSSD reductions (Heffernan et al., 2009). Similarly, Adamopoulos et al. (1995) found significant RMSSD and pNN50 reductions during an 8-week detraining period after an aerobic intervention in chronic heart failure patients. These findings align with earlier studies highlighting parasympathetic declines post-intervention (Ammar et al., 2021; Dias Reis et al., 2017; Gamelin et al., 2007; Heffernan et al., 2009; Uthiravelu et al., 2023).

A plausible explanation for the decline in vagal activity observed in the LAG may involve a gradual reduction in baroreflex homeostasis over time (Selig et al., 2004). Additionally, possible vascular adaptations induced by the 16-week HSRT, such as improved forearm blood flow (Selig et al., 2004) and increased nitric oxide bioavailability (Macedo et al., 2016), might have reverted to pre-intervention levels, particularly among participants who did not engage in regular PA (seven participants). These findings underscore the role of sustained PA as a protective factor against ANS imbalance, supporting parasympathetic activity and mitigating the age-related decline previously observed in this population (Soares-Miranda et al., 2014). Nonetheless, future studies are needed to confirm such hypothetical adaptations.

SDNN is a well-established indicator of overall ANS function (Shaffer & Ginsberg, 2017; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). In short-term resting assessments, its primary source of variation is parasympathetically mediated respiratory sinus arrhythmia (Shaffer et al., 2014). In the present study, opposite patterns were observed for both groups (Table 1). The LAG exhibited a 22% decrease from post-intervention to the 12-month follow-up (*small ES*), whereas the MVAG preserved their post-intervention values within an acceptable range. This retention aligns with a previous research following the cessation of RT (Heffernan et al., 2007). Additionally, after the HSRT program, the LAG exhibited a 17% reduction in TP values (*small ES*) at the 12-month follow-up, while the MVAG presented a 56% increase (*small ES*). A *moderate ES* for the interaction between groups supports these results, aligning with findings from previous studies with shorter follow-up periods (Adamopoulos et al., 1995; Uthiravelu et al., 2023). As TP reflects the group dynamics of overall ANS function (Shaffer & Ginsberg, 2017), its increase may be attributed to a rise in HF component. The sympathetic activation typically induces tachycardia and a pronounced reduction in TP, whereas vagal activation leads in the opposite effect

(Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Regarding sympathetic activity, DFA α_1 stabilized near a value of one in both groups after HSRT (Table 1). Despite a significant group difference at the 6-month follow-up, with higher values observed in the MVAG compared to the LAG (*medium ES*), both groups displayed higher values at the 12-month follow-up than pre-intervention. This finding is relevant, as values near one (indicative of pink noise), reflect an optimal balance that helps biological systems avoiding excessive order or chaos (Stergiou, 2020). On the other hand, a loss of fractal complexity compromises control systems and reduces adaptability (Harrison & Stergiou, 2015).

Similarly, the complexity measure SampEn exhibited significantly higher values in the LAG at the 12-month follow-up (*large ES*). According to Stergiou (2020), higher SampEn values suggest more random or chaotic behavior. Even though the significant differences at baseline could have influenced the results, the observed changes warrant attention by exercise and health professionals. Previously, two studies on young populations (Heffernan et al., 2007, 2009) reported similar patterns, with post-intervention values returning to baseline. Potential mechanisms (i.e., multiple attractors) could have contributed to these results, such as the baroreflex was reset to a new pressure operating range after HSRT (Tatro et al., 1992), thus augmenting cardiac vagal outflow and the releasing of high doses of atropine can reduce heart complexity (Porta et al., 2007). However, limited research has examined post-intervention patterns of DFA α_1 and SampEn. Given their promising potential as indicators of physiological adaptations (Stergiou, 2020), future studies should incorporate them to deepen our understanding.

Finally, the blood pressure results demonstrated beneficial reductions in both groups. Specifically, the MVAG showed a significant decrease in SBP from pre-intervention to the 6-month follow-up (*large ES*), with a cumulative 8% reduction by 12 months. Both groups also experienced meaningful reductions in PP, with declines of 6% in LAG and 8% in MVAG across the study period (M0–M3). These findings align with Cancela-Carral et al. (2025), who reported that for older adults with hypertension, 60 min of aerobic or RT twice a week is adequate, with RT providing greater advantages. The observed improvements may be attributed to reduced arterial stiffness, heightening the clinical relevance of PA habits in mitigating cardiovascular risk factors and reducing the likelihood of cardiovascular events in this population (Kannel, 2000).

Nonetheless, some limitations of this study should be acknowledged. First, although a priori power analysis indicated that a sample of 24 participants would be sufficient to detect meaningful effects with 82% power, the final sample sizes in subgroup analyses may limit the generalizability of the findings and increase the risk of

Type II errors. Second, although the 6-min HRV recording duration followed established international HRV standards, it may not fully capture diurnal variations or provide the depth information available from long-term recordings. Future studies should consider using 24-hr Holter monitoring to more comprehensively assess ANS modulation across different times of day and activity contexts. Third, the assessor's awareness of the study objectives could have introduced bias due to expectancy effects. Second, while participants were instructed to maintain normal breathing during the assessments, the number of breathing cycles was not recorded. Lastly, PA levels were assessed using a self-reported, indirect tool, which may have introduced inaccuracies in estimating individual PA behavior.

Conclusion

This study highlights that maintaining at least moderate levels of PA (i.e., an active lifestyle) following a HSRT program is important for sustaining the HRV benefits achieved in older adults. Specifically, participants in the MVAG demonstrated better retention of parasympathetic-related indices, including RMSSD, HF (ms²), and SD1, and overall ANS markers, such as SDNN and TP. Furthermore, the results suggest that non-linear HRV indices such as DFA $\alpha 1$ and SampEn may offer valuable insights into autonomic adaptability and should be explored further in future research.

Overall, these findings reinforce the key role of sustained PA in promoting long-term cardiovascular and autonomic health in older adults. Encouraging active lifestyles beyond structured programs may be key to maximizing and maintaining physiological adaptations during aging.

Acknowledgments

We would like to express our sincere gratitude to all the participants for their willingness to take part in this study. We also extend our heartfelt thanks to the dedicated research staff for their invaluable contributions and support throughout the research process. Lastly, the authors would like to pay a public tribute of deep honor to *Mr. Ribeiro†*, a dedicated employee of the University's sports center, who tragically and unexpectedly passed away recently due to a car accident. His unwavering support was invaluable throughout the pre-intervention, data collection, and intervention phase. Among his many contributions, *Mr. Ribeiro†* ensured the facilities were always accessible, even outside regular hours. He meticulously cleaned the spaces, painted the gym room before the intervention began, and regularly inspected and replaced the cables on the weight machines as needed. His commitment and efforts were deeply appreciated, and he will be greatly missed.

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Ethical Considerations

The University of Evora approved all study procedures with clearance number 22030. All study procedures were followed in a compliance with the Declaration of Helsinki and according to the CONSORT (Consolidated Standards of Reporting Trials) guidelines. This clinical trial was registered on clinical-trial.gov (ID: NCT05586087).

Consent to Participate

All participants were informed about the study's aims, potential benefits and risks and gave their written informed consent to be enrolled in the study.

Consent for Publication

All the informed consent procedures and documents involved in the study were reviewed and approved by the University of Evora.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was funded by the Portuguese Foundation for Science and Technology (FCT), I.P., Grant/Award Number 2021.04598.BD (<https://doi.org/10.54499/2021.04598.BD>), and within the framework of the CHRC UID/04923. João Paulo Brito and Rafael Oliveira are research members of the Research Center in Sports Sciences, Health and Human Development which was funded by National Funds by FCT under the following project UI/04045. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author.

Supplemental Material

Supplemental material for this article is available online.

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