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Impact of caffeine on heart rate variability and fatigue indexes during recovery after endurance exercise: a crossover and double-blind study

ZAHRA RAHIMI[1], JAVAD MEHRABANI[1], SIAMAND ABDOLLAHPOUR[1], HADI NOBARI[2]

Abstract

Introduction. Caffeine increases heart rate by increasing sympathetic activity and it may also delay post-exercise cardiac recovery. Aim of Study. The present study investigates the effect of different caffeine doses on autonomic control and fatigue indexes after high-intensity endurance exercise. Material and Methods. Twelve trained males (20.7 ± 1.9 years; 72.4 ± 4.8 kg; height 1.7 ± 0.0 m; body fat 14.6 ± 2.2%; VO_2max 50.9 ± 3.3 ml/kg/min) were assigned to three trials of 6 or 9 mg/kg caffeine dose or placebo. The protocol included running on a treadmill for 12 minutes at 75% VO_2max, followed by 60 minutes of recovery. Heart rate variability (HRV) indexes such as root mean square of successive differences (RMSSD), low frequency (LF), high frequency (HF), and LF/HF were recorded initially, 30 minutes after supplementation, and 0-5, 15-20, 35-40, and 55-60 minutes post-exercise. Systolic and diastolic blood pressure (SBP and DBP) and rate pressure product (RPP), rating of perceived exertion, and perceived pain index were measured during recovery. Results. RMSSD was significantly lower during the 0-5 minutes of recovery interval in the placebo condition compared to the 6 mg/kg caffeine dose (P = 0.017). LF, HF, and LF/HF showed no significant changes compared to the placebo trial. A significant decrease in SBP was observed up to 20 minutes of the recovery period in placebo compared to two caffeine doses (P < 0.003). RPP was significantly lower up to 20 minutes of recovery in placebo compared to the 9 mg/kg caffeine dose (P < 0.002). Conclusions. It is shown that the acute effect of caffeine can delay blood pressure recovery after endurance activity but does not affect the HRV frequency domain, fatigue, and RPP indexes.

KEYWORDS: autonomic nervous system, HRV, blood pressure, recovery, caffeine.

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Corresponding author: Javad Mehrabani, mehrabanij@guilan.ac.ir

[1] Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, University of Guilan, Iran
[2] LFE Research Group, Department of Health and Human Performance, Faculty of Physical Activity and Sport Science (INEF), Universidad Politécnica de Madrid, Spain

Introduction

Caffeine is one of the most widely consumed psychoactive substances, found in everyday products such as coffee, tea, and energy drinks. A typical cup of coffee can contain up to 300 mg of caffeine. It is well-documented that caffeine exerts multiple physiological effects, primarily through the stimulation of the autonomic nervous system (ANS), which plays a central role in cardiovascular and metabolic regulation [1-4]. Caffeine primarily acts by blocking adenosine receptors, which can delay the onset of fatigue and muscle soreness, lower perceived exertion (RPE), and enhance neural activity. It also stimulates the release of catecholamines, such as epinephrine and norepinephrine, which increase heart rate, myocardial contractility, and blood pressure – potentially contributing to sympathetic overactivity and, in extreme cases, arrhythmias [3, 5]. Caffeine dosage may acutely influence cardiovascular regulation, including

baroreflex function, heart rate variability (HRV), and QT interval duration, which are clinically relevant for cardiovascular safety and exercise performance [6, 7].

HRV is a non-invasive marker commonly used to assess cardiac autonomic function during and after physical exertion [8]. It reflects fluctuations in the time intervals between heartbeats and provides insight into parasympathetic reactivation and overall autonomic modulation. HRV analysis includes time-domain indexes such as the root mean square of successive differences (RMSSD), as well as frequency-domain measures including low frequency (LF), high frequency (HF), and the LF/HF ratio. While RMSSD and HF primarily reflect parasympathetic activity, LF and LF/HF have traditionally been interpreted as indicators of sympathetic influence. However, this interpretation remains controversial, as recent studies suggest these indexes may also be influenced by baroreflex mechanisms and do not exclusively represent sympathetic tone [8].

Post-exercise declines in HRV are linked to autonomic imbalance, heightened fatigue, and delayed recovery, particularly following high-intensity or prolonged exertion. In such conditions, parasympathetic reactivation is often delayed, increasing cardiovascular strain and the risk of arrhythmia [9]. Full autonomic recovery may take up to 24 hours after strenuous activity, highlighting the importance of interventions that could modulate this recovery window.

Although caffeine has been studied extensively for its ergogenic effects, its role in post-exercise autonomic recovery remains unclear. Some studies report enhanced sympathetic activity and delayed parasympathetic return after caffeine ingestion, while others find no significant impact. Moreover, findings are inconsistent regarding the influence of different caffeine doses on endurance performance: some report benefits at high doses (>9 mg/kg), while others find no difference between high and moderate doses [10-13].

Based on existing literature suggesting delayed parasympathetic reactivation following caffeine ingestion, we hypothesized that caffeine – particularly at higher doses – would impair post-exercise autonomic recovery. Therefore, this study aimed to examine the effect of two different caffeine doses (moderate and high) on cardiac autonomic recovery after high-intensity endurance exercise, using HRV indexes as the primary outcome measures.

Aim of Study

The study aims to investigate the effects of two different doses of caffeine (moderate and high) on cardiac autonomic recovery following high-intensity endurance exercise. Specifically, it seeks to assess how caffeine influences post-exercise ANS modulation, as reflected by HRV metrics, to better understand its role in physiological stress response, parasympathetic reactivation, and potential cardiovascular risks.

Materials and Methods

Participants

Twelve trained men (20.7 ± 1.9 years; 72.4 ± 4.8 kg; height 1.7 ± 0.0 m; body fat 14.6 ± 2.2%; VO_2max 50.9 ± 3.3 ml/kg/min) participated in three supplementary groups of 6 mg/kg, 9 mg/kg caffeine dose, and placebo with a double-masked crossover design (Table 1). All participants were endurance-trained athletes with a minimum of three years of regular aerobic training (e.g., running, cycling) at a frequency of 4-5 sessions per week. Cardiovascular diseases, metabolic disorders, smoking, hypertension, caffeine intake above 300 mg/day and multivitamin use were set as exclusion criteria. A threshold of 300 mg/day was chosen because habitual high caffeine consumption can lead to tolerance, blunting both physiological and perceptual responses to supplementation, and thereby confounding the study outcomes. Multivitamin use was also excluded because micronutrients such as magnesium, B vitamins, and antioxidants can modulate cardiovascular function, autonomic balance, and perceived fatigue, which could independently influence HRV or recovery parameters. These exclusions ensured a more homogeneous sample and reduced potential confounding effects. Participants' VO_2max values were assessed two weeks prior to the main trials using the Bruce treadmill protocol (Cosmed T150 MED, Italy), a graded exercise test

Table 1. Descriptive characteristics of study participants (n = 12)

Variable	Mean ± SD
Age (years)	20.7 ± 1.9
Height (m)	1.70 ± 0.001
Body mass (kg)	72.4 ± 4.8
Body fat (%)	14.6 ± 2.2
VO_2max (ml/kg/min)	50.9 ± 3.3
Training experience (years)	5.2 ± 1.8
Habitual caffeine*	<3 mg/kg/day

* Verified via questionnaire

with progressive increases in speed and incline every three minutes. The test was continued to volitional exhaustion, and VO_2max was estimated based on peak performance. The protocol demonstrated high reliability (ICC = 0.96) in this sample. The study was approved by the Ethical Committee of the Sports Science Research Institute (SSRI) under the protocol number IR.SSRI.REC.1400.1230, and registered at the Iranian Registry of Clinical Trials (IRCT20190720044283N4). The study was conducted in the Exercise Physiology Laboratory of the University of Guilan, Rasht, Iran. All participants signed written informed consent in accordance with the Declaration of Helsinki.

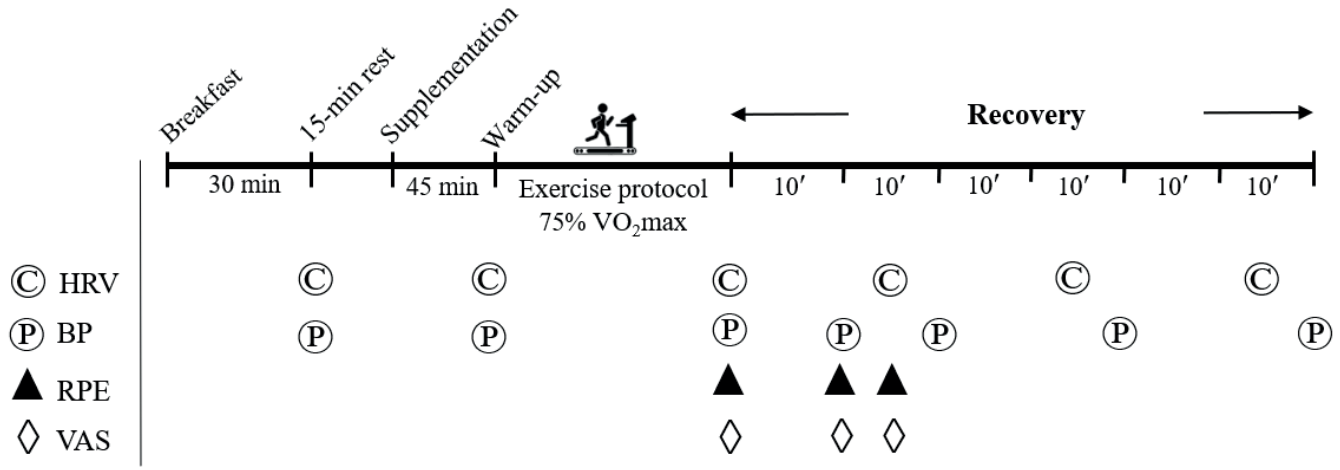
Supplementation

The caffeine capsules (containing either 6 mg/kg or 9 mg/kg of caffeine) and the placebo capsules were identical in appearance, odor, and weight, and were administered in a double-blind fashion. Capsules were prepared weekly based on individual body mass (e.g., 434 mg for 72.4 kg at 6 mg/kg). Placebo contained microcrystalline cellulose matched by weight. Participants consumed the capsules 60 minutes before the test protocol based on pharmacokinetic data indicating that peak plasma caffeine levels typically occur between 30 to 60 minutes post-ingestion [10]. The caffeine used was in anhydrous powder form encapsulated in gelatin capsules. To control for habitual caffeine consumption, participants who reported a daily intake exceeding 3 mg/kg/day were excluded. This relative threshold was based on prior literature suggesting that intakes above 3 mg/kg/day may induce tolerance and blunt the ergogenic and autonomic effects of acute caffeine supplementation, rather than a universally accepted physiological cutoff [10, 12]. Additionally, participants completed a caffeine consumption questionnaire and were classified as low-to-moderate habitual users. A list of common dietary caffeine sources was provided, and participants were instructed to completely avoid all caffeine-containing products for at least 72 hours before each session. A 72-hour washout period was implemented, consistent with average half-life of caffeine and previous HRV research protocols, to minimize residual effects.

Experimental design

Participants visited the laboratory on three different days, separated by at least 72 hours at the same time. This interval was selected to allow for sufficient recovery and to minimize carryover effects, while also ensuring participant availability and consistency with prior crossover study protocols in sports science. Participants consumed a standardized light meal (~375 kcal; 60% carbohydrates, 20% protein, 20% fat) one and a half hours before testing to ensure consistent pre-exercise metabolic conditions without affecting caffeine absorption. Timing was based on typical caffeine pharmacokinetics and standard pre-exercise feeding guidelines. Participants were instructed not to engage in strenuous exercise 72 hours before visits and tests (Figure 1). After 15 minutes of rest, baseline heart rate (HR), HRV indexes, systolic and diastolic blood pressure (SBP and DBP) were recorded in the sitting position. They immediately consumed 6 mg/kg, 9 mg/kg caffeine, or placebo capsules with 300 ml of water 60 minutes before the test protocol. HRV, SBP, and DBP were recorded 45 minutes after supplementation.

The protocol included running on a treadmill (Cosme T150 MED, Italy) at 75-80% VO_2max for 12 minutes



HRV – heart rate variability, BP – blood pressure, RPE – rate of perceived exertion, VAS – visual analog scale

Figure 1. Research desig

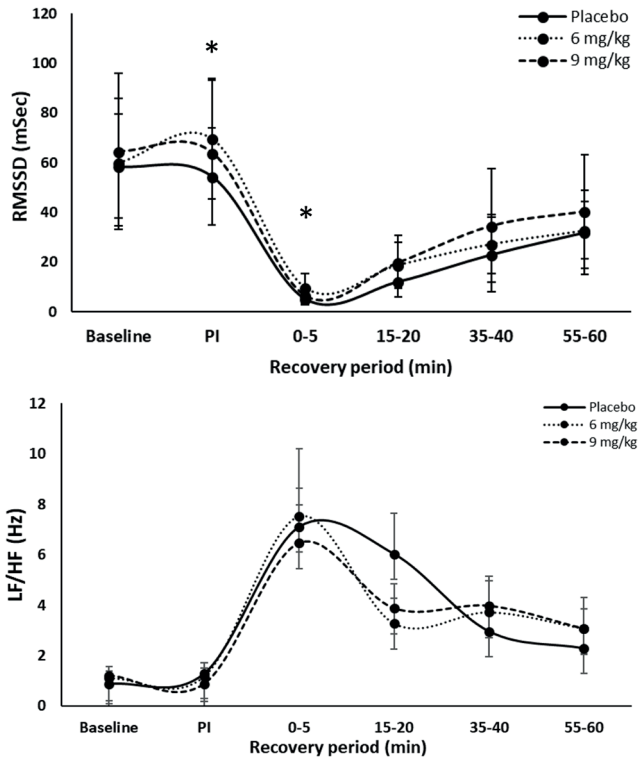
without inclination. HR, SBP, and DBP were measured immediately, and 1, 2, 3, 5, 10, 20, 40, and 60 minutes post-exercise. HRV was measured during four recovery intervals in 0-5, 15-20, 35-40, and 55-60 minutes.
Participants described the activity rate of perceived exertion on the identical 6-20 Borg scale (RPE). According to the visual analog scale (VAS), the perceived pain index (PPI) was recorded immediately after exercise and in the 1, 2, 3, 5, 10, and 15-minute recovery periods. RPE and PPI were assessed immediately after training in the 1, 2, 3, 5, 10, and 15-minute recovery periods.

Heart rate variability
Standard protocols for HRV assessment and reporting were followed [14]. A short-term recording of 5 minutes was conducted for each HRV measurement (baseline and post-exercise). This duration is considered the standard for short-term HRV assessments and has shown high reliability for variables [15]. The baseline heart rate values were determined after a 15-minute rest period using a HR monitor (Polar H10, Kempele, Finland) for 5-minute intervals during rest and recovery by recording with a smartphone app (Elite HRV, Version 4.7). The data were analyzed using Kubios HRV software (Kubios, Ver 2.2, Kuopio, Finland), which applied a filter to correct artifacts by cubic spline interpolation.
HRV measurements were taken before (baseline) and after exercise (post-exercise) in a controlled environment with a stable temperature and humidity while participants were seated. All measurements were done on the same day of the week and at the same time for exercise sessions.
The analysis covered time-domain measures (mean HR, R-R intervals, RMSSD) and frequency-domain metrics (LF/HF ratio). HR was measured at predefined intervals rather than continuously, as the focus of the study was on HRV and recovery trends, and not real-time exercise dynamics. According to frequency analysis, HRV can be divided into high frequency (HF; 0.15 ± 0.4 Hz) and low frequency (LF; 0.04 ± 0.15 Hz). The HFnu component is a marker of parasympathetic nervous system activity, and LFnu fluctuations have been suggested to reflect both sympathetic and parasympathetic nerve activity. However, baroreflex sensitivity has also been implicated, while the LF component divided by the HF component (LF/HF) acts as a marker of sympathovagal balance [16]. The time-domain changes were analyzed by the root mean square of successive differences of R-R intervals (RMSSD) as a marker of parasympathetic activity.

Blood pressure and rate pressure product
Baseline and post-exercise SBP were determined by a hand barometer (Zenith, ZTH-5001, Switzerland) in a seated position and in the 1, 2, 3, 5, 10, 20, 40, and 60 minutes of the recovery period. Rate pressure product (RPP) is a non-invasive measure of myocardial oxygen demand and is calculated by multiplying SBP by HR [17].

Statistical analysis
The Shapiro–Wilk test was used to verify the normality, sphericity, and homogeneity of the data variances, followed by repeated measure ANOVA analysis. Bonferroni post hoc tests were performed to determine parameter differences between stages and groups. The statistical significance level was set at 0.05. RPE and PPI were evaluated using the Friedman test and Wilcoxon post hoc test. The data were analyzed using IBM SPSS Statistics software version 22.0 (SPSS Inc., Chicago, IL, USA). Cohen's *d* effect size with 95% CI was used for variables in this study.
A formal a priori sample size calculation was not performed; however, the sample size (n = 12) is consistent with similar crossover HRV studies in trained populations [15, 18]. Future research should consider larger, powered samples to validate these findings.

Results
The results showed a significant change for RMSSD in the recovery period at each trial. There was a substantial increase in RMSSD after consuming 6 mg/kg of caffeine compared to placebo (P = 0.037, d = 0.72, 95% CI [0.10, 1.30]). Also, RMSSD decreased significantly to 0-5 minutes of recovery in a placebo trial compared to 6 mg/kg of caffeine (P = 0.017, d = 0.85, 95% CI [0.22, 1.47]). An increase in LFnu was observed immediately after exercise, although it did not reach statistical significance (P > 0.05) (Figure 2).
There was no significant difference in HFnu between trials, and no significant LF/HF changes were observed between the caffeine and placebo groups during recovery period. Although no significant differences were found in LF, HF, or LF/HF across conditions, the patterns observed suggest that acute caffeine intake did not substantially alter sympathovagal balance during recovery. These results are consistent with prior findings indicating limited sensitivity of frequency-domain HRV components to moderate exercise and supplementation in trained individuals.
Significant increases were observed in SBP after consuming two doses of caffeine compared to placebo.



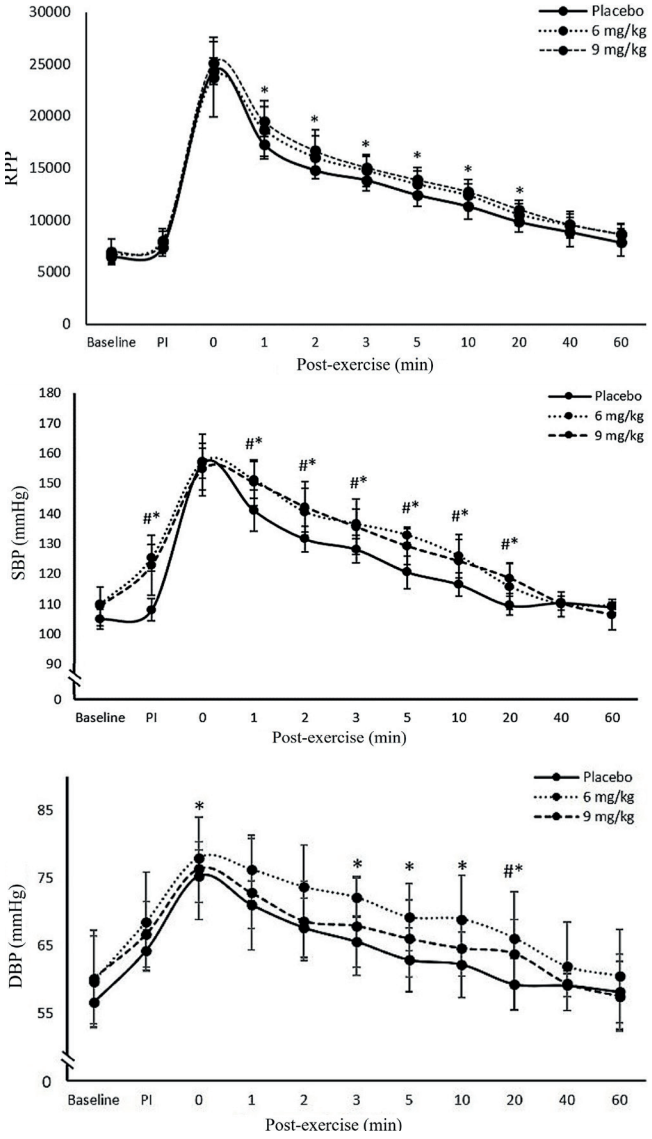
RMSSD – root mean square of successive differences, LF – low frequency, HF – high frequency

Figure 2. Post-exercise heart rate variability indices. Data are presented as mean ± SD; n = 12 participants; * indicates $p < 0.05$ 6 mg/kg caffeine compared to placebo

A significant decrease in SBP was observed immediately after exercising for up to 20 minutes during recovery period in the placebo group compared with the caffeine group ($P < 0.003$, $d = 0.91$, 95% CI [0.30, 1.54]) (Figure 3).

A significant increase in DBP was observed immediately after exercise at 6 mg/kg of caffeine compared with the placebo group. DBP was also significantly decreased in the placebo group from 3 to 20 minutes post-exercise during recovery ($P < 0.05$, $d = 0.69$, 95% CI [0.06, 1.32]). Compared with 9 mg/kg of caffeine, RPP was significantly decreased in the placebo group 1 to 20 minutes after exercise during recovery period ($P < 0.05$, $d = 0.76$, 95% CI [0.15, 1.40]).

No significant differences were found among the caffeine and placebo conditions for either RPE or PPI at any time point. Although differences were not statistically significant, both RPE and PPI scores showed a tendency to be lower in the caffeine groups during early recovery. This trend may reflect known effects of caffeine on perceived exertion and discomfort, though individual variability and sample size may have limited detection of significant differences.



RPP – rate pressure product, SBP – systolic blood pressure, DBP – diastolic blood pressure

Figure 3. Post-exercise blood pressure indices. Data are presented as mean ± SD; n = 12 participants; *# indicates $p < 0.05$ respectively 6 and 9 mg/kg caffeine compared to placebo

Discussion

The main findings show that acute consumption of 9 and 6 mg/kg of caffeine did not impair the sympathetic and parasympathetic recovery compared with placebo after high-intensity endurance exercise. This study evaluated the acute effect of 9 and 6 mg/kg of caffeine consumption on HRV and muscle soreness after 12 minutes of endurance activity with 75% VO_2max in trained men. Caffeine directly affects the cardiovascular system by, among others, increasing blood pressure

(BP), activating sympathetic nervous system and causing increased circulating catecholamines [19]. These changes are typically associated with altered HRV during rest and exercise.

HRV indexes, such as RMSSD and HF, primarily reflect parasympathetic modulation, while LF and LF/HF are commonly interpreted as indicators of sympathetic activity – though this interpretation is still debated in recent literature. In our study, RMSSD was associated with a significant increase compared to baseline after 9 and 6 mg/kg caffeine doses (Figure 2). There was no significant difference in post-activity RMSSD after 9 and 6 mg/kg of caffeine compared with the placebo group. Comparable RMSSD results were reported by Gonzaga et al. [18], which reported a significant decrease in RMSSD at the beginning of the recovery period and a substantial increase in the caffeine group compared with placebo. These results suggest that caffeine may influence post-exercise cardiovascular recovery, particularly in measures related to blood pressure, though its effects on HRV-based autonomic modulation remain limited. Sondermeijer et al. [20] also reported that RMSSD was significantly reduced by caffeine consumption. However, the subjects were inactive in their study, and the study process was performed without intervention and testing, which could explain the inconsistent results in our study.

After the exercise and up to 40 minutes after recovery, LF/HF was significantly reduced in all three placebo groups and 9 and 6 mg/g caffeine doses. These results show that LF/HF after acute doses of 9 and 6 mg/kg of caffeine is not significantly different from the placebo group, and caffeine doses did not impair parasympathetic control of heart rate recovery. Gonzaga et al. [18] reported no significant changes in HRV indexes after activity, and the results are consistent with our findings. Despite the discrepancy in the results of studies on BP responses, it has been observed that BP decreases after a session of endurance or acute resistance activity compared to baseline values. The mechanism associated with peripheral vasodilation during activity is the release of adenosine by active tissues, which leads to post-exercise hypotension. Some researchers have reported that this effect can be reduced by blocking adenosine (A2) receptors and can minimize its impact on post-exercise BP [21]. Exactly how caffeine causes these effects is a matter of debate among scientists. On the other hand, it has been suggested that the secretion of catecholamines, especially the increased secretion of epinephrine, can be an essential reason for responding to changes in the doses of caffeine [21].

In the placebo group and the dose of 6 mg/kg of caffeine, SBP showed a significant decrease compared to baseline immediately after exercise for up to 20 minutes of recovery (Figure 3). However, in the 9 mg/kg of caffeine group SBP decreased significantly until the third minute of the recovery period. In the first minute of the recovery in the placebo group, SBP showed a significant decrease compared to 6 and 9 mg/kg of caffeine. In the third and fifth minute of recovery, SBP was significantly reduced in the placebo group at 6 mg/kg. Also, SBP in the placebo group in 20 minutes of recovery significantly decreased compared to the dose of 9 mg/kg, while 20 minutes after an activity, there was no significant difference between the groups.

A significant increase in DBP was observed after supplementation in the placebo group and immediately after activity. DBP at 6 and 9 mg/kg doses decreased until the fifth and second minute of recovery. DBP in the placebo group significantly decreased compared to the dose of 6 mg/kg in the third minute of the recovery. In other stages, there was no significant difference between the groups. An et al. [22] reported no difference in SBP and DBP recovery levels after maximal activity after consuming caffeinated energy drinks with 2.5-1.5 mg/kg concentrations. The increase in BP response in caffeine-induced exercise is dose-dependent, and changes in activity intensity can change the amplitude of this response. Bunsawat et al. [7] reported that DBP remained elevated for up to 30 minutes after strenuous activity. They reported that caffeine consumption delayed parasympathetic heart rate recovery. The inconsistency of the results in this study may be caused by the difference in the intensity of the activity performed. Secretion of catecholamines during high-intensity activity can delay parasympathetic recovery. Sondermeijer et al. [20] reported that SBP and DBP increased significantly 90 minutes after caffeine consumption, whereas in the present study, no significant change was observed after caffeine consumption and before exercise. The discrepancy in the findings of the two studies may be due to the age difference between the participants and the amount of caffeine consumed. The response of vascular endothelial cells to caffeine may vary depending on the dose and age differences.

Ergogenic effects of caffeine may be mediated through mechanisms such as adenosine receptor antagonism and increased catecholamine release, which together influence cardiovascular and perceptual responses [23]. Myocardial oxygen consumption (RPP) is an indicator of myocardial function by which the amount of stress applied to the myocardium or cardiovascular recovery

can be assessed in different situations [21]. Immediately after exercise, up to 20 minutes of the recovery period, a significant decrease in RPP was observed in all three groups. However, RPP from 20 to 60 minutes of recovery was insignificant in either placebo or caffeine groups. In the first and fifth minute of the recovery period, a significant decrease in RPP was observed in the placebo group compared to the 9 mg/kg dose; therefore, in some recovery stages, the high dose of caffeine compared to placebo has delayed the recovery of RPP. These results are consistent with the results of Warnock et al. [24].

Caffeine binds to adenosine receptors, and as a result, it can reduce the RPE. The results showed that RPE was decreased in all three groups during recovery compared to immediately after exercise. There was no significant difference in RPE after acute use of 9 and 6 mg/kg of caffeine in the recovery period compared to placebo. Glaister et al. [25] reported that RPE was significantly lower in the caffeine group compared with placebo 15 seconds after each incremental stage and during recovery. Differences in the results may be due to differences in the age range and type of exercise performed. Arazi et al. [26] studied the effect of moderate doses (i.e., 5 mg/kg) on adolescent karate girls and reported that caffeine consumption reduced RPE levels compared with placebo. The difference in the results obtained may be due to differences in the sex of the subjects and the type of activity performed.

Although differences in RPE and PPI were not statistically significant, slight reductions were observed in caffeine conditions during early recovery. These trends are consistent with reported effects of caffeine on perceived exertion and discomfort and warrant further investigation with larger samples.

Caffeine increases actin and myosin function by increasing calcium flow and catecholamines and reducing pain caused by high-intensity exercise. The amount of pain in all three groups immediately post-exercise decreased, but no significant difference was observed between the groups. Gliottoni et al. [27] examined the effect of acute caffeine consumption (5 mg/kg) on muscle pain perception. They reported that caffeine consumption reduced the average feeling of muscle pain in the caffeine group. Arazi et al. [26] reported that the rate of pain after a maximum repetition of foot press and RAST test with a moderate dose of caffeine (5 mg/kg) was significantly reduced compared with placebo [26]. Inconsistent results may be due to differences in the subjects' type, activity intensity, and gender.

The small sample size (n = 12) limits statistical power and increases the possibility of type II error, meaning that subtle but potentially relevant effects of caffeine on HRV or recovery may have been overlooked. Even though habitual caffeine consumption was regulated by a relative threshold (3 mg/kg/day) and participants refrained from caffeine for 72 hours before each trial, differences in individual caffeine metabolism and tolerance may have still affected the results. Moreover, the lack of biochemical verification of compliance may have contributed to variability. Another important consideration is inter-individual variability in caffeine metabolism, largely influenced by genetic polymorphisms such as CYP1A2 and ADORA2A. These genetic factors can modulate both ergogenic and adverse responses, and their absence in our design may have contributed to inter-subject variability.

Although our results are consistent with prior research, the present study contributes by directly comparing moderate versus high caffeine doses under a standardized crossover design in trained athletes, thereby clarifying dose-response effects that have been inconsistently reported. This study helps clarify inconsistencies in the literature by controlling for habitual intake and comparing moderate and high caffeine doses under standardized conditions.

It is important to note that a caffeine dose of 9 mg/kg, although commonly used in research, exceeds typical real-world consumption levels. Such high doses have been associated with adverse effects, including gastrointestinal discomfort, sleep disturbances, anxiety, and in rare cases, arrhythmias. Therefore, while our findings provide controlled evidence regarding acute recovery responses, caution must be exercised when extrapolating them to habitual use or broader populations.

Conclusions

In conclusion, the results showed that acute consumption of 6 mg/kg and 9 mg/kg of caffeine before endurance exercise did not impair the recovery of sympathetic and parasympathetic parameters of heart rate control compared with placebo. However, caffeine consumption in moderate and high doses may delay SBP and RPP recovery, while its effects on HRV-based autonomic modulation were limited and should be interpreted cautiously. These findings are specific to young, trained male participants, and should not be generalized to other populations such as females, older adults, or untrained individuals without further research. These findings should be interpreted with caution, given the methodological limitations of the present study. Further studies involving larger, more diverse populations and

incorporating complementary autonomic measures (e.g., continuous blood pressure, catecholamines) are needed to confirm and expand upon these results.

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Conflict of Interest
The authors declare no conflict of interest.

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