International Journal of EXERCISE SCIENCE

Original Research

Effects of Low Dose Caffeine on Post-Exercise Heart Rate Variability: A Double-Blind Placebo-Controlled Trial

CAGNEY T. SARGENT*[1], TAREQ K. SHAHBAL†[1], ANDRES E. CARRILLO‡[1,2], TANIA AMORIM‡[2], JASON R. EDSALL‡[1], EMILY J. RYAN‡[3] and EDWARD J. RYAN‡[1]

[1]Department of Exercise Science, Chatham University, Pittsburgh, PA 15232, USA; [2]FAME Laboratory, Department of Physical Education and Sports Science, University of Thessaly, Trikala, GR42100, Greece; [3]Department of Exercise Physiology, West Virginia University School of Medicine, West Virginia University, Morgantown, WV 26505, USA

*Denotes undergraduate student author, †Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 15(2): 103-112, 2022. Caffeine may impact post-exercise heart rate variability (HRV); although, studies have yielded inconsistent findings. We examined the effects of low dose caffeine on post-exercise HRV. Healthy, college-aged adults [n = 18; age: 22.1 ± 2.6 years; BMI: 26.9 ± 4.3 kg/m^2; estimated maximal oxygen consumption (VO_2max): 45.1 ± 8.3 ml·kg^{-1}·min^{-1}] participated in a repeated-measures, double-blind, placebo-controlled trial. During the experimental trials, participants were fitted with a heart rate monitor and a mouthpiece with a one-way nonrebreathing valve and then rested for 10 min during baseline HRV and expired gas assessments. Participants chewed either caffeine (~170mg) or placebo gum for 5 min. Following expectoration and a 5 min warmup, participants walked on a treadmill for 20 min at 60% of estimated VO_2max and then rested for 30 min. HRV indices were calculated from 10 min measurements during baseline and post-exercise (post 1, 2, and 3). A main effect of treatment was found for standard deviation of RR intervals (SDNN), absolute power of low frequency band (LF), absolute power of high frequency band (HF), and the standard deviation perpendicular to the line-of-identity in Poincaré plot (SD1) (p < 0.05). Further, a trend for higher root mean square of successive RR interval differences (RMSSD) with caffeine was observed (p = 0.066). Post hoc t-tests revealed that post-exercise SDNN, LF, HF, and SD1 were higher with caffeine compared to placebo (p ≤ 0.012). Results demonstrated that low dose caffeine did not delay the recovery of HRV indices reflective of parasympathetic nervous system activity following an acute bout of moderate exercise.

KEY WORDS: Ergogenic aids, physical activity, autonomic nervous system, sympathetic nervous system, parasympathetic nervous system

INTRODUCTION

The opposing branches of the autonomic nervous system (ANS), the sympathetic (SNS) and parasympathetic nervous systems (PNS), continuously modulate heart rate and cardiac function. Input from these opposing branches can be assessed by analyzing the variation in time intervals between consecutive heart beats or heart rate variability (HRV). Generally, an increase in HRV reflects PNS activation while low HRV may be indicative of SNS activity (17, 28). Research has demonstrated that time-domain and frequency-domain indices of HRV may be predictive of cardiac health, disease, and mortality (2, 8, 9, 12). Further, investigators have reported that pronounced SNS and low PNS input immediately following an acute bout of exercise (low HRV post-exercise) may be comparable to that observed during a cardiac event (27, 42). While several exercise related factors, such as intensity, may impact post-exercise HRV (29), research investigating non-exercise related factors that modulate post-exercise HRV is warranted.

Caffeine is one of the most widely used drugs in the world and is available in many mediums. It has been recently reported that nearly 90% of the US population consumes caffeine with an average per capita daily intake of 186mg (13). There is no clear consensus in the literature regarding the impact of caffeine on HRV (24). Research has demonstrated that lower-to-moderate doses (≤ 6mg/kg) of caffeine increases (22, 31, 36), has no effect (33), or reduces (38) HRV at rest. Similarly, studies investigating the effects of caffeine on HRV following a bout of maximal intensity and/or exhaustive exercise have yielded inconsistent findings. Two studies reported that caffeine delayed HRV recovery after maximal intensity exhaustive exercise (4, 42). Equivocally, Clark et al. (6) reported that the consumption of an energy drink containing 140mg of caffeine had no significant impact on HRV recovery after a graded exercise test to exhaustion, while Sarshin et al. (36) reported that caffeine enhanced HRV recovery following a short bout of maximal intensity anaerobic exercise.

The effects of caffeine on post-exercise HRV following moderate exercise has received less attention. One study demonstrated that 300mg of caffeine delayed HRV recovery after 30 minutes of moderate (60%) treadmill exercise (20). In a similar study by the same group, the authors split volunteers into groups based on fitness level (high vs low) and reported that caffeine (300mg) delayed HRV recovery following 30 minutes of moderate (60%) treadmill exercise in the low fitness group only (19). It is recommended by the American College of Sports Medicine that most adults engage in moderate aerobic exercise 3-5 days/week for the development and maintenance of cardiovascular fitness (16). With this recommendation, the investigation of factors that impact HRV following moderate exercise is warranted. Further, while it is estimated that the majority of the US population uses caffeine (13), elucidating how caffeine impacts the recovery of HRV following moderate exercise may be useful for Exercise Physiologists and other Clinicians to make more informed decisions regarding exercise recommendations for the general public. To our knowledge, the effects of low dose (comparable to the US average per capita intake) caffeine intake on HRV following an acute bout of moderate exercise remains inconclusive. Thus, the purpose of the present study was to examine the effects of low dose (expectant apparent dose of 170mg) caffeine intake on HRV after a single bout of

moderate exercise. We hypothesized that low dose caffeine intake would not delay the recovery of HRV after completing the bout of moderate exercise.

METHODS

Participants

Eighteen (9 female, 9 male) healthy, college-aged, physically active adults volunteered to participate in the present study (Table 1). This study followed procedures in accordance with the ethical standards of the Helsinki Declaration. Informed consent was obtained from all volunteers and the study protocol was approved by the local Institutional Review Board for the Protection of Human Subjects. Further, this manuscript adheres to the ethical policies of the Editorial board of the International Journal of Exercise Science and guidelines detailed by Navalta et al. (32). Following informed consent, potential volunteers were screened for eligibility via a health history questionnaire. Eligible participants were free of cardiopulmonary disease, reported zero-to-moderate caffeine usage (≤ 6mg/kg consumed daily) and were not taking medication that could influence HRV (e.g. beta blockers, sympathomimetics). Further, female subjects were not taking estrogen based oral contraceptives that impact the elimination half-life of caffeine (26).

Table 1. Subject characteristics.

Sex	Age (years)	Weight (kg)	BMI (kg/m^2)	VO_2max (ml/kg/min)
Female (n = 9)	21.2 ± 1.2	80.3 ± 13.0	28.4 ± 5.3	39.8 ± 5.5
Male (n = 9)	22.9 ± 3.3	79.7 ± 6.4	25.4 ± 2.4	50.4 ± 7.2*
All Subjects (n = 18)	22.1 ± 2.6	80.0 ± 10.0	26.9 ± 4.3	45.1 ± 8.3

Values are expressed as mean ± SD. BMI: body mass index; VO_2max: estimated maximal oxygen consumption.
* Significantly different from females ($p < 0.05$).

Protocol

The present study employed a repeated-measures, double-blind, placebo-controlled design. Following informed consent and a determination of eligibility, participants reported to the laboratory on three separate occasions. During the first visit (pre-experimental testing) body mass and height were measured via a balance beam scale/stadiometer (Health o meter Professional 402KL, Pelstar LLC Designs, Countryside, IL). Thereafter, participants completed a submaximal treadmill test (walking at a constant speed at 5% grade) for the estimation of maximal oxygen consumption (VO_2max) (10).

For the next two visits (experimental trials, one-week washout), volunteers were asked to refrain from strenuous exercise and caffeine consumption for 24 hours prior to testing. Participants reported to the laboratory at the same time of day for both experimental trials and all experimental testing occurred between 0600 and 1200. Upon arrival, participants were immediately fitted with a heart rate monitor (Polar, RS800CX, Kempele, Finland) and mouthpiece that was worn during the entire experimental trial and HRV/expired air samples (Parvomedics Metabolic System, Parvomedics, Sandy, UT) were assessed for 10 minutes (baseline). Following baseline measurements, participants chewed two pieces of gum (caffeine or placebo, Military Energy Gum, Ford Gum and Machine Co., Akron, NY) for 5 minutes and

then expectorated. In one of the two experimental visits, the gum contained caffeine (100mg per piece). Kamimori et al. (23) compared the pharmacokinetic parameters of caffeine administered in gum vs. capsule across 50, 100, and 200mg doses in 84 healthy volunteers and reported that the bioavailability of caffeine in gum (dose normalized to manufacturer reported 85% dose release) and the capsule were similar while the rate of absorption was significantly faster for the gum formulation. Based on these findings we approximate that subjects in the present study consumed an expectant apparent dose of 170mg of caffeine during the caffeine trial. The caffeine and placebo gum are virtually identical in shape, color, taste, and texture and the gum was administered in a double-blind, counterbalanced manner. A researcher not associated with the study performed the blinding and counterbalancing with the gum being prepackaged into coin envelopes.

Following expectoration, the participants warmed-up on a treadmill at a slow walking pace for 5 minutes. Thereafter, the volunteers completed 20 minutes of treadmill walking at a workload corresponding to 60% (calculated) of estimated VO_2max. The target workload was accomplished by adjusting grade while keeping speed consistent with that chosen for the VO_2max estimation. The same workload was employed in both experimental trials. Upon completion of the treadmill exercise, subjects rested for 30 minutes in an upright seated position. Post-exercise HRV was assessed between 0 – 10 minutes (post 1), 10 – 20 minutes (post 2), and 20 – 30 minutes (post 3) immediately following exercise cessation. Seven HRV indices from the time, frequency, and non-linear domains were calculated for statistical analysis. HRV data were calculated as described previously (39). Briefly, recorded heart rate data were transferred from the heart rate monitors to the Polar Pro Trainer 5 software (Polar Electro, Oy, Kempele, Finland). R-R interval data were saved and subsequently used to calculate HRV indices using the Kubios HRV Standard software v. 3.3 (Kubios, Kuopio, Finland). A description of the seven HRV indices is provided in Table 2.

Table 2. Heart Rate Variability Indices.

Acronym (units)	Description	Domain
Mean RR (ms)	Mean of RR intervals	Time
SDNN (ms)	Standard deviation of RR intervals	Time
RMSSD (ms)	Root mean square of successive RR interval differences	Time
LF (ms^2)	Absolute power of low frequency band (0.04-0.15 Hz)	Frequency
HF (ms^2)	Absolute power of high frequency band (0.15-0.4 Hz)	Frequency
LF/HF	Ratio between LF and HF band powers	Frequency
SD1 (ms)	In Poincaré plot, the standard deviation perpendicular to the line-of-identity	Nonlinear

Statistical Analysis

Differences in age, weight, BMI, and VO_2max between male and female participants were determined using independent samples t-tests. HRV indices not normally distributed (RMSSD, LF, HF, LF/HF, and SD1) were log transformed prior to analysis (29). Log transformations were successful in normalizing (Shapiro-Wilk) HRV data. HR and HRV indices were analyzed using a (4 x 2 x 2) three-way, mixed model ANOVA (Time x Treatment x Sex). Post-hoc paired samples

t-tests were conducted using all participants only when a main effect of treatment was observed in the mixed model ANOVA. A Bonferroni-corrected p value was used to interpret all post-hoc paired samples *t*-tests. Effect size (d) values were calculated (Cohen's $d = (M_2 - M_1)/SD_{pooled}$) for HRV indices found to have a main effect of treatment (SDNN, LF, HF, SD1). Effect size values were interpreted as none (00-0.19), small (0.2-0.49), medium (0.5-0.79), or large (≥ 0.8) (7). Statistical analyses were conducted using SPSS (version 26) with differences considered significant at $p < 0.05$ or $p < 0.0125$ (Bonferroni-corrected).

RESULTS

Age, weight, and BMI were similar between male and female participants ($p > 0.05$). Estimated VO_2max, however, was significantly higher in men compared to women ($p = 0.001$) (Table 1).

We observed a main effect of treatment (placebo vs. caffeine) for SDNN ($F_{1,16} = 7.882$, $p = 0.013$), LF ($F_{1,16} = 7.389$, $p = 0.015$), HF ($F_{1,16} = 15.181$, $p = 0.001$), and SD1 ($F_{1,16} = 7.154$, $p = 0.017$) with a trend for a higher RMSSD with caffeine ($F_{1,16} = 3.898$, $p = 0.066$). A significant treatment x time interaction was observed for SDNN ($F_{3,14} = 3.382$, $p = 0.026$) and SD1 ($F_{3,14} = 4.868$, $p = 0.005$) with LF trending towards significance ($F_{3,14} = 2.567$, $p = 0.065$). No significant treatment x time interaction was observed for HR ($F_{3,14} = 0.544$, $p = 0.655$), Mean RR ($F_{3,14} = 0.155$, $p = 0.926$), RMSSD ($F_{3,14} = 0.752$, $p = 0.526$), HF ($F_{3,14} = 1.606$, $p = 0.200$), and LF/HF ($F_{3,14} = 0.100$, $p = 0.959$). Post-hoc tests revealed a significantly higher HF at post1 ($p = 0.006$; $d = 0.24$), post2 ($p = 0.001$; $d = 0.34$), and post3 ($p = 0.004$; $d = 0.29$) during caffeine compared to placebo. SD1 was significantly higher at post2 ($p = 0.003$; $d = 0.41$) and post3 ($p = 0.012$; $d = 0.28$) during the caffeine trial compared to placebo. SDNN ($p = 0.001$; $d = 0.59$) and LF ($p < 0.001$; $d = 0.70$) were significantly higher during caffeine compared to placebo only at post2 (Table 3). No significant differences between treatments were observed at baseline for SDNN, LF, HF, and SD1 ($p > 0.05$) (Table 3).

HR and all HRV indices (Mean RR, SDNN, RMSSD, LF, HF, LF/HF, and SD1) significantly changed over time ($F_{3,14} \geq 4.119$, $p \leq 0.011$). Specifically, HR and LF/HF were higher, whereas Mean RR, SDNN, RMSSD, LF, HF, and SD1 were lower during post-exercise compared to baseline ($p < 0.05$). We did not observe a significant main effect of sex ($F_{1,16} \leq 2.464$, $p \geq 0.136$) or a significant interaction between sex and treatment ($F_{1,16} \leq 2.404$, $p \geq 0.141$), time ($F_{3,14} \leq 0.760$, $p \geq 0.522$), or treatment*time ($F_{3,14} \leq 1.427$, $p \geq 0.246$) for HR and all HRV indices.

Table 3. Baseline and post-exercise heart rate variability.

Variable	Baseline		Post-exercise 1		Post-exercise 2		Post-exercise 3	
	Placebo	Caffeine	Placebo	Caffeine	Placebo	Caffeine	Placebo	Caffeine
HR (bpm)	75.7 ± 10.4	74.2 ± 10.3	92.4 ± 14.0	89.5 ± 12.9	81.1 ± 11.7	78.7 ± 12.0	78.8 ± 10.2	77.2 ± 11.6
Mean RR (ms)	806.5 ± 105.1	823.4 ± 108.6	663.4 ± 101.8	684.8 ± 105.2	754.6 ± 110.8	780.2 ± 121.3	773.3 ± 97.7	792.6 ± 116.1
SDNN (ms)	47.3 ± 21.6	51.0 ± 22.3	28.9 ± 10.4	34.7 ± 14.1	38.4 ± 13.5	48.4 ± 19.1*	45.1 ± 17.4	51.2 ± 17.7
RMSSD (ms)	41.0 ± 28.7	44.0 ± 25.4	22.2 ± 14.8	28.0 ± 17.3	31.7 ± 19.5	40.0 ± 24.4	36.2 ± 20.3	41.8 ± 20.5
LF (ms^2)	1490.2 ± 1487.9	1787.2 ± 1680.8	639.3 ± 397.8	1024.6 ± 835.4	948.1 ± 535.4	1587.1 ± 1141.7*	1415.0 ± 1182.0	1794.5 ± 1213.7
HF (ms^2)	830.8 ± 1465.6	1020.0 ± 1270.6	284.3 ± 418.3	391.7 ± 441.3*	528.9 ± 694.9	787.0 ± 785.6*	638.3 ± 788.8	867.3 ± 735.9*
LF/HF	3.1 ± 2.1	2.6 ± 1.7	5.5 ± 4.9	4.1 ± 2.5	5.0 ± 4.5	3.7 ± 3.3	3.9 ± 2.9	3.6 ± 3.3
SD1 (ms)	29.0 ± 20.3	31.1 ± 18.0	15.7 ± 10.5	19.8 ± 12.3	22.4 ± 13.8	28.8 ± 16.3*	25.6 ± 14.4	29.7 ± 14.4*

HR: heart rate; Mean RR: mean of RR intervals; SDNN: standard deviation of RR intervals; RMSSD: root mean square of successive RR interval differences; LF: absolute power of low frequency band (0.04-0.15 Hz); HF: absolute power of high frequency band (0.15-0.4 Hz); LF/HF: ratio between LF and HF band powers; SD1: in Poincaré plot, the standard deviation perpendicular to the line-of-identity. All values are presented as means ± SD. * significant difference between placebo and caffeine trials ($p < 0.0125$).

DISCUSSION

Our primary finding was that an expectant apparent dose of 170mg of caffeine did not delay recovery of HRV indices reflective of PNS activity following an acute bout of moderate exercise. Further, these results support our hypothesis and the contention that lower doses of caffeine may facilitate HRV recovery following moderate exercise in young, zero-to-moderate caffeine users. Our results demonstrated that four HRV indices (SDNN, LF, HF, SD1) were higher (indicating a faster recovery of HRV) during post-exercise with caffeine relative to placebo (Table 3). Further, a trend for a higher RMSSD with caffeine was observed. Effect sizes for HF (post1: $d = 0.24$; post2: $d = 0.34$; post3: $d = 0.29$) and SD1 (post1: $d = 0.35$; post2: $d = 0.41$; post3: $d = 0.28$) were small but highest values were reported during 10–20 minutes post-exercise (i.e. post2). A medium effect size was calculated for SDNN (post2: $d = 0.59$) and LF (post2: $d = 0.70$) during 10 – 20 minutes post-exercise. This suggests that the potential benefits of low dose caffeine in facilitating HRV recovery during post-exercise may become most apparent at least 10 minutes after exercise cessation. HF is representative of PNS activity (21) and the nonlinear SD1 is considered similar to the RMSSD HRV metric (5), which has been suggested as highly reflective of PNS driven changes to HRV (37). Thus, our finding that these indices were higher post-exercise with caffeine treatment supports the contention that low dose caffeine may facilitate PNS recovery following moderate exercise. We also found that SDNN and LF were higher post-exercise with caffeine. Research has demonstrated that these indices are influenced by both the SNS and PNS (34, 40), with recent evidence suggesting a stronger PNS input (9, 28).

Our results differ from Gonzaga et al. (20) who reported that caffeine delayed HRV recovery following a bout of moderate exercise comparable to the exercise used in the current study. A possible reason for the equivocal findings is that a lower dose of caffeine was utilized in the current study (170mg) compared to Gonzaga (300mg) (20). Research has demonstrated that post-exercise HRV is dependent on exercise intensity such that an increase in intensity may further delay HRV recovery (30). A similar relationship between caffeine's impact on physiological arousal and post-exercise HRV is biologically plausible. For example, higher doses of caffeine may promote more physiological arousal and SNS drive and thus delay the recovery of HRV indices reflective of PNS activity, while lower doses may not result in sufficient physiological arousal that would impact post-exercise HRV. One study demonstrated that post-exercise HRV was similar with 3 and 6mg/kg of caffeine following a short bout of maximal intensity exercise (36). But both 3 and 6mg/kg doses of caffeine may be considered moderate and, to our knowledge, the effects of low vs. moderate vs. high doses of caffeine on post-exercise HRV following a bout of moderate intensity exercise remains to be explored. It is possible that exercise intensity and the dose of caffeine synergistically influence physiological arousal and post-exercise HRV. Further, several factors including caffeine dosage, timing of administration, fitness level, time of day, habitual consumption, and genetic variability impact the physiological consequences of caffeine (3, 14, 15, 18, 19, 35) and thus should be considered when investigating caffeine's impact on post-exercise HRV.

This study has some limitations. Previous research has demonstrated that sex and menstrual cycle phase may impact autonomic function (1, 25) and hemodynamic responses to caffeine (11). We did not control for menstrual cycle phase in the present study, and our relatively small sample size may have not provided adequate statistical power to observe any sex related differences. We estimated VO_2max with a single stage treadmill test (20) and used that estimation to calculate workload. Research has shown that the validity of this estimate may decrease when approaching the limits of the specified heart rate ranges (41), and measurement of VO_2max with a graded exercise test to exhaustion while collecting expired gas samples would have likely resulted in more accurate VO_2max values. Further, studies investigating the test-retest reliability of post-exercise HRV have demonstrated inconsistent findings (29); therefore, we believe that the interpretation of post-exercise HRV should be approached with caution. Lastly, we did not assess or control for food intake immediately prior to, and sleep the night before, experimental testing sessions. While we did request that participants get adequate sleep and consume an identical breakfast prior to the first and second experimental trials, we cannot verify compliance. Thus, we cannot exclude diet and sleep as potential sources of error variance in the present study.

In conclusion, our results demonstrated that a low dose of caffeine did not delay recovery of HRV indices reflective of PNS activity following an acute bout of moderate exercise in a sample of young, healthy adults. When considering the HRV indices that demonstrated a main effect of treatment (SDNN, LF, HF, SD1), our results suggest that low dose caffeine may facilitate HRV recovery when compared to placebo. Nonetheless, interindividual variability, as evident in the data presented herein, may have contributed, at least in part, to the study outcomes. Additional research is recommended to clearly elucidate the effects of low dose caffeine on HRV assessed

following the completion of moderate exercise. Given the current exercise recommendations (16) and widespread use of caffeine throughout the US (13), further study of this topic is applicable to the health and wellbeing of the general public. We also recommend that future studies investigate factors, such as individual variability, that may modulate caffeine's impact on post-exercise HRV across a variety of exercise intensities.

ACKNOWLEDGEMENTS

We would like to thank Marketright Inc. for providing the Military Energy Gum.

REFERENCES

1. Bai X, Li J, Zhou L, Li X. Influence of the menstrual cycle on nonlinear properties of heart rate variability in young women. Am J Physiol Heart Circ Physiol 297(2): H765-774, 2009.

2. Bigger JT, Jr., Steinman RC, Rolnitzky LM, Fleiss JL, Albrecht P, Cohen RJ. Power law behavior of rr-interval variability in healthy middle-aged persons, patients with recent acute myocardial infarction, and patients with heart transplants. Circulation 93(12): 2142-2151, 1996.

3. Boyett JC, Giersch GE, Womack CJ, Saunders MJ, Hughey CA, Daley HM, Luden ND. Time of day and training status both impact the efficacy of caffeine for short duration cycling performance. Nutrients 8(10) 2016.

4. Bunsawat K, White DW, Kappus RM, Baynard T. Caffeine delays autonomic recovery following acute exercise. Eur J Prev Cardiol 22(11): 1473-1479, 2015.

5. Ciccone AB, Siedlik JA, Wecht JM, Deckert JA, Nguyen ND, Weir JP. Reminder: Rmssd and sd1 are identical heart rate variability metrics. Muscle Nerve 56(4): 674-678, 2017.

6. Clark NW, Herring CH, Goldstein ER, Stout JR, Wells AJ, Fukuda DH. Heart rate variability behavior during exercise and short-term recovery following energy drink consumption in men and women. Nutrients 12(8) 2020.

7. Cohen J. Statistical power for the behavioral sciences. 2nd ed.: Routledge; 1988.

8. Dekker JM, Crow RS, Folsom AR, Hannan PJ, Liao D, Swenne CA, Schouten EG. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: The aric study. Atherosclerosis risk in communities. Circulation 102(11): 1239-1244, 2000.

9. Dinas PC, Koutedakis Y, Flouris AD. Effects of active and passive tobacco cigarette smoking on heart rate variability. Int J Cardiol 163(2): 109-115, 2013.

10. Ebbeling CB, Ward A, Puleo EM, Widrick J, Rippe JM. Development of a single-stage submaximal treadmill walking test. Med Sci Sports Exerc 23(8): 966-973, 1991.

11. Farag NH, Vincent AS, McKey BS, Al'Absi M, Whitsett TL, Lovallo WR. Sex differences in the hemodynamic responses to mental stress: Effect of caffeine consumption. Psychophysiology 43(4): 337-343, 2006.

12. Farrell TG, Bashir Y, Cripps T, Malik M, Poloniecki J, Bennett ED, Ward DE, Camm AJ. Risk stratification for arrhythmic events in postinfarction patients based on heart rate variability, ambulatory electrocardiographic variables and the signal-averaged electrocardiogram. J Am Coll Cardiol 18(3): 687-697, 1991.

13. Fulgoni VL, 3rd, Keast DR, Lieberman HR. Trends in intake and sources of caffeine in the diets of us adults: 2001-2010. Am J Clin Nutr 101(5): 1081-1087, 2015.

14. Fulton JL, Dinas PC, Carrillo AE, Edsall JR, Ryan EJ, Ryan EJ. Impact of genetic variability on physiological responses to caffeine in humans: A systematic review. Nutrients 10(10) 2018.

15. Ganio MS, Klau JF, Casa DJ, Armstrong LE, Maresh CM. Effect of caffeine on sport-specific endurance performance: A systematic review. J Strength Cond Res 23(1): 315-324, 2009.

16. Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM, Nieman DC, Swain DP, American College of Sports M. American college of sports medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: Guidance for prescribing exercise. Med Sci Sports Exerc 43(7): 1334-1359, 2011.

17. Goldberger JJ, Arora R, Buckley U, Shivkumar K. Autonomic nervous system dysfunction: Jacc focus seminar. J Am Coll Cardiol 73(10): 1189-1206, 2019.

18. Goldstein ER, Ziegenfuss T, Kalman D, Kreider R, Campbell B, Wilborn C, Taylor L, Willoughby D, Stout J, Graves BS, Wildman R, Ivy JL, Spano M, Smith AE, Antonio J. International society of sports nutrition position stand: Caffeine and performance. J Int Soc Sports Nutr 7(1): 5, 2010.

19. Gonzaga LA, Vanderlei LCM, Gomes RL, Garner DM, Valenti VE. Involvement of cardiorespiratory capacity on the acute effects of caffeine on autonomic recovery. Medicina (Kaunas) 55(5) 2019.

20. Gonzaga LA, Vanderlei LCM, Gomes RL, Valenti VE. Caffeine affects autonomic control of heart rate and blood pressure recovery after aerobic exercise in young adults: A crossover study. Sci Rep 7(1): 14091, 2017.

21. Grossman P, Taylor EW. Toward understanding respiratory sinus arrhythmia: Relations to cardiac vagal tone, evolution and biobehavioral functions. Biol Psychol 74(2): 263-285, 2007.

22. Hibino G, Moritani T, Kawada T, Fushiki T. Caffeine enhances modulation of parasympathetic nerve activity in humans: Quantification using power spectral analysis. J Nutr 127(7): 1422-1427, 1997.

23. Kamimori GH, Karyekar CS, Otterstetter R, Cox DS, Balkin TJ, Belenky GL, Eddington ND. The rate of absorption and relative bioavailability of caffeine administered in chewing gum versus capsules to normal healthy volunteers. Int J Pharm 234(1-2): 159-167, 2002.

24. Koenig J, Jarczok M, Kuhn W, Morsch K, Schafer A, Hillecke T, Thayer J. Impact of caffeine on heart rate variability: A systematic review. J Caffeine Res 3(1): 22-37, 2013.

25. Koenig J, Thayer JF. Sex differences in healthy human heart rate variability: A meta-analysis. Neurosci Biobehav Rev 64: 288-310, 2016.

26. Magkos F, Kavouras SA. Caffeine use in sports, pharmacokinetics in man, and cellular mechanisms of action. Crit Rev Food Sci Nutr 45(7-8): 535-562, 2005.

27. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation 84(2): 482-492, 1991.

28. McCorry LK. Physiology of the autonomic nervous system. Am J Pharm Educ 71(4): 78, 2007.

29. Michael S, Graham KS, Davis GMO. Cardiac autonomic responses during exercise and post-exercise recovery using heart rate variability and systolic time intervals-a review. Front Physiol 8: 301, 2017.

30. Michael S, Jay O, Halaki M, Graham K, Davis GM. Submaximal exercise intensity modulates acute post-exercise heart rate variability. Eur J Appl Physiol 116(4): 697-706, 2016.

31. Monda M, Viggiano A, Vicidomini C, Viggiano A, Iannaccone T, Tafuri D, De Luca B. Espresso coffee increases parasympathetic activity in young, healthy people. Nutr Neurosci 12(1): 43-48, 2009.

32. Navalta JW, Stone WJ, Lyons TS. Ethical issues relating to scientific discovery in exercise science. Int J Exerc Sci 12(1): 1-8, 2019.

33. Rauh R, Burkert M, Siepmann M, Mueck-Weymann M. Acute effects of caffeine on heart rate variability in habitual caffeine consumers. Clin Physiol Funct Imaging 26(3): 163-166, 2006.

34. Reyes del Paso GA, Langewitz W, Mulder LJ, van Roon A, Duschek S. The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: A review with emphasis on a reanalysis of previous studies. Psychophysiology 50(5): 477-487, 2013.

35. Ryan EJ, Kim CH, Fickes EJ, Williamson M, Muller MD, Barkley JE, Gunstad J, Glickman EL. Caffeine gum and cycling performance: A timing study. J Strength Cond Res 27(1): 259-264, 2013.

36. Sarshin A, Naderi A, da Cruz CJG, Feizolahi F, Forbes SC, Candow DG, Mohammadgholian E, Amiri M, Jafari N, Rahimi A, Alijani E, Earnest CP. The effects of varying doses of caffeine on cardiac parasympathetic reactivation following an acute bout of anaerobic exercise in recreational athletes. J Int Soc Sports Nutr 17(1): 44, 2020.

37. Shaffer F, McCraty R, Zerr CL. A healthy heart is not a metronome: An integrative review of the heart's anatomy and heart rate variability. Front Psychol 5: 1040, 2014.

38. Sondermeijer HP, van Marle AG, Kamen P, Krum H. Acute effects of caffeine on heart rate variability. Am J Cardiol 90(8): 906-907, 2002.

39. Thomas RM, Algrain HA, Ryan EJ, Popojas A, Carrigan P, Abdulrahman A, Carrillo AE. Influence of a cyp1a2 polymorphism on post-exercise heart rate variability in response to caffeine intake: A double-blind, placebo-controlled trial. Ir J Med Sci 186(2): 285-291, 2017.

40. Umetani K, Singer DH, McCraty R, Atkinson M. Twenty-four hour time domain heart rate variability and heart rate: Relations to age and gender over nine decades. J Am Coll Cardiol 31(3): 593-601, 1998.

41. Waddoups L, Wagner D, Fallon J, Heath E. Validation of a single-stage submaximal treadmill walking test. J Sports Sci 26(5): 491-497, 2008.

42. Yeragani VK, Krishnan S, Engels HJ, Gretebeck R. Effects of caffeine on linear and nonlinear measures of heart rate variability before and after exercise. Depress Anxiety 21(3): 130-134, 2005.