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Cardiac autonomic dynamics during and after soleus push-up exercise in healthy young adults: a single-group repeated-measures study

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Abstract

Background

The soleus push-up is a seated exercise designed to activate the soleus muscle with little cardiorespiratory demand. Its acute cardiac autonomic effects and post-exercise recovery pattern remain unclear.

Methods

Thirty-two healthy young adults completed five minutes of seated soleus push-up exercise after a five-minute seated baseline, followed by twenty minutes of seated recovery. Electrocardiographic activity and respiratory frequency were recorded continuously. Time-domain, frequency-domain, and nonlinear heart rate variability measures were evaluated across baseline, exercise, and four consecutive five-minute recovery periods. Repeated-measures analyses and a respiratory-adjusted linear mixed-effects model were used.

Results

Exercise intensity averaged 11.64 ± 5.01% of heart rate reserve, and median perceived exertion was 3. Heart rate and respiratory frequency increased during exercise, while time-domain, frequency-domain, and nonlinear heart rate variability measures decreased. During the first five minutes of recovery, vagally related time-domain and nonlinear measures rose above baseline, indicating rapid parasympathetic rebound. Frequency-domain power returned to baseline, whereas low-frequency power increased later in recovery. Adjustment for respiratory frequency removed the time effect on high-frequency power. No sex difference was detected at individual time points, although recovery trajectories differed by sex.

Conclusions

A single soleus push-up session was associated with transient vagal withdrawal during exercise, rapid parasympathetic rebound during early recovery, and delayed low-frequency heart rate variability changes. Soleus push-up may therefore represent a very-light-intensity exercise model characterized by low physiological demand and rapid cardiovagal recovery.

Clinical trial number:

not applicable.

Background

In modern society, technological advancements and the rise of desk-based occupations have dramatically increased sedentary time, creating a major global public health challenge [1, 2]. Although the protective effects of regular exercise on cardiovascular [3] and autonomic [4] health are well established, barriers such as lack of time [5], insufficient motivation [6], and reduced functional capacity [7] often limit participation in conventional exercise programs. Consequently, there is growing interest in metabolically sustainable, low-intensity exercise strategies that can be easily incorporated into sedentary lifestyles. One such approach is ‘exercise snacks’, defined as short, intense 1-to-3-minute bouts of physical activity performed intermittently throughout the day, providing a time-efficient alternative to traditional prolonged exercise [8, 9, 10]. In contrast to these brief, high-intensity interventions, the soleus push-up (SPU) represents a distinct strategy that enables sustained, low-intensity metabolic activity while remaining seated. The SPU consists of rhythmic heel raises performed with the forefeet maintained in contact with the floor, specifically designed to selectively activate the soleus muscle [11]. Unlike conventional exercise modalities, the SPU has been shown to provide substantial metabolic benefits while imposing only minimal cardiorespiratory demand [11].

The autonomic nervous system regulates cardiac rhythm through the coordinated actions of its sympathetic and parasympathetic branches, while heart rate variability (HRV) quantifies the beat-to-beat fluctuations resulting from this regulation and is widely recognized as a key biomarker of cardiac autonomic balance [12, 13, 14]. Current guidelines recommend HRV as a valuable tool for evaluating acute exercise responses, post-exercise recovery, and cardiac autonomic adaptations [15]. Accordingly, the acute and chronic effects of exercise on cardiac autonomic function have been extensively investigated using HRV parameters, particularly focusing on parasympathetic withdrawal during exercise and vagal reactivation throughout post-exercise recovery following aerobic, high-intensity interval, and resistance exercise [16, 17].

During exercise and post-exercise recovery, cardiac autonomic regulation is primarily governed by the coordinated interaction of two major neural pathways: central command, originating from higher motor centers, and peripheral reflexes arising from active muscles and the cardiovascular system, including the cardiopulmonary baroreflex and the exercise pressor reflex [18]. Although post-exercise autonomic recovery is influenced by several factors, including exercise intensity, duration, modality, and individual exercise capacity [19, 20], exercise intensity is consistently recognized as the primary determinant of recovery dynamics [15]. Consistent with this, studies comparing recovery responses across different exercise intensities have demonstrated that autonomic and physiological parameters return to baseline more rapidly after low-intensity exercise, whereas recovery is markedly delayed following high-intensity exercise [21, 22, 23]. Even during traditional low-intensity isometric or dynamic exercise, activation of central command together with the accumulation of local metabolites induces rapid vagal withdrawal and sympathetic activation, thereby attenuating parasympathetic reactivation and delaying the restoration of systemic autonomic balance [24, 25, 26, 27, 28].

In contrast to traditional exercise modalities, which rely heavily on central command activation and are accompanied by substantial local metabolite accumulation, the SPU selectively targets the soleus

muscle. Often referred to as the ‘second heart’ because of its extensive venous sinuses [29, 30, 31], the soleus possesses a remarkable venous pumping capacity that enhances venous return while imposing minimal fatigue and cardiovascular strain. Previous studies have demonstrated that the SPU favorably improves glucose tolerance [11, 32, 33, 34], lipid metabolism, and local oxidative metabolism [35]. However, the acute cardiac autonomic adjustments and reflex responses elicited by this unique venous pumping mechanism, as reflected by HRV, remain poorly understood.

The aim of this study was to investigate the acute cardiac autonomic responses to a standardized SPU protocol in healthy untrained individuals using HRV parameters. We hypothesized that the SPU would induce transient reductions in HRV indices associated with cardiac vagal modulation during exercise, followed by rapid post-exercise autonomic recovery despite its minimal cardiovascular and perceptual demands.

Methods

Study design

This study used a single-group repeated-measures laboratory design to characterize cardiac autonomic responses during and after a standardized soleus push-up protocol.

Participants

Sample size was estimated a priori using G*Power software (version 3.1.9.7) based on previous methodological studies in autonomic and exercise physiology [36]. Assuming a medium effect size ($f = 0.25$), a statistical power of 0.95, $\alpha = 0.05$, a correlation among repeated measures of 0.50, and a nonsphericity correction (ε) of 1.0, the required sample size for a one-group repeated-measures design with six measurements was calculated as 28 participants. To compensate for potential data loss, 37 healthy volunteers were initially recruited. Five participants were subsequently excluded because of cardiac arrhythmia (n = 1) or motion-induced ECG (Electrocardiogram) artifacts that precluded reliable HRV analysis (n = 4). Consequently, the final analyses included 32 healthy young adults (15 females, 46.9%; 17 males, 53.1%).

Table 1
Descriptive statistics of participants (n = 32)

Characteristics	
Age (years)	19.41 ± 1.05
Body mass (kg)	69.79 ± 17.61
Height (cm)	172.45 ± 11.87
BMI (kg/m^2)	23.09 ± 3.57

Values are presented as mean ± SD. BMI: body mass index.

Participants were university students aged 18–22 years with a body mass index (BMI) between 18.0 and 30.0 kg/m². Eligibility was determined through an initial health screening interview and self-report. Exclusion criteria included cardiovascular, metabolic, neurological, or psychiatric disorders affecting autonomic function, cardiac arrhythmias, regular medication use, smoking, alcohol consumption, regular participation in competitive sports or structured athletic training, vigorous exercise within 24 h before testing, acute stress, anxiety, active infection or fever, orthopedic conditions limiting SPU performance, and menstruation at the time of testing for female participants.

To ensure recording reliability, participants were instructed to fast for at least 2 h, abstain from caffeine for at least 4 h, sleep 6–8 h the night before testing, and report no bladder fullness or thirst before data collection. The study was approved by the Ethics Committee of Ankara Yıldırım Beyazıt University Faculty of Medicine (19 February 2026; Protocol No. 88). Written informed consent was obtained from all participants before testing. All procedures were conducted in accordance with the Declaration of Helsinki.

Procedures

The experimental protocol consisted of a single laboratory visit (Fig. 1). Upon arrival, participants were informed about the study procedures, provided written informed consent, and completed sociodemographic and anthropometric assessments. Following a 5-min seated preparations period for instrumentation and signal stabilization, baseline (rest) ECG and respiratory frequency (fR) were continuously recorded for 5 min. Participants then completed a 5-min SPU exercise session while ECG and fR were recorded continuously, followed by a 20-min seated recovery period with uninterrupted ECG-fR recording. Immediately after the recovery period, participants reported their rating of perceived exertion (RPE), and the experimental session was concluded.

Although the supine position has traditionally been regarded as the standard posture for short-term HRV recordings [12], previous studies have shown that it accelerates parasympathetic reactivation during

post-exercise recovery [37, 38, 39]. Consequently, recovery measurements obtained in the supine position may exaggerate parasympathetic responses and partially mask the physiological effects of exercise. Because the SPU was performed in a seated position and the recovery period was recorded immediately after exercise without any postural transition, all HRV measurements in the present study were obtained with participants remaining seated.

All recordings were performed in a quiet, temperature-controlled (22–23°C), dimly lit physiology laboratory (Faculty of Medicine, Ankara Yıldırım Beyazıt University). To minimize the influence of circadian variation on cardiac autonomic function, all testing sessions were conducted between 10:00 and 13:00.

ECG and Respiratory Recordings

Continuous ECG and fR were recorded simultaneously using a 4-channel PowerLab 26T data acquisition system (ADInstruments, Australia) connected to a Windows laptop and using LabChart 8 software (ADInstruments, Australia), following the methodology described by Soni et al. [40]. After seated stabilization and preparations period, continuous data acquisition was initiated and maintained throughout the 5-min baseline, 5-min SPU exercise, and 20-min recovery periods. Cardiac activity was recorded using a modified torso Lead II configuration.

Respiratory activity was monitored using a Respiratory Belt Transducer (TN1132/ST, ADInstruments, Australia) positioned securely around the rib cage, as previously described by Czyżewski et al. [41]. The belt was adjusted to detect thoracic movement without restricting normal breathing. Participants were instructed to breathe spontaneously and to avoid consciously altering either their breathing rate or tidal volume throughout the experimental protocol.

HRV and fR Analysis

HRV indices were derived offline from continuous ECG recordings sampled at 1000 Hz (1-ms resolution) using the LabChart 8 HRV Pro module (version 2.0.3, ADInstruments, Australia), following the procedures described by Samanta et al. [42] and Sreenath et al. [43]. Analyses were performed over six consecutive 5-min periods comprising baseline, SPU exercise, and four post-exercise recovery intervals (0–5, 5–10, 10–15, and 15–20 min). R waves were detected automatically and subsequently verified by manual inspection. Ectopic beats identified during manual inspection were corrected by interpolation using adjacent normal-to-normal intervals. Recordings containing more than 5% ectopic beats or exhibiting unreliable R-wave detection due to a low signal-to-noise ratio were excluded from the analyses.

fR was obtained automatically from the Respiratory Belt Transducer signals recorded synchronously with the ECG. fR was determined for the same six 5-min periods using the Cyclic Measurements module and Data Pad functions in LabChart 8. All automatically generated values were verified manually, and the final fR for each participant and time interval was expressed as breaths/min.

The following variables were extracted for each participant: heart rate (HR), fR, root mean square of successive differences (RMSSD), standard deviation of normal-to-normal intervals (SDNN), percentage of successive normal-to-normal intervals differing by more than 50 ms (pNN50), low-frequency power (LF), high-frequency power (HF), low-frequency to high-frequency ratio (LF/HF), total power (TP), Poincaré plot standard deviation perpendicular to the line of identity (SD1), and Poincaré plot standard deviation along the line of identity (SD2). Because several HRV variables exhibited the typical non-normal distribution, natural logarithmic (ln) transformation was applied to RMSSD, LF, HF, LF/HF, and SD1 before statistical analysis. In addition, to characterize the rapid autonomic changes occurring during early recovery, RMSSD was further analyzed in consecutive 30-s windows throughout the first 5 min post-exercise [16, 44].

For each participant, maximal heart rate (HRmax) was estimated using the age-predicted formula 220 − age [45]. Percentage heart rate reserve (%HRR), an important indicator used to quantify relative exercise intensity [46], was calculated as follows: *%HRR = [(HRexercise − HRrest) / (HRmax − HRrest)] × 100*, where HRexercise and HRrest represent the mean heart rates recorded during the SPU and baseline periods, respectively.

SPU Protocol

Participants sat upright on a chair with their shoes removed, knees flexed at 90°, and both feet resting flat on the floor. Their hands rested loosely on the thighs to avoid upper-limb assistance, and they were instructed to breathe normally throughout the protocol while avoiding the Valsalva maneuver.

Following baseline recordings, the SPU movement was explained and demonstrated, after which participants completed five familiarization trials. The 5-min SPU exercise was then initiated with continuous ECG and fR recordings. During the exercise, the forefeet remained in contact with the floor while both heels were raised simultaneously to approximately 45° and then lowered back to the starting position in a controlled manner.

Movement cadence was standardized at 120 beats/min (1 repetition/s) using the auditory cues of a digital metronome application (Pro Metronome, version 4.4.4; EUMlab Polybeat Limited, 2023). Participants performed the exercise continuously for 5 min, completing a total of 300 repetitions. Immediately after exercise, they remained seated quietly in the same position for a 20-min recovery period with uninterrupted ECG and fR recordings. Following completion of all recordings, RPE was assessed using the modified Borg 0–10 scale.

Statistical analysis

Data distribution was assessed using coefficients of variation, skewness and kurtosis values, histograms, normal Q–Q plots, and the Shapiro–Wilk and Kolmogorov–Smirnov tests. Variables violating normality assumptions were ln-transformed before analysis, when appropriate. Accordingly, the LF/HF ratio was analyzed as ln(LF/HF).

Normally distributed variables were analyzed using one-way repeated-measures analysis of variance (ANOVA). Sphericity was assessed with Mauchly's test, and Greenhouse–Geisser corrections were applied when necessary. Bonferroni-adjusted pairwise comparisons were used for post hoc analyses. Variables that remained non-normally distributed after transformation were analyzed using the Friedman test followed by Bonferroni-adjusted Wilcoxon signed-rank tests.

To determine whether fR influenced lnHF across measurement periods, a linear mixed-effects model was constructed with time as a fixed effect, participant as a random effect, and fR entered as a time-varying covariate. Alternative covariance structures were compared using the Akaike Information Criterion (AIC), and the model with the lowest AIC was retained for the final analysis.

Sex-related differences in HRV responses were examined using a mixed repeated-measures ANOVA with time as the within-subject factor and sex as the between-subject factor.

Effect sizes were reported as partial eta squared (ηp^2) for repeated-measures ANOVA and Kendall's coefficient of concordance (W) for Friedman tests.

Continuous variables are presented as mean ± standard deviation (SD), whereas non-normally distributed and ordinal variables are presented as median (interquartile range, IQR). For visualization purposes, lnRMSSD data presented in Fig. 3 and Fig. 4 are expressed as mean ± standard error of the mean (SEM). Statistical analyses were performed using IBM SPSS Statistics version 22.0, figures were generated using GraphPad Prism version 10.4.2, and statistical significance was accepted at $p < 0.05$.

Results

Changes in physiological parameters

The mean exercise intensity corresponded to 11.64 ± 5.01% HRR, whereas the median rating of perceived exertion (modified Borg 0–10 scale) was 3 (IQR = 2).

HR changed significantly across the measurement periods ($p < 0.001$, Table 2). Post hoc analyses demonstrated that HR was significantly higher during SPU than during baseline and all recovery periods (all $p < 0.001$). No significant differences were observed between baseline and any recovery period or among the recovery periods themselves (all $p > 0.05$), indicating that HR returned to resting levels immediately after exercise.

A similar pattern was observed for fR ($\chi^2(5) = 72.135$, $p < 0.001$). Pairwise comparisons showed that fR was significantly higher during SPU than during baseline and all recovery periods (all $p < 0.001$). No significant differences were found between baseline and any recovery period or among the recovery periods themselves (all $p > 0.05$), indicating that the increase in fR was confined to the exercise period.

Table 2 Physiological responses before, during, and after SPU (n = 32)

Parameters	Baseline 5 min	SPU 5 min	Post 0-5 min	Post 5-10 min	Post 10-15 min	Post 15-20 min	*p* value	Effect size
HR	80.9 ± 9.9	94.9 ± 10	80.9 ± 11	80.8 ± 11.1	80 ± 10.5	79.6 ± 10.1	<0.001	0.728
fR	16.4 (4)	25.9 (4.7)	17 (3.7)	16.9 (3.7)	17.4 (5.3)	16.5 (4.6)	<0.001	0.451

Values are presented as mean ± SD for HR and median (IQR) for fR. HR was analyzed using repeated-measures ANOVA, whereas fR was analyzed using the Friedman test. Effect size is presented as partial eta squared (ηp^2) for HR and Kendall's coefficient of concordance (W) for fR. SPU, soleus push-up; HR, heart rate (beats/min); fR, respiratory frequency (breaths/min).

Changes in heart rate variability parameters

Table 3 and Fig. 2 summarize the changes in HRV indices before, during, and after SPU.

Time-domain and nonlinear HRV indices

lnRMSSD decreased significantly during SPU compared with baseline and all recovery periods (all $p < 0.001$). During the first 5 min of recovery, lnRMSSD increased above baseline values ($p = 0.003$) before returning toward baseline, with no significant differences between baseline and the subsequent recovery periods.

A comparable pattern was observed for SDNN. SDNN was significantly lower during SPU than at baseline ($p < 0.001$), increased above baseline during the 0–5 min recovery period ($p < 0.001$), and then declined between the 0–5 and 5–10 min recovery periods ($p = 0.020$). No further differences were detected among the remaining recovery periods.

Similarly, pNN50 differed significantly across the six measurement periods ($p < 0.001$). Compared with baseline, pNN50 decreased during SPU ($p < 0.001$), increased during all recovery periods relative to SPU (all $p < 0.001$), and exceeded baseline values during the 0–5 min recovery period ($p = 0.001$). No significant differences were observed between baseline and the later recovery periods.

lnSD1 was significantly lower during SPU than during baseline and all recovery periods (all $p < 0.001$). During the 0–5 min recovery period, lnSD1 increased above baseline values ($p = 0.002$) and subsequently returned toward baseline, with no significant differences between baseline and the later recovery periods or among recovery periods.

SD2 also changed significantly over time ($p < 0.001$). SD2 was significantly lower during SPU than at all other time points (all $p < 0.001$). During the 0–5 min recovery period, SD2 exceeded both baseline ($p = 0.001$) and the 5–10 min recovery period ($p = 0.022$).

Frequency-domain HRV indices

lnLF was significantly lower during SPU than at all other time points (all $p < 0.001$). During recovery, lnLF increased progressively and exceeded baseline values at 10–15 min ($p = 0.004$) and 15–20 min ($p = 0.010$). Moreover, lnLF values at 10–15 and 15–20 min were significantly higher than those at 0–5 and 5–10 min of recovery (all $p < 0.05$).

lnHF was significantly lower during SPU than during baseline and all recovery periods (all $p < 0.001$), whereas no significant differences were found between baseline and any recovery period or among the recovery periods themselves. To determine whether this response was influenced by changes in fR, lnHF was further analyzed using a linear mixed-effects model with fR entered as a time-varying covariate. fR had a significant effect on lnHF ($F(1, 134.723) = 51.623$, $p < 0.001$), whereas the main effect of time was no longer significant after adjustment ($F(5, 45.528) = 2.206$, $p = 0.070$).

ln(LF/HF) ratio also differed significantly across the measurement periods ($p = 0.003$). Compared with baseline, ln(LF/HF) was significantly higher during SPU ($p = 0.030$) and during the 10–15 min ($p = 0.010$) and 15–20 min ($p = 0.028$) recovery periods. No other pairwise differences were identified.

TP was significantly lower during SPU than during baseline and all recovery periods ($p < 0.05$). Following exercise cessation, TP returned to baseline, with no significant differences between baseline and any recovery period or among the recovery periods.

Table 3
Changes in HRV indices before, during, and after SPU (n = 32)

Parameters	Baseline 5 min	SPU 5 min	Post 0–5 min	Post 5–10 min	Post 10–15 min	Post 15–20 min	*p* value	Effect size
SDNN	48.4 ± 12.2	36.5 ± 10.4	59.2 ± 14.9	52.3 ± 13.7	54.9 ± 13	54.6 ± 14.4	< 0.001	0.439
lnRMSSD	3.48 ± 0.37	3.13 ± 0.47	3.60 ± 0.39	3.52 ± 0.37	3.56 ± 0.34	3.58 ± 0.35	< 0.001	0.449
pNN50	11.01 (21.07)	3.27 (15.70)	15.84 (25.01)	13.48 (21.43)	14.29 (20.46)	16.65 (22.44)	< 0.001	0.256
lnSD1	3.13 ± 0.37	2.79 ± 0.47	3.25 ± 0.39	3.17 ± 0.37	3.22 ± 0.34	3.23 ± 0.35	< 0.001	0.439
SD2	63.77 ± 16.17	48.76 ± 13.46	78.03 ± 19.48	68.91 ± 18.02	72.92 ± 17.04	72.93 ± 19.34	< 0.001	0.423
lnLF	6.54 ± 0.56	5.58 ± 0.66	6.59 ± 0.58	6.70 ± 0.69	6.97 ± 0.63	6.97 ± 0.68	< 0.001	0.555
lnHF#	6.25 ± 0.79	4.84 ± 1.06	6.33 ± 0.92	6.29 ± 0.77	6.32 ± 0.80	6.34 ± 0.78	< 0.001	0.568
ln(LF/HF)	1.06 ± 0.13	1.20 ± 0.23	1.06 ± 0.17	1.08 ± 0.14	1.11 ± 0.14	1.11 ± 0.14	0.003	0.178
TP	7.60 ± 0.63	6.74 ± 0.64	7.77 ± 0.65	7.75 ± 0.66	7.89 ± 0.59	7.92 ± 0.59	< 0.001	0.532

Values are presented as mean ± SD, except for pNN50, which is presented as median (interquartile range). Effect size was expressed as partial eta squared (ηp^2) for repeated-measures ANOVA and Kendall's coefficient of concordance (W) for the Friedman test. SPU: soleus push-up, SDNN: standard deviation of normal-to-normal intervals, lnRMSSD: natural logarithm of root mean square of successive differences, pNN50: percentage of successive NN intervals differing by more than 50 ms, lnSD1: natural logarithm of poincaré plot standard deviation perpendicular to the line of identity, SD2: poincaré plot standard deviation along the line of identity, lnLF: natural logarithm of low-frequency power, lnHF: natural logarithm of high-frequency power, ln(LF/HF): natural logarithm of low-to-high frequency power ratio, TP: total power.

Although a significant time effect was observed for lnHF, this effect was no longer significant after adjustment for respiration in the linear mixed-effects model.

To further characterize the early vagal recovery response, lnRMSSD was analyzed in consecutive 30-s intervals during the first 5 min after SPU (Fig. 3). A significant effect of time was observed across the recovery epochs ($F(4.586, 142.156) = 9.141$, $p < 0.001$). Post hoc analyses showed that lnRMSSD at 30–60 s was significantly higher than at 90–120, 120–150, 150–180, 180–210, 210–240, 240–270, and

270–300 s (all $p < 0.01$). In addition, lnRMSSD during the initial 0–30 s interval was significantly higher than at 150–180 s and 180–210 s (both $p = 0.013$). No significant differences were observed among the remaining recovery intervals, indicating that the largest changes in lnRMSSD occurred during the early phase of recovery.

Figure 3 detailed time course of cardiac vagal recovery during the first 5 min following soleus push-up (SPU) exercise. lnRMSSD was calculated in consecutive 30-s recovery intervals from 0–30 s to 270–300 s after exercise cessation. The horizontal red line indicates the mean lnRMSSD value for the 0–5 min recovery period. Data are presented as mean ± SEM. *$p < 0.05$ versus the 150–180 s and 180–210 s intervals. #$p < 0.01$ versus all intervals from 90–120 s to 270–300 s.

Sex differences

Sex-specific changes in lnRMSSD are presented in Fig. 4. A significant main effect of time was observed ($F(2.680, 80.399) = 28.079$, $p < 0.001$), whereas the main effect of sex was not significant ($F(1, 30) = 0.270$, $p = 0.607$). However, a significant time × sex interaction was detected ($F(2.680, 80.399) = 4.432$, $p = 0.008$), indicating different temporal lnRMSSD responses between female and male participants. In both sexes, lnRMSSD decreased during SPU and increased during recovery (Fig. 4). Despite the significant interaction, post hoc pairwise comparisons revealed no significant sex differences at any individual time point (all $p > 0.05$).

Discussion

The present study investigated acute cardiac autonomic responses and post-exercise recovery following a single session of SPU using HRV analysis. The principal findings were that SPU induced significant vagal withdrawal, while HR and fR increased during exercise and returned to resting levels within the first 5 minutes of recovery. The protocol was well tolerated by all participants. Consistent with an RPE score of 3 and a %HRR of 11.64%, the SPU imposed only a mild physiological demand and was classified as a very light intensity exercise according to the American College of Sports Medicine [47] guidelines. To our knowledge, this is one of the first studies to characterize acute cardiac autonomic modulation and post-exercise recovery dynamics in response to a single SPU session using HRV.

Among the HRV indices, RMSSD, SDNN, pNN50, SD1, and SD2 decreased significantly during exercise but increased above baseline during the first 5 minutes of recovery. Similarly, LF, HF, and TP declined during exercise, yet all three returned to baseline within the first 5 minutes post-exercise. However, LF power exhibited a delayed autonomic response, exceeding baseline values during the 10–15 and 15–20 minute recovery intervals, whereas HF and TP did not rise significantly above resting levels throughout recovery. Likewise, the LF/HF ratio increased during exercise and remained significantly elevated above baseline during the 10–15 and 15–20 minute recovery periods.

In many studies investigating post-exercise parasympathetic reactivation, the first few minutes of recovery are often excluded from analysis because of signal artifacts or other methodological

limitations. A major methodological strength of the present study was the use of continuous beat-to-beat recordings, which enabled uninterrupted monitoring from exercise cessation through the recovery period without data loss. This approach revealed a recovery pattern that has rarely been described in acute post-exercise models: a rapid and pronounced parasympathetic reactivation exceeding baseline values occurred within the first 5 minutes after a single SPU session, with the greatest response observed during the initial 90 seconds (Fig. 3).

The recovery profile observed in the present study resembles the parasympathetic response reported following passive stretching exercises [48]. Despite this similarity, however, the physiological mechanisms involved are likely to differ. Passive interventions, which do not involve voluntary muscle contractions, provide less activation of the skeletal muscle pump and metaboreflex than active exercise and therefore generally produce relatively modest hemodynamic responses [49]. In contrast, SPU involves rhythmic, isolated contractions of the soleus muscle that activate its venous pumping function. This mechanical action of the soleus has been shown to produce measurable hemodynamic changes [50, 51].

The rapid parasympathetic reactivation observed following SPU may be explained, at least in part, by the unique hemodynamic function of the soleus muscle. During SPU, rhythmic contractions of the soleus activate its powerful venous pump, substantially increasing venous return and stroke volume through activation of the skeletal muscle pump [31]. Upon the abrupt cessation of exercise, both the skeletal muscle pump and central command are rapidly withdrawn [52]. However, cardiac output and the transient elevation in arterial blood pressure do not immediately return to resting levels [53, 54].

This transient hemodynamic state is detected by stretch-sensitive arterial baroreceptors located in the carotid sinuses and aortic arch, thereby engaging arterial baroreflex mechanisms that increase cardiac vagal outflow and produce a powerful vagal braking response at the sinoatrial node [55]. Consequently, this mechanism may contribute to the rapid restoration of vagal activity and provides a plausible physiological explanation for the marked increase in vagally mediated HRV indices observed during the first 5 min of recovery, particularly within the initial 90 s after SPU.

A distinctive histological characteristic of the soleus muscle may further explain the pronounced vagal reactivation observed following SPU. The soleus is composed predominantly of slow-twitch (Type I) muscle fibers [56, 57], which are characterized by a high oxidative capacity [58]. Consequently, the rhythmic, isolated contractions performed during SPU are unlikely to produce substantial intramuscular metabolite accumulation, such as lactate, hydrogen ions, or pyrophosphate. This limited metabolic disturbance minimizes activation of the muscle metaboreflex, which would otherwise sustain sympathetic outflow and suppress parasympathetic reactivation during recovery. In the relative absence of opposing sympathetic metaboreflex and chemoreflex activation, baroreflex-mediated vagal outflow can proceed unimpeded from the earliest moments of recovery. This distinctive autonomic profile may explain why SPU exhibits a recovery pattern that differs from those observed following traditional exercise modalities.

Collectively, we believe that localized, low-metabolic-stress exercise modalities that avoid extensive recruitment of large muscle groups may have the potential to promote rapid parasympathetic rebound or even parasympathetic overshoot beyond resting values during early recovery. Previous studies have shown that conventional low-intensity endurance exercise allows vagal indices to recover rapidly toward baseline during the early post-exercise period [59]. In contrast, the SPU not only restored vagal activity to baseline but also elicited a marked parasympathetic overshoot, with RMSSD, SDNN, and pNN50 exceeding resting values. This finding may indicate that the SPU possesses a unique capacity to enhance cardiovagal recovery. Our findings are consistent with previous evidence demonstrating that a single bout of light-intensity exercise in untrained individuals enhances post-exercise parasympathetic activity, resulting in SDNN values above baseline [60]. However, unlike that study, the present investigation demonstrated significant increases not only in SDNN but also in RMSSD and pNN50, short-term HRV indices that more specifically reflect cardiac vagal modulation [14]. Similarly, Ogata et al. [61] reported that the SD1 index exceeded baseline values during the later stages of recovery (25–30 min) following acute isometric exercise involving the upper limbs and trunk. In contrast, our findings show that both SD1 and SD2 surpassed resting values during the early phase of recovery (0–5 min), suggesting a substantially more rapid restoration of vagal activity following SPU.

Another condition characterized by rapid parasympathetic recovery following exercise is yoga. A study comparing modified yoga postures with light-intensity aerobic exercise (cycling) demonstrated that although cardiovagal withdrawal and heart rate responses during yoga were comparable to those observed during light aerobic exercise, a marked parasympathetic reactivation occurred during the recovery phase in Savasana yoga posture [62]. Likewise, a recent investigation examining the acute autonomic effects of different Suryanamaskar (Sun Salutation) paces reported that slower-paced practice promoted greater parasympathetic dominance than faster-paced protocols, highlighting the capacity of low-intensity yoga-based movements to facilitate vagal recovery [63]. While this comparison is indirect, the similarities in autonomic recovery patterns suggest that shared physiological mechanisms may underlie these responses. Similar to the postural awareness and somatosensory feedback inherent to yoga, the rhythmic low-intensity activation of the soleus muscle during SPU may provide abundant proprioceptive afferent input owing to its high muscle spindle density [64]. This continuous proprioceptive input may further facilitate autonomic regulation during the transition from exercise to recovery. Therefore, we propose that exercise modalities combining low metabolic stress with rich proprioceptive feedback, rather than relying on high metabolic demand, may be particularly effective in accelerating post-exercise cardiovagal recovery kinetics.

An important methodological finding of the present study is that changes in HF power were largely explained by concurrent changes in fR. Although conventional univariate analyses identified a significant effect of time, this effect disappeared after fR was included as a covariate in the linear mixed-effects model, indicating that fluctuations in the HF band cannot be attributed solely to changes in cardiac autonomic modulation. This observation is consistent with previous evidence identifying respiratory sinus arrhythmia as a major determinant of HF power [12, 65]. Indeed, changes in fR can substantially influence HF values independent of actual cardiac vagal outflow [66, 14]. Therefore, our findings suggest

that interpreting HF power as a direct marker of parasympathetic activity during exercise without accounting for concurrent changes in fR may lead to misleading conclusions.

In contrast, LF power and the LF/HF ratio appeared to reflect a different autonomic mechanism during the later stages of recovery. A previous study suggested that the increases in LF power and the LF/HF ratio observed following systemic or maximal exercise are associated with compensatory autonomic responses triggered by post-exercise hypotension and venous blood pooling, and that LF power primarily reflects baroreflex function rather than direct sympathetic activity [67]. Consistent with this view, accumulating evidence indicates that the LF band and the LF/HF ratio are more closely related to arterial baroreflex sensitivity and vasomotor tone modulation (0.1 Hz Mayer waves) than to pure cardiac sympathetic activation [68, 69]. In our study, the LF/HF ratio increased during SPU, returned to baseline within the first 5 minutes of recovery, and subsequently rose above resting values during the 10–15 and 15–20 minute recovery intervals in parallel with LF power. Notably, these delayed increases occurred despite the restoration of resting heart rate and the pronounced vagal reactivation reflected by elevated RMSSD and HF values. Collectively, these findings further support previous evidence that LF power and the LF/HF ratio should not be interpreted as simplified markers of sympathovagal balance or sympathetic activation [68, 70]. Instead, we believe that the late-phase elevations observed following SPU represent a dynamic autonomic–vascular adaptation in which arterial baroreflex mechanisms recalibrate vascular tone following the enhanced venous return generated by rhythmic soleus contractions.

Although no statistically significant sex differences were detected at any individual time point, the significant time × sex interaction indicates that the temporal pattern of autonomic recovery differed between women and men. Female participants appeared to exhibit a more dynamic pattern of vagally mediated HRV responses throughout exercise and recovery despite comparable absolute lnRMSSD values. This temporal response may reflect sex-specific differences in autonomic regulation, potentially influenced by estrogen-mediated vagal responsiveness [71] and differences in vascular compliance during rhythmic soleus contractions [30]. However, further studies specifically designed to investigate sex-related autonomic mechanisms are needed to clarify these interpretations.

Taken together, our findings identify SPU as a unique physiological model characterized by minimal cardiovascular demand yet rapid cardiovagal recovery, providing new insights into the autonomic regulation of low-intensity exercise and establishing a physiological foundation for future mechanistic and clinical investigations.

Limitations

Several methodological limitations should be considered when interpreting the present findings. First, no time-matched non-exercise seated control session was included. Therefore, the contribution of SPU cannot be fully distinguished from time-dependent stabilization or passive recovery effects. Although the standardized 5-min baseline period likely allowed participants to acclimatize to the experimental setting,

the characteristic biphasic autonomic pattern observed during SPU—consisting of acute vagal withdrawal followed by rapid post-exercise recovery—may be consistent with an exercise-related response; however, this interpretation requires confirmation in future studies incorporating a time-matched seated control condition. In addition, although exercise cadence was standardized using a digital metronome and visual supervision, electrogoniometry or real-time surface electromyography was not employed, precluding quantitative assessment of fatigue-related movement deviations. Furthermore, although several physiological mechanisms were proposed to explain the rapid parasympathetic recovery observed following SPU, baroreflex sensitivity, stroke volume, cardiac output, blood pressure, and venous return were not measured directly. Likewise, although fR was statistically controlled, tidal volume was not assessed and may have independently influenced HF power. Consequently, future studies incorporating direct physiological measurements are needed to further clarify the proposed mechanisms. Moreover, only a single 5-minute SPU session was investigated, therefore, the autonomic responses observed in the present study cannot be generalized to longer-duration SPU protocols or repeated exercise sessions. Finally, the study included only healthy young adults (n = 32), and training status was determined by self-report rather than a standardized physical activity questionnaire. Accordingly, these findings should be interpreted with caution and confirmed in larger and more diverse populations, including older adults, individuals with autonomic dysfunction, and trained athletes.

Conclusions

A single isolated SPU session, despite imposing only a very low physiological load, was associated with marked vagal withdrawal during exercise, followed by a pronounced parasympathetic rebound during early recovery. Given its seated nature and minimal physical demands, SPU may represent a potentially accessible exercise modality for sedentary individuals, older adults, and clinical populations with limited exercise capacity. The rapid autonomic recovery observed in the present study also provides a rationale for investigating SPU as an active recovery strategy following more strenuous exercise. However, this potential application requires direct evaluation in controlled studies. Whether repeated SPU sessions produce sustained improvements in cardiac autonomic regulation remains unknown. Because no time-matched non-exercise control condition was included, the findings should be interpreted as within-session temporal associations rather than causal effects. Nevertheless, they provide a physiological basis for future controlled studies evaluating the cardiovascular and autonomic effects of SPU.

Abbreviations

BMI Body Mass Index

ECG Electrocardiogram

fR Respiratory Frequency

HF High-Frequency Power

HR Heart Rate

HRmax Maximum Heart Rate

%HRR Percentage of Heart Rate Reserve

HRV Heart Rate Variability

LF Low-Frequency Power

LF/HF Low-Frequency to High-Frequency Ratio

ln Natural Logarithm

pNN50 Percentage of Successive Normal-to-Normal Intervals Differing by More Than 50 ms

RMSSD Root Mean Square of Successive Differences

RPE Rating of Perceived Exertion

SD1 Poincaré Plot Standard Deviation Perpendicular to the Line of Identity

SD2 Poincaré Plot Standard Deviation Along the Line of Identity

SPU Soleus Push-Up

TP Total Power

Declarations

Ethics approval and consent to participate The study was approved by the Ethics Committee of Ankara Yıldırım Beyazıt University Faculty of Medicine (19 February 2026, Protocol No. 88). Written informed consent was obtained from all participants before testing.

Clinical trial number: not applicable.

Consent for publication Not applicable.

Availability of data and materials The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests The authors declare that they have no competing interests.

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Authors' contributions YB, DG and SAT conceived and designed the study. YB, EB and DG conducted the experiments and collected the data. YB and EB performed the statistical analyses. YB, SAT and FB

drafted the manuscript. All authors interpreted the findings, critically revised the manuscript, and read and approved the final manuscript.

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Figures

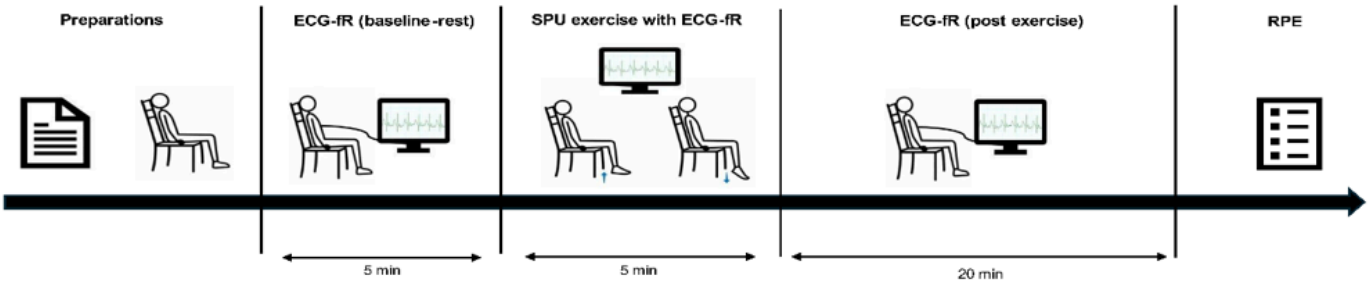


Figure 1

Experimental Timeline ECG: electrocardiogram, fR: respiratory frequency, RPE: rating of perceived exertion

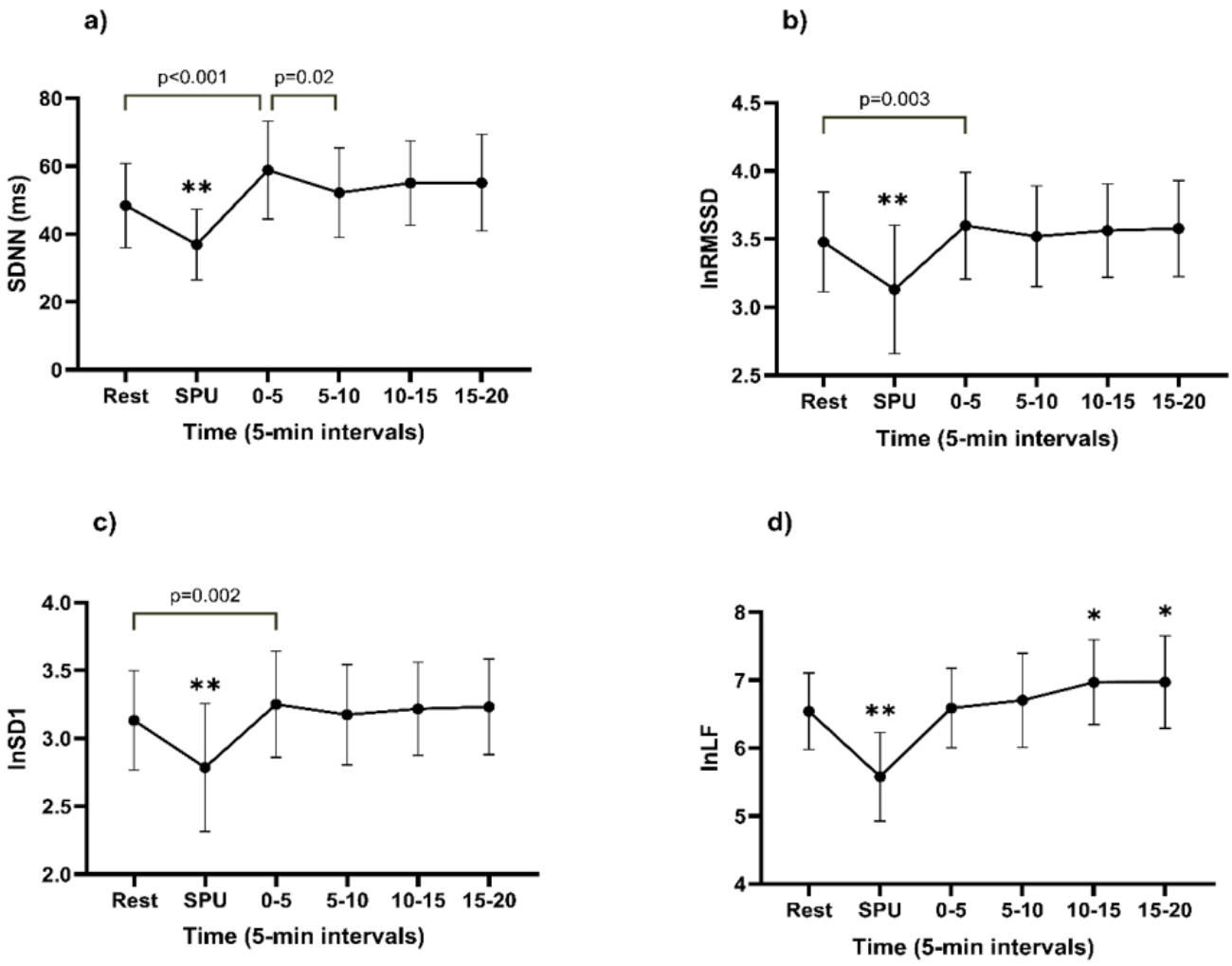


Figure 2

Changes in heart rate variability (HRV) parameters before, during, and after soleus push-up (SPU) exercise. (a) SDNN, (b) lnRMSSD, (c) lnSD1, and (d) lnLF measured during the baseline period (Rest), SPU, and four consecutive 5-min recovery intervals (0–5, 5–10, 10–15, and 15–20 min). Data are presented as mean ± SD. **p < 0.001 versus all other time points. *p < 0.05 versus Rest. Additional significant pairwise comparisons identified by post hoc analyses are indicated above the corresponding graphs.

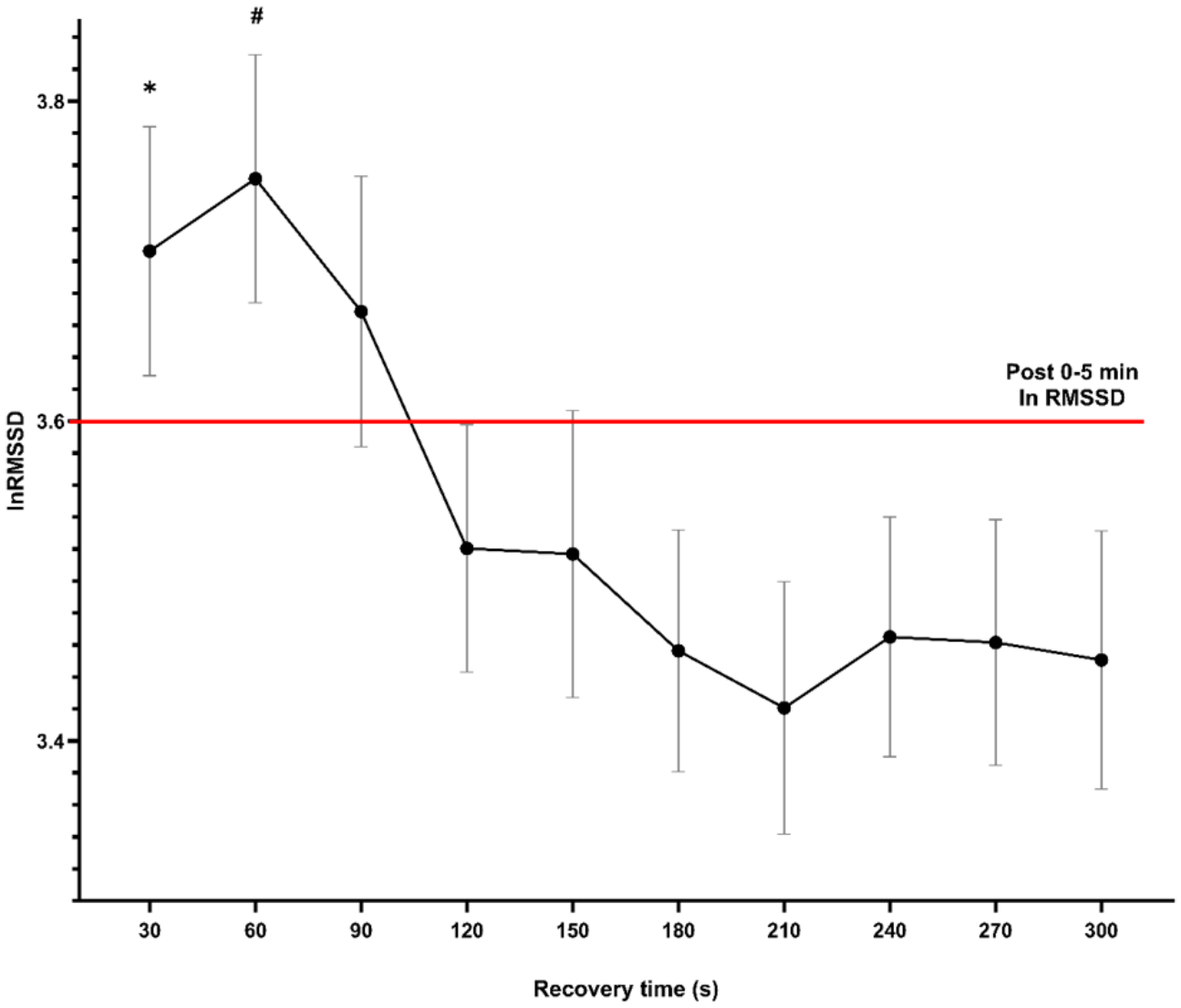


Figure 3

Detailed time course of cardiac vagal recovery during the first 5 min following soleus push-up (SPU) exercise. lnRMSSD was calculated in consecutive 30-s recovery intervals from 0–30 s to 270–300 s after exercise cessation.The horizontal red line indicates the mean lnRMSSD value for the 0–5 min recovery period. Data are presented as mean ± SEM. *p < 0.05 versus the 150–180 s and 180–210 s intervals. #p < 0.01 versus all intervals from 90–120 s to 270–300 s.

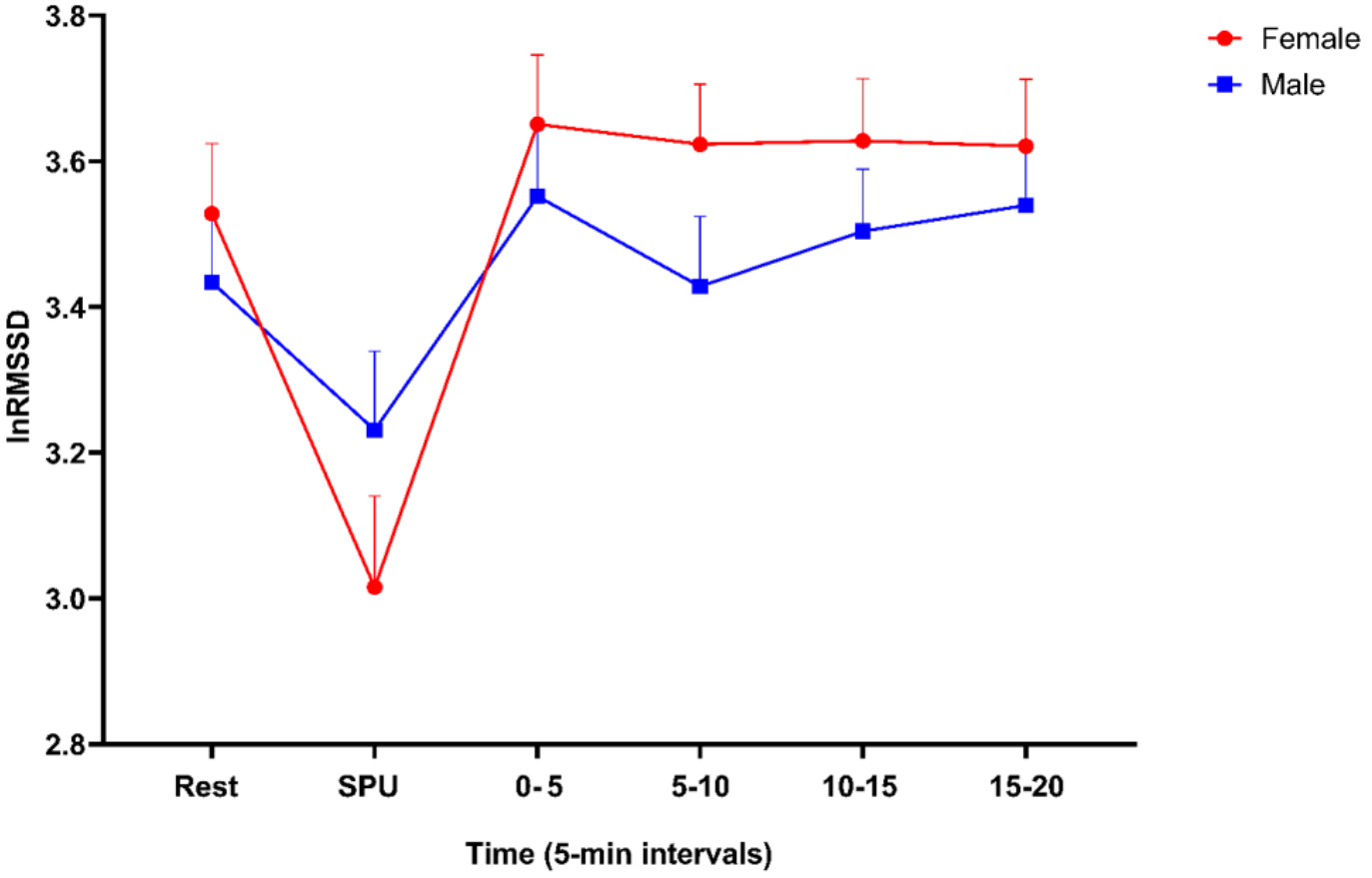


Figure 4

Sex-specific changes in lnRMSSD before, during, and after SPU. lnRMSSD was measured during baseline (rest), SPU, and four consecutive 5-min recovery intervals (0–5, 5–10, 10–15, and 15–20 min). Data are presented as mean ± SEM.