RECOVERY OF HEART RATE VARIABILITY FOLLOWING EXERCISE: IMPACT OF EXERCISE PERFORMANCE AND PREFERENCE

by

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A THESIS

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Reduced heart rate variability (HRV) following resistance (RE), compared to aerobic (AE) exercise indicates greater cardiovascular strain (Forjaz et al., 2000; Pescatello et al., 1999); however, performance and preference are not commonly considered as determinants of this response. Therefore, we assessed the impact of RE and AE on HRV and further examined responses based on modality performance and preference. Twelve young, healthy individuals (11M, 1F) performed separated bouts of RE, 6x10 reps at 70% of 1-repetition maximum (1-RM) and AE, 30min at 70% of their peak oxygen consumption (VO_{2peak}). HRV was assessed at baseline (BL), immediately following (IP), and at 30-minute intervals for 2 hours after exercise. All variables were assessed via 2-way repeated measures ANOVA and results reported as mean±SD. When exercise conditions were examined separately, HF was significantly reduced IP in AE only, despite R-R interval returning to baseline values by 60 minutes *(p*=0.13). Although not statistically significant following RE (*p*=0.13), lower trends may have influenced a slower return of R-R interval at 60 (*p*=0.05). These data suggest an earlier restoration of vagal tone following AE, compared to RE. However, when performance and preference are considered between the conditions HF was reduced at IP *(p*=0.03; *p*=0.05) and tended to remain lower at 30-min *(p*=0.051; *p*=0.09). Relative exercise workloads achieved during exercises that are favored, or preferred may influence HRV following exercise regardless of modality.

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Introduction

The National Health and Nutrition Examination Survey (NHANES) from 2011 to 2016 revealed an estimated 70% of all major cardiovascular disease-related events were attributed to low and moderate cardiovascular health status. Modification of health status from moderate to high, or even partial improvements from low to moderate, are considered a source for reducing annual cardiovascular disease-related events (*Life's Simple 7*, 2015; Tsao et al., 2022). Regular physical activity, in the form of aerobic exercise, is recommended as a first-line non-pharmacological treatment for managing cardiovascular disease risk, driving improvements in insulin sensitivity, providing protection against oxidative stress, and regulating inflammatory signaling (DeSouza et al., 2000; Lavie et al., 2015). Similarly, the benefits associated with resistance exercise display a strong overlap with aerobically mediated adaptations; however, despite patterns of rapid growth in popularity, resistance exercise lacks conclusive evidence as an alternative to aerobic exercise for delaying, or reducing cardiovascular disease risk (Konopka & Harber, 2014).

Defined by the American College of Sports Medicine (ACSM), aerobic exercise is any exercise that recruits large muscle groups, is maintained continuously, and is rhythmic in nature (Patel et al., 2017). Further, the American Heart Association (AHA) defines minimum weekly aerobic exercise requirements of at least 150 minutes of moderate-, or 75 minutes of intense-aerobic exercise necessary to maintain or improve health status (American Heart Association, 2024; Pelliccia et al., 2021). Importantly, in the United States, only 26% of adult men and 19% of adult women achieve guideline-directed activity levels, suggesting a direct impact of inactivity on cardiovascular health and related to exercise adherence (Bays et al., 2021). Therefore, research that extends our understanding of resistance exercise effects on

cardiovascular health may provide an alternative to classic aerobic exercise prescription and may improve adherence to exercise necessary to improve health status.

Commonly, resistance exercise is associated with a high degree of strain eliciting acute, and potentially deleterious effects, on indices of cardiovascular health (Daniela et al., 2022); however, current literature aimed at elucidating effects of resistance exercise training on blood pressure has revealed robust reductions following 14 weeks of resistance training (Nascimento et al., 2014). Despite similar findings by others, and in some cases an ability of resistance exercise to reduce blood pressure in young, healthy normotensive individuals (Carter et al., 2003), investigations focused on acute autonomic modulatory responses to exercise during the acute recovery window following exercise (Forjaz et al., 2000; Halliwill, 2001; Halliwill et al., 2013; Pescatello et al., 1999) suggest resistance exercise may negatively influence parasympathetic tone, and subsequently autonomic modulation of the heart during recovery (Kingsley & Figueroa, 2016).

Importantly, the above evidence is limited by use of consistent participant populations that may result in interpretations drawn from comparisons between trained to untrained populations, and by examining cardiovascular responses to exercises of differing type, time, and intensity with a general disregard for participant preference and performance. Therefore, the collective purpose of this investigation was twofold, as we sought to determine the acute impact of aerobic compared to resistance exercise on autonomic modulation, assessed via heart rate variability, in a cohort of well-trained individuals displaying moderate-high levels of performance between aerobic and resistance exercise modes. We hypothesized that the use of relatively intense aerobic and resistance exercise models would yield similar effects on parasympathetic outflow and explain a delayed return of heart rate toward baseline values.

Second, we attempted to examine the relationship between exercise performance, and exercise preference as determinants of exercise-related cardiovascular responses. We hypothesized the better exercise performance or presence of a self-reported preference for aerobic or resistance exercise would elicit a suppression of parasympathetic outflow, and subsequently, account for a slower return of heart rate toward baseline values.

We anticipate that these findings will extend our understanding of the exercise intensity-related impact on acute cardiovascular responses to exercise and while acutely may be viewed negatively, should not preclude its use as a tool to combat cardiovascular disease progression. Further, we anticipate these results will highlight the importance of exercise preference and performance bias that, regardless of modality, suggest the acute modulatory effect on autonomic modulation of the heart are resulting from a participant's comfortability or desire to complete exercise. Collectively, we aim to extend the use of resistance exercise as a model to combat cardiovascular disease progression, emphasizing similar acute responses to two modes of exercise of similar intensities, and separately to bolster findings supporting exercise responses may be driven by participant preferences and/or exercise performance biases.

Background

The Role of Heart Rate Variability in Exercise Physiology

Mechanisms of Heart Rate Variability

Our nervous system is structurally divided into the central nervous system (CNS) and the peripheral nervous system (PNS). The central nervous system, comprised of the brain and spinal cord, is responsible for processing and directing neuronal signals within pathways of higher-level brain functions. Specific brain regions are thus tasked with integrating feedback signals from tissues and our environment to create a response that permits effective interaction with our world (Thau et al., 2024). In the case of exercise, the influence of this integration is carried out through the peripheral system via autonomic neuronal transmission (Anne M. R. Agur & Arthur F. Dalley, 2019) further subdivided into two branches, and capable of modulating physiologic function by altering the balance between sympathetic and parasympathetic tone. Commonly recognized as the "fight or flight" response, sympathetic innervation remains tonically inactive or low at rest (Battipaglia & Lanza, 2015). Chronic elevation of sympathetic tone is commonly associated with poorer health status, including the development of hypertension (high blood pressure) and other cardiovascular diseases, as well as negatively associated cardiovascular outcome measures such as increased arterial stiffness, peripheral vascular resistance, and pulse wave velocity (Fisher et al., 2009; Waxenbaum et al., 2024). Conversely, vagal tone, or tonic activation of parasympathetic nervous system at rest is responsible for "resting and digesting" as the predominant signal in a healthy, resting individual (Waxenbaum et al., 2024). Although these two branches have opposing effects, in most regions of the body, their dichotomous actions coordinate responses to stress (Anne M. R. Agur & Arthur F. Dalley, 2019). In particular, these

effects are more easily visualized via measurements of heart rate variability and are often used as a measurement for determining the strain of a single or multiple exercise sessions.

Sinoatrial (SA) nodal cells display a characteristic autorythmicity that guide the pace of the heart and its overall electrical activity. The membrane potential of the SA is altered as ions pass through membrane-specific channels, altering the charge of the SA node culminating in a membrane depolarization. In response, an electrical signal is generated and transmitted to cardiomyocytes of the atria and terminates at the ventricles to complete a cardiac cycle (Ashley & Niebauer, 2004). Depolarizations coordinating cardiomyocyte contraction can be monitored through an electrocardiogram (ECG) and displayed as electrical signals via electrodes placed on the skin's surface (Ashley & Niebauer, 2004) and measurement of the variability between successive heart beats is used to determine acute changes in sympathovagal innervation at the SA node (G. Billman, 2011). Specifically, heart rate variability reflects the variations in the length between the R waves, termed the R-R interval identified as the peak within the QRS complex and representing the variation between the depolarization of the main body of the heart (Dong, 2016).

The cardiac action potential begins with a slow increase in membrane potential due to an influx of sodium. This movement of sodium creates a spontaneous depolarization. At threshold, there is an influx of calcium causing a rapid depolarization until the max potential is reached in which calcium channels are closed and there is an efflux of potassium ions causing repolarization. Once membrane potential has once again reached -60, potassium channels close and sodium begins to flow into the cell again, beginning a new action potential. Observed at rest, parasympathetic tone maintains a low rate of depolarization, and subsequently maintains a low heart rate through the release of acetylcholine via vagal nerve activation that binds to muscarinic

receptors on the SA node. Conversely, in response to an increased sympathetic innervation at the SA node, commonly observed during exercise, there is an increase in the rate of depolarization resulting in an increase in heart rate due to the release of norepinephrine that opens sodium and calcium channels allowing threshold to be reached faster, resulting in faster depolarization and re-polarization rates, and therefore a faster heart rate (Battipaglia & Lanza, 2015). Following exercise, the ability to reestablish a parasympathetic dominant signal controlling heart rate may be sensitive to session strain; thus resistance exercise may augment sympathetic signaling during recovery and further disrupt sympathovagal balance compared to aerobic exercises (James et al., 2012; Marasingha-Arachchige et al., 2022).

Measuring Heart Rate Variability – Clinical tool in the field

Although the physiologic explanations may vary between medical disciplines, the most researched aspect of heart rate variability has focused on its sensitivity to exercise. There exists a variety of heart rate variability collection and analysis techniques that have been utilized in research examining responses within an ultra-short- (<5 minutes), short- (5 minutes), or long-term (24h) timeframe of recording, with little consensus on their effectiveness (Esco & Flatt, 2014; Shaffer & Ginsberg, 2017). Early work examined ultra-short and short-term measurement effectiveness, yielding little relevance within clinical settings. However, many studies have since been conducted to test the efficacy of short-term compared to long-term recordings. Esco et al. (2014) determined that recordings as short as 60s are appropriate for assessing changes to heart rate variability in collegiate athletes performing maximal graded treadmill exercise. Heart rate and ECG were measured at baseline, 10-, 30-, 60-seconds, and every 5-minutes following exercise for 30 minutes. This study revealed no differences between measurement types before

exercise; however, following exercise, use of the ultra-short-term indices of 10- and 30-seconds were not consistent with the 60-second and 5-minute measurements (Esco & Flatt, 2014)

These findings are consistent with the findings of Nussinovitch et al. (2011) but may be an artifact of longer recordings in the field compared to longer measurement in a less complex, more standardized clinical setting, highlighting the greater utility of longer measurements for use as a diagnostic tool in patients with cardiovascular-related disorders (Shaffer & Ginsberg, 2017). Specifically, analyzing the standard deviation between all R-R intervals over 24 hours (SDNN) has been deemed the most reliable measurement for determining cardiac morbidity and mortality, with values below 50 ms being classified as unhealthy and above 100 ms as healthy (Shaffer & Ginsberg, 2017). Conversely, there have been many studies assessing the efficacy of short-term recordings in determining mortality risk following a cardiovascular event, such as a heart attack, and for assessing future risk of a cardiovascular event. Fang et al. (2020) examined data from 3,094 patients, exploring the association between heart rate variability and risk of all-cause mortality or cardiovascular-related events in patients with CVD during a 1-year follow up. Patients exhibiting lower heart rate variability, fewer differences between successive R-R intervals due to lower parasympathetic tone and increased sympathetic tone, had a 112% and 46% higher risk of all-cause death and cardiovascular events. With respect to all-cause mortality, a low SDNN or increase of low frequency (LF) power, indicative of a greater sympathetic influence, are associated with greater cardiovascular risk opposed to increased high frequency (HF) power, associated with vagal tone or parasympathetic influence (Fang et al., 2020).

Extrapolating these findings to exercise in healthy individuals is less straightforward; however, investigations have sought to determine the impact of exercise with heart rate variability used as a tool to monitor recovery status. Briefly, at the onset of exercise,

parasympathetic activity decreases until it is withdrawn completely, allowing heart rate to increase to ~80-100 beats per minute; further increases in heart rate are sympathetically mediated (Figure 1; Rowell, 1993).

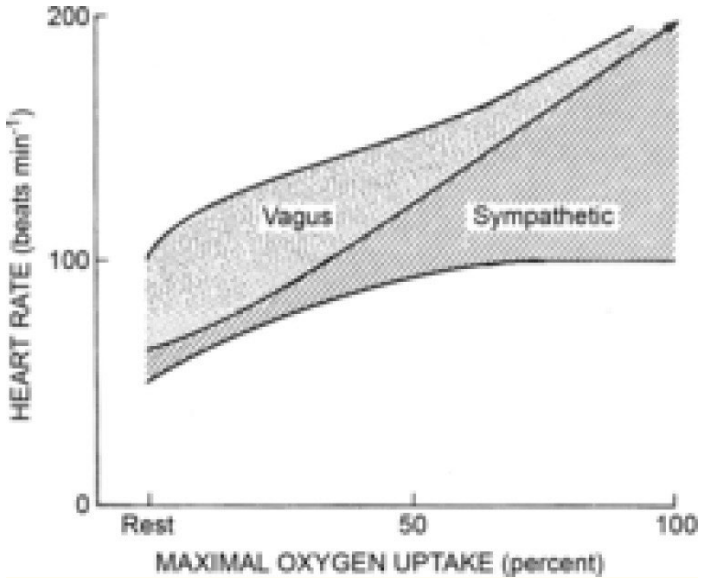

Figure 1: The balance between the sympathetic and parasympathetic nervous systems at rest and during exercise (Rowell, 1993).

Measurements of heart rate variability allow for an assessment of autonomic modulation of the heart following exercise and reflect the strain-induced by the preceding exercise session (Buchheit et al., 2007; Parekh & Lee, 2005). Commonly, the decline in high frequency power observed after exercise is tied to an acute decline in parasympathetic influence observed at the heart, and the extent of this suppression related to exercise strain (James et al., 2012; Marasingha-Arachchige et al., 2022). Following aerobic exercise, the short-lived reduction of high frequency power allows for a more rapid return of heart rate, and R-R interval, toward resting values is associated with positive cardiovascular outcomes (Heffernan et al., 2006). However, following resistance exercise, a sustained reduction of parasympathetic tone suggests sympathetic activity is longer-lived and persists, sometimes beyond 30 minutes following more

strenuous exercise, and may therefore be considered a negative cardiovascular response (Hayashi et al., 1992).

Within exercise physiology, there are few studies that have determined the differences in cardiovascular responses between aerobic and resistance exercise directly. This has led to a negative connotation towards resistance exercise suggesting a poorer acute cardiovascular recovery response profile. As mentioned, training load can have a significant effect on autonomic responses and recovery following exercise; however, it is mechanistically challenging to match loads between resistance and aerobic exercise. That said, the overlap between adaptive signaling mechanisms and the potential to extend exercise adherence to reduce instances of inactivity-related cardiovascular disease progression highlights an immediate need for research investigating the impact of alternative exercises on the acute cardiovascular responses.

Methods

This study took place in the Exercise and Environmental Physiology Lab at the University of Oregon and was approved by the Institutional Review Board (IRB) at the University of Oregon. This study examines a subset of data collected as part of a larger project supporting a doctoral dissertation. Importantly, the data presented within the current honors thesis examines the heart rate response, and measurements of heart rate variability, only. Any description of additional measurements is to provide study timeline context, and to maintain transparency regarding participant requirements during measurement periods.

Participants

12 subjects, 1 female and 11 males, between the ages of 18-35 were recruited to participate in this study. Subjects were recruited from the University of Oregon and surrounding Eugene area and provided with written, informed consent following a verbal briefing of experimental procedures. Subjects must have not met any of the exclusion criteria (Appendix A) and be within the inclusion criteria (Appendix B).

Prior to participation, subjects were asked to arrive to the lab for a pre-screening visit in order to determine their general health, training history, and training status in order to ensure there were no confounding variables due to unaccustomed stress-responses in one or both of the exercise modalities. Qualifying participants based on specified inclusion criteria (Appendix B) were then asked to return to the lab for two separate performance screening visits to determine maximal oxygen consumption on a cycle ergometer (VO_{2peak}) and a 1-repetition maximum value for the traditional back squat (1-RM).

Experimental Procedure

Eligible subjects completed a total of five visits. Visit 1 was an initial screening/orientation visit. Visits 2 and 3 consisted of a cycling VO_{2peak} test and assessment of 1-repetition maximum (1-RM) back-squat. Visits 4 and 5 consisted of an aerobic exercise protocol and a resistance exercise protocol.

Screening Visit (1 or 2): Assessment of Maximal Oxygen Consumption (VO_{2peak})

Subjects completed a three-minute warm-up on the cycle ergometer set at a resistance of 60 watts (W). Immediately following this, the resistance increased by 0.5 watts per second (W/s) and continued until volitional exhaustion. The test was terminated when subjects were no longer able to maintain a cadence greater than 60 revolutions per minute despite strong verbal encouragement. Heart rate was recorded using a Garmin heart rate monitor. Expired gasses were analyzed for determination of VO_2 (ParvoMedics, Salt Lake City, UT, USA). A subject's VO_{2peak} is considered to have been reached if their respiratory exchange ratio (RER) exceeds 1.1, rating of perceived exertion (RPE) exceeds 17, or a plateau in heart rate or VO_2 is observed, despite further increases in workload. Subjects VO_{2peak} was confirmed with a supramax test in which the intensity was set to 110% of the final wattage completed during the VO_2 test. VO_{2peak} is confirmed if there are no significant changes in oxygen consumption despite the increase in workload.

Screening Visit (1 or 2): Assessment of 1-Repetition Maximum (1-RM)

Subjects completed a brief warm-up that included 5 minutes of cycling on a cycle ergometer at a self-selected pace, followed by 10 body weight squats and 10 walking lunges. Subjects were then asked to perform three warm up sets each separated by two minutes in the following order: 10 repetitions of approximately 40-50% of their estimated 1-RM, 5 repetitions at approximately 60-70% estimated 1-RM, and 3 repetitions of approximately 80-85% estimated

1-RM. Subjects were then given 5 attempts to complete one-repetition at loads which increased following each successful attempt. Three to five minutes of rest were given prior to each 1-RM attempt. Parallel to the VO_{2peak} assessment, each participant received strong verbal encouragement from the trained study team members. The greatest load that was performed successfully with good form and without assistance from a spotter was deemed their 1-RM.

Aerobic Exercise Protocol

Subjects were asked to complete a cycling session set at 70% of their previously determined VO_{2peak} for 30 minutes that included a five-minute warm-up at a self-selected pace. During exercise, heart rate and ratings of perceived exertion were monitored throughout and recorded every 5 minutes. To ensure exercise intensity was appropriately maintained throughout the experimental visit we also spot checked the subject's oxygen consumption at the protocol mid-point (after approximately fifteen-minutes of exercise). If the subject's VO_2 exceeded 70% of their VO_{2peak}, the wattage was decreased. Preceding exercise initiation and beginning immediately following completion, we began our hemodynamic assessments. Hemodynamic assessments included femoral blood flow via ultrasound, blood pressure, heart rate, and a brief paced-breathing determination of heart rate variability measure via 3-lead electrocardiogram. All measurements were completed prior to exercise, immediately following exercise completion, 30-, 60-, 90-, and 120-minutes into the recovery period.

Resistance Exercise Protocol

The resistance exercise protocol consisted of 6 sets of 10 repetitions set at 10RM, or 75% of the previously determined 1-RM with each set separated by 2 minutes of rest. The intensity of each set was load-adjusted, when needed, to elicit volitional fatigue within a 10-repetition target window. During exercise, heart rate and ratings of perceived exertion were monitored throughout

and recorded following each set. Additionally, a visual analog scale was used to determine perceived recovery from the previously completed set (Laurent et al., 2011). In the event subjects reported a readiness value ≤2, their rest interval was extended by 30 seconds and perceived recovery was reassessed. Our resistance exercise prescription was designed to elicit a metabolic stress response utilizing short rest periods between sets (Bartolomei et al., 2017; Freitas de Salles et al., 2010; Gonzalez et al., 2015). As such, if a subject indicates ≤2 on the perceived recovery scale for more than two consecutive reassessments (after adding an additional minute of recovery), then the exercise session was terminated. Preceding exercise initiation and beginning immediately following completion we began our hemodynamic assessments. Hemodynamic assessments included femoral blood flow via ultrasound, blood pressure, heart rate, and a brief paced-breathing determination of heart rate variability measure via 3-lead electrocardiogram. All measurements were completed prior to exercise, immediately following exercise completion, 30-, 60-, 90-, and 120-minutes into the recovery period.

Heart Rate Variability

Heart rate variability was assessed prior to exercise, immediately following exercise completion, 30-, 60-, 90-, and 120-minutes into the recovery period. Following 15 minutes of supine rest, participants were instructed to breathe along with a metronome set at a frequency of 0.25 Hz, equivalent to 15 breaths per minute. EEG data was measured through three electrodes and continuously collected for 8 minutes while the participants continued to breathe with the metronome. Data was collected at a frequency of 250 Hz and digitized using Windaq Data Acquisition Software (DATAQ Instruments, Akron, OH, USA). In order to reliably analyze the ECG waveforms, it must be free of noise which can be attributed to many factors such as movement, ectopic beats, and irregular rhythms. Ectopic beats originate at the atrioventricular

junction or ventricles rather than at the sinoatrial node and can significantly affect the R-R intervals (Nabil & Bereksi Reguig, 2015). Data was processed by automated marking the peak of each R wave of electrocardiogram waveforms originating at the sinoatrial node, with visual inspection to ensure appropriate and accurate waveform identification. After each R wave was successfully marked, the initial and final 90 seconds of each recording were removed so that only the interim 5 minutes remained. A recording duration of 5 minutes has been demonstrated to be the optimal duration for detection of high, low, and very low-frequency fluctuations on heart rate (Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology, 1996). Following, a time series data file of R-R interval duration was generated using Advanced CODAS Analysis Software (DATAQ Instruments, Akron, OH, USA). The time series data were then input into a custom LabView program for the analysis of frequency domain measures of heart rate variability (UO HRV_G1, University of Oregon, Eugene, OR, USA). Imported data were first interpolated to 4 Hz to obtain equidistant time intervals and then divided into five equal length overlapping segments of 256 samples each. Each segment was de-trended, Hanning-filtered, and fast-Fourier-transformed to derive a periodogram. The five periodograms were averaged to produce the spectrum estimate for the entire time series. For this procedure, the frequency resolution was 0.016 Hz. We defined the high or respiratory frequency band as 0.15-0.40 Hz, a range inclusive of all respiratory power, and the low frequency band as 0.04-0.15 Hz. Total power was reported as the band $\leq$ 0.40 Hz. Mathematically, very low frequency fluctuations ($<$ 0.04 Hz) in heart rate variability are considered non-harmonic, and therefore do not contribute to the overall variability in recordings of such a short duration (Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology, 1996). To avoid erroneous interpretation of low- and high-

frequency components of heart rate variability, very low-frequency power was excluded from this analysis. Data are presented and interpreted as absolute low- and high-frequency power, as well as total power.

Data Analysis

Prior to statistical procedures, all data was assessed for sphericity. If the assumption of sphericity was violated, a Greenhouse-Geisser correction was applied. To determine the changes in heart rate variability, data were analyzed using a two factor (time x conditions) repeated measures analysis of variance (2-way ANOVA) and reported as mean±SD. To determine the influence of exercise preference and performance bias, data were first aggregated based on participant reported preference between aerobic or resistance exercise (qualitative) and analyzed via 2-way repeated-measures ANOVA to determine the influence of preference on heart rate variability during preferred vs non-preferred exercise conditions. Separately, a 2-way repeated measures ANOVA was used to determine the impact of exercise performance on heart rate variability responses to exercise in conditions where participants displayed a performance bias determined by the ratio between maximal aerobic and resistance exercise performance ($V0_{2peak}$:1-RM).

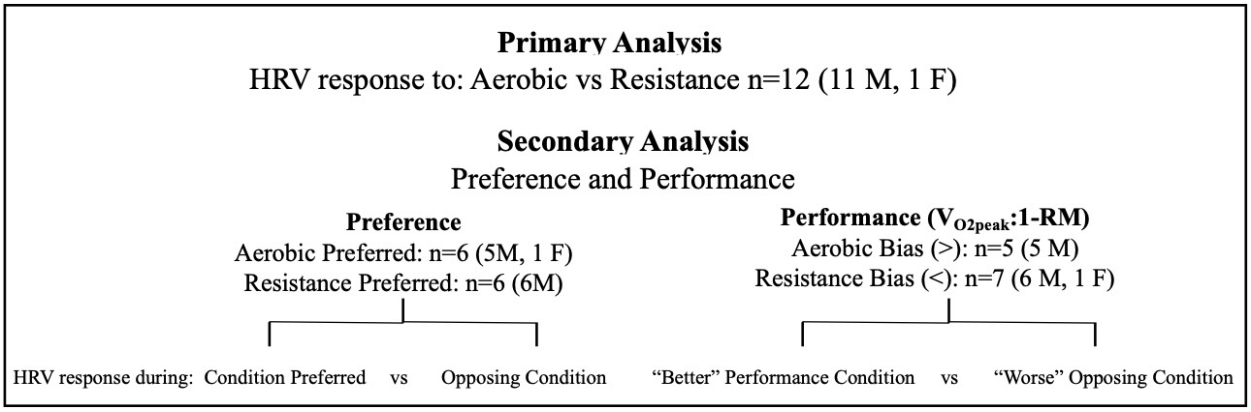

Figure 2: Data Analysis Plan

Results

Heart Rate

Heart rates during both conditions remained elevated relative to baseline (BL), immediately following (IP; $p<0.05$) exercise and until 60-minutes into recovery ($p<0.05$). By 90-minutes, both aerobic and resistance exercise condition responses yielded a return of heart rates toward baseline ($p=0.20$; $p=0.58$, respectively).

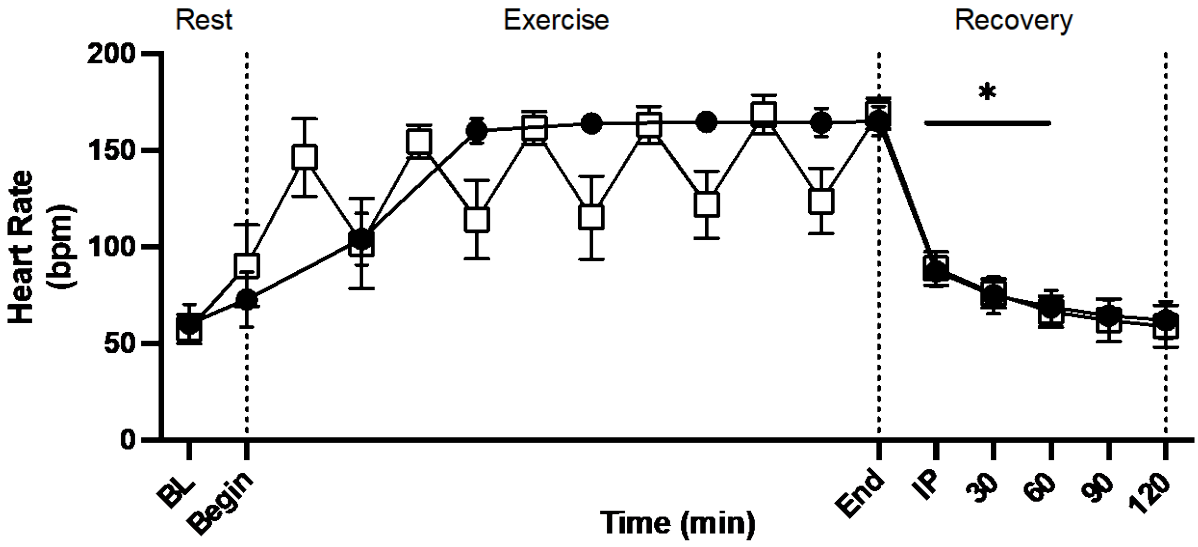

Figure 3: Heart rate response at baseline, during aerobic and resistance exercise, and throughout respective exercise condition recovery periods.

RR-interval

In response to aerobic exercise, participants displayed a reduction in RR-interval immediately following (IP) exercise ($p<0.05$), until 30-minutes of recovery ($p<0.05$) before returning to BL at 60-minutes ($p=0.13$). Conversely, resistance exercise yielded a further suppression of RR-interval present until 60-minutes into exercise recovery ($p<0.05$), before returning toward baseline values at 90-minutes ($p=0.95$).

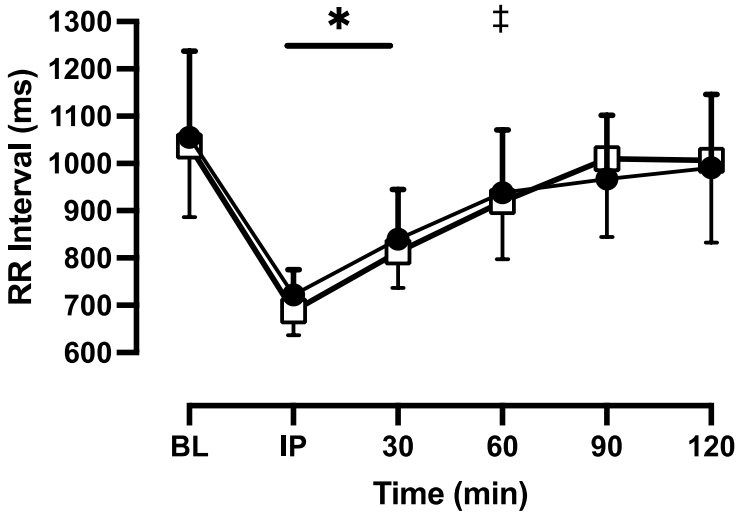

Figure 4: Change in RR-interval at baseline (BL) and throughout recovery from aerobic and resistance exercises.

Preferred exercise reveals suppressed values IP (p<0.05), that remained reduced until 60-minutes after exercise (p<0.05) before returning to baseline values at 90-minutes (p=0.18). Conversely, during exercise that wasn't preferred, RR-interval was reduced IP (p<0.05) and until 30-minutes (p<0.05) before returning to baseline values by 60-minutes (p=0.21).

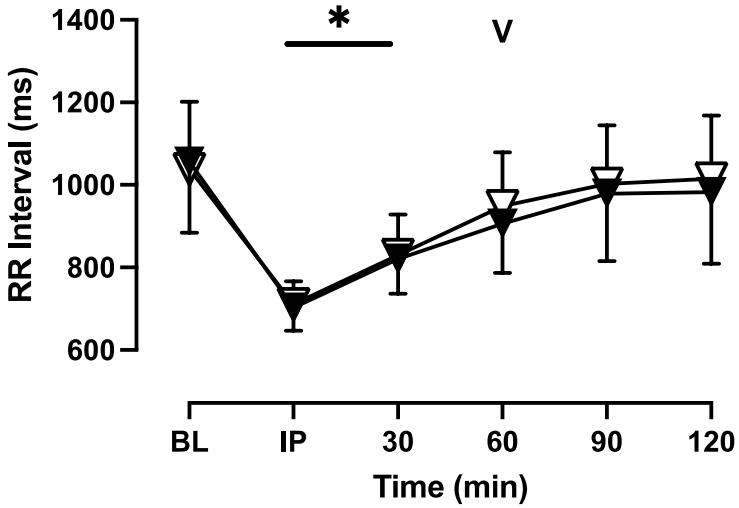

Figure 5: Qualitative analyses of exercise preference comparing preferred exercise effects on RR-interval

Following exercise, participants were “better” at displayed a pattern of reduced RR-interval that remained through 60-minutes of recovery (p<0.05) before returning toward baseline values by 90-minutes (p=0.30). Conversely, when participants completed exercise, they were “worse” at, reductions in RR-interval were observed only until 30-minutes after exercise (p<0.05), before returning to baseline values by 60-minutes (p=0.26).

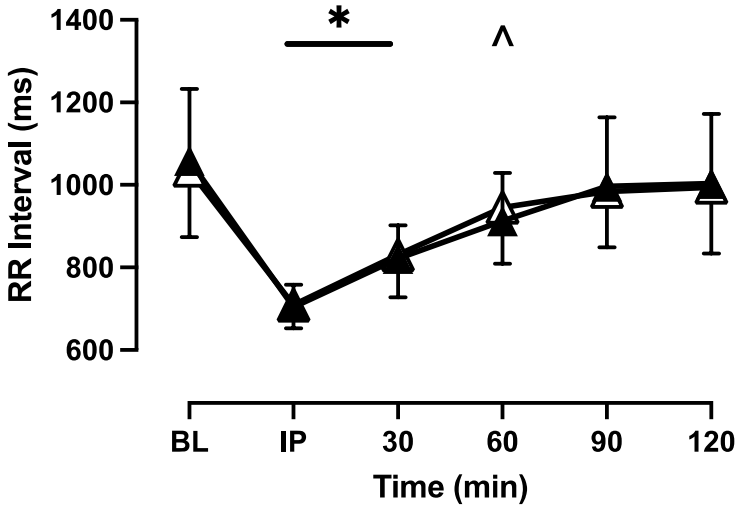

Figure 6: Quantitative analyses determining the effect of exercise bias on R-R interval.

High Frequency Power (0.15Hz-0.40Hz)

In response to only aerobic exercise, a significant reduction in high frequency power was noted immediately following exercise relative to baseline ($p<0.05$). Interestingly, a rapid return of high frequency power was reported at 30-minutes following aerobic exercise ($p>0.99$), whereas the tendency for high frequency power to remain low immediately following resistance exercise ($p=0.13$) persisted until 30min ($p=0.13$).

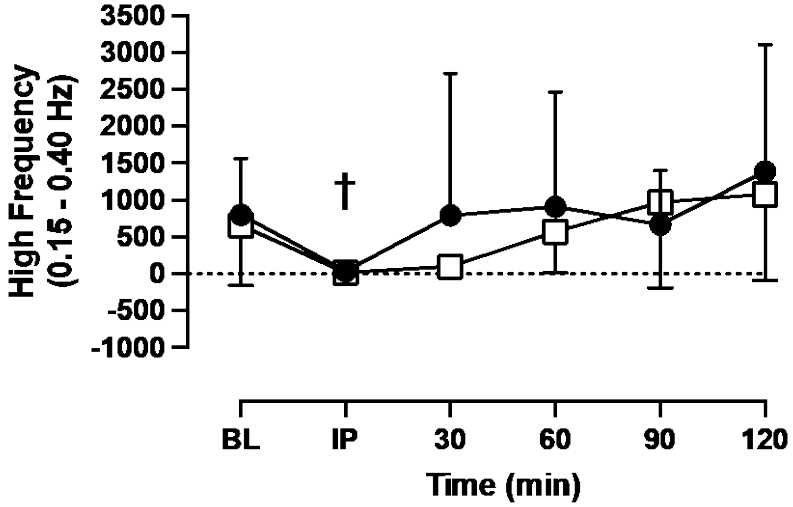

Figure 7: Comparison of high frequency power between aerobic and resistance conditions.

Participants completing exercise they were "better" at displayed a pattern of reduced high frequency power immediately following exercise (p<0.05) that tends to remain reduced at 30min (p=0.051). Although a similar trend (p=0.06) was observed immediately following exercise participants were "worse" at, the tendency for high frequency power to remain reduced at 30min was not observed (p>0.99).

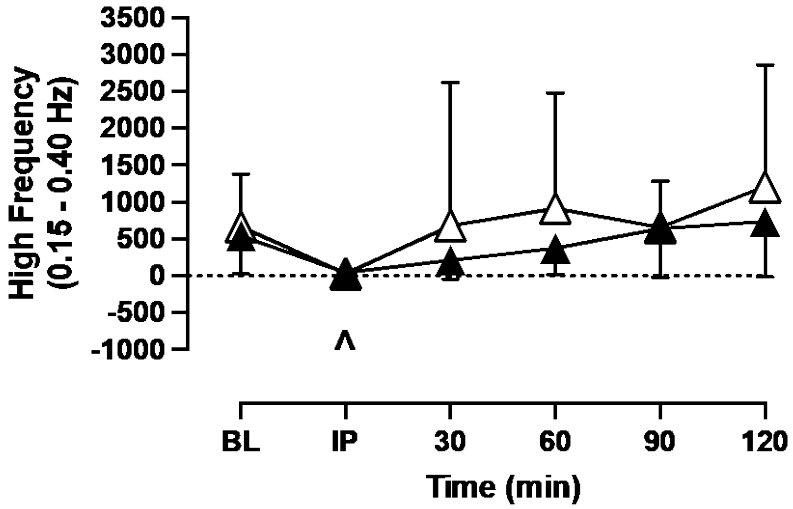

Figure 8: Quantitative analyses determining the effect of exercise bias on high frequency power.

Similar to “better” exercise, a significant reduction of high frequency power was observed immediately following exercise ($p<0.05$) that tended to remain reduced until 30min post-exercise ($p=0.08$). The same tendency to be reduced following non-preferred exercise was observed immediately post ($p=0.06$), but again absent by 30min ($p=0.99$).

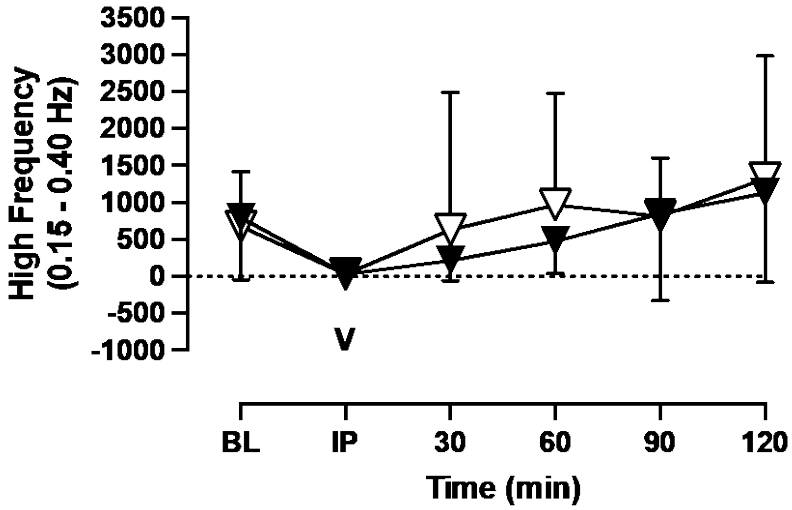

Figure 9: Qualitative analyses determining the effect of self-reported exercise preference on high frequency power.

Exercise Preference and Performance Bias Comparisons

During exercise sessions that participants displayed a performance bias, greater resistance exercise workloads (*p<0.05)* were achieved, while aerobic workloads tended to be different (*p*=0.07) between biased exercise sessions.

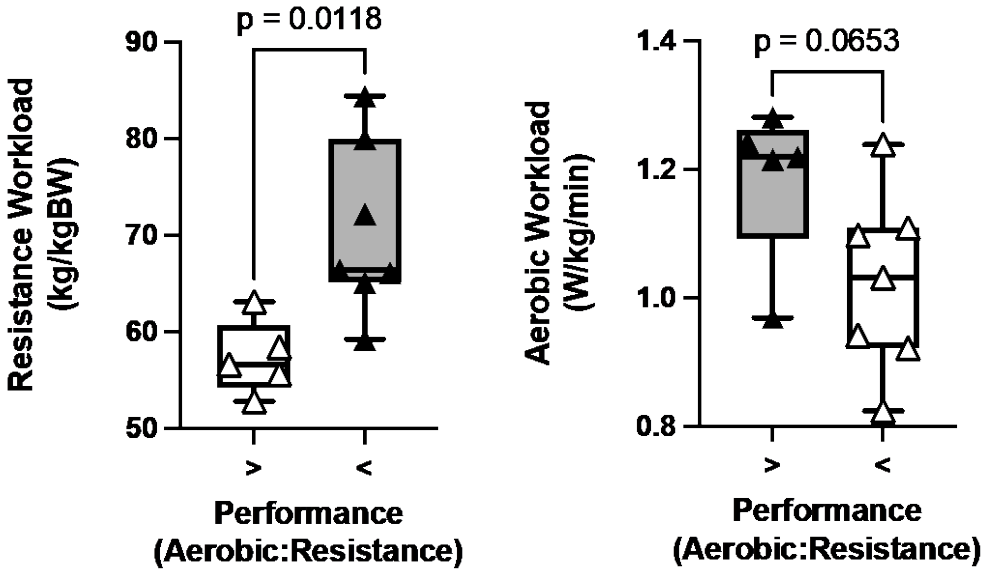

Figure 10: Resistance and aerobic exercise workloads achieved during exercises participants displayed a performance bias toward (Performance).

Similar to Performance, during exercises that were preferred, participants achieved greater resistance exercise workloads ($p<0.05$), and aerobic workloads tended to be different (p=0.06) compared to exercise workloads achieved during non-preferred exercise sessions.

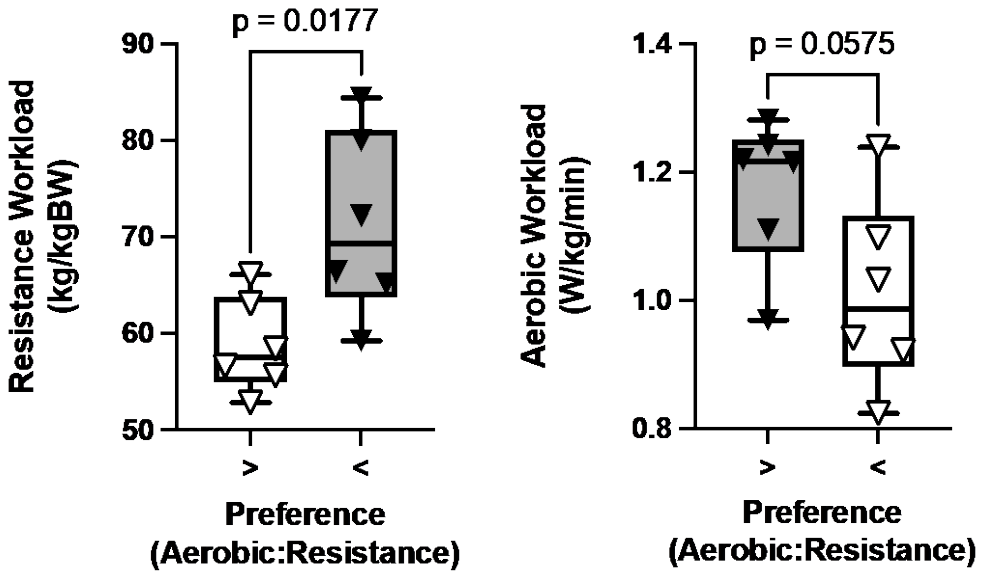

Figure 11: Resistance and aerobic exercise workloads achieved during exercises participants self-reported as their preferred exercise (Preference).

Discussion

This investigation sought to examine the impact of aerobic and resistance exercise on measurements of heart rate variability in moderate-highly trained individuals. Our findings partially support our hypothesis as we observed a greater delay in R-R interval recovery following resistance exercise; however, we note the greater reduction in high frequency power following aerobic exercise only. Second, the current design assessed the impact of heart rate variability responses during exercise that was self-reported as preferred, as well as examining these responses during exercises that participants displayed a performance bias toward. In support of our hypothesis, we revealed exercise performance bias and self-reported preference were associated with a superior suppression of parasympathetic activity, and a significantly greater delay in R-R interval recovery that suggests exercise volume attained, or the strain during these conditions yielded this more negatively associated result.

Heart Rate Variability following Aerobic vs Resistance Exercise

Previous research has examined the contribution of parasympathetic and sympathetic innervation on heart rate variability, often expressed as a ratio between low to high frequency power (Ferreira & Zanesco, 2016); however, these measurements tend to overestimate the contribution of sympathetic activation within the low frequency power band (G. E. Billman, 2013; Houle & Billman, 1999). Low frequency power, unlike high frequency power as a sole representation of parasympathetic tone, includes some contribution from both parasympathetic and sympathetic influence, creating complicated interpretation of findings between investigations (G. E. Billman, 2013; Hopf et al., 1995). Therefore, in the current investigation we cautiously limit our interpretation to the more informative changes to R-R interval, heart rate, and

parasympathetic modulation via high frequency power. Although high frequency values following resistance exercise did not reveal a significant reduction throughout the acute recovery period, we report a tendency for high frequency power to be reduced through 30-minutes of recovery following resistance exercise only. Thus, a tendency for parasympathetic tone to remain low may contribute to the statistically slower return in R-R interval observed following resistance exercise, and a common finding amongst other investigations (Heffernan et al., 2006).

Interestingly, our findings were consistent with that of Heffernan et al. (2006), despite stark contrast in total exercise volume accumulated during a single resistance exercise session as their protocol included 3 sets of 10 repetitions of the barbell bench press, bent-over row, leg extension, leg curl, military press, biceps curl, close-grip bench press, and abdominal crunch combined with shorter inter-set rest periods lasting just 90 seconds. This discrepancy emphasizes the role of fatigue as modulator of autonomic function after exercise. Resistance exercise in the current investigation was continued in a single group of muscles to elicit fatigue; thus, a greater sympathetic signal may be associated with exercise carried out to this extent. Indeed, the number of sets and number of exercises affects sympathetic modulation as 3 or more sets increases sympathetic stress and slows recovery metrics (Marasingha-Arachchige et al., 2022); thus, relative to Heffernan et al., the current design may have elicited a greater strain within the legs after completing 6 sets of 10 repetitions of traditional back squat.

Further, high frequency power appears affected by rest time between sets, where less than 2 minutes of rest allotted between sets causes greater parasympathetic withdrawal after exercise and is associated with delayed recovery metrics (Marasingha-Arachchige et al., 2022). Thus, the manipulation of resistance exercise variables related to set number, repetition and load amount can be compounded by diminished rest to increase the overall intensity associated with lower

high frequency values (Figueiredo et al., 2015; Rezk et al., 2006), although not always a consistent finding between investigations, as some have found higher intensities have less of an effect on high frequency power compared to low intensity (Marasingha-Arachchige et al., 2022). Within the current study, high frequency power appears to be more sensitive to the relatively lower intensity of aerobic exercise. Importantly, this effect may have been brought on by the continuous nature of aerobic cycling exercise, opposed to intermittent nature of resistance exercise. Taken together, the intermittent nature of resistance exercise may explain variability between individual responses limiting statistical reductions of high frequency power immediately after exercise whereas the consistent, and relatively high intensity nature of the aerobic exercise condition in the current design imposes a greater, continuous sympathetic signal that quickly subsides following exercise termination (Figueiredo et al., 2015; Marasingha-Arachchige et al., 2022).

The Impact of Subject Preference and Performance: Metrics of overtraining

To investigate the differences in recovery of heart rate variability after exercise further, we analyzed measurements based on subjects' preference and performance bias between both aerobic and resistance exercise sessions. Measurements of heart rate variability were assessed following exercise participants self-reported as their preferred modality compared to responses observed following the opposing, less-preferred, exercise condition. Separately, a performance ratio was used to assess heart variability responses to exercise deemed their "better" or "worse" modality and calculated from their maximal performance during the VO_{2peak} and 1-RM determinations (VO_{2peak}:1-RM). Intuitively, participants that preferred or displayed a performance bias toward one modality accumulated greater exercise volume when compared to exercise they did not prefer or perform worse in (Figures 10 and 11). These finding, when paired with

measurements of heart rate variability highlight a sensitivity to exercise volume or the workload achieved, and not a direct result of aerobic or resistance exercise. Thus, regardless of exercise type, when exercise was preferred, or performance biased participants were capable of inducing more strain during an acute exercise session the resulting evidence suggests a greater reduction of high frequency persists under these conditions.

Importantly, our population includes individuals with a strong training history, and of moderate-high training status in both exercise modalities; however, these are not elite-level athletes that are likely receiving coaching or strictly adhere to an exercise training regimen. Effective training methods in athletes are centered around optimizing training duration, frequency, and intensity (Seiler, 2010); however, shifts between these variables may be present in recreational athletes that display a preference for, or perform better during specific exercises. Therefore, disruption to the balance between duration, frequency, and intensity may lead to ineffective programming that predisposes an individual to maladaptation, non-functional overreaching, and overtraining (Seiler, 2010), and because these symptoms are expressed across a continuum between individuals, the ability to independently assess the presence of positive or negative responses to programming is advantageous across populations (Singh et al., 2018). In this case, monitoring the acute changes to high frequency power, recovery heart rates or R-R interval may be less informative and incapable of predicting overtraining progression; however, our results emphasize the importance of monitoring acute responses to better inform individuals of the immediate disruptions to recovery status following strenuous exercise, regardless of type.

Research investigating the utility of heart rate variability assessments as a training tool have revealed increased performance when compared to traditional training regimens. In particular, Javaloyes et al., (2019) examined performance metrics of 17 well-trained male

cyclists split into a heart rate variability-guided training program group and a traditionally prescribed exercise regimen based on volume and intensity. After 8 weeks of training with these protocols, peak power output, upper threshold power, and 40 km time trial performance were significantly greater in the heart rate variability-guided group despite no differences in overall intensity and volume (Javaloyes et al., 2019). Kiviniemi et al. (2007) report similar trends with running that together highlight the importance of timing exercise intensity and volume. Heart rate variability-guided training in this case was based on R-R interval changes recorded daily, if an increase or no change in R-R was observed the use of high-intensity training was implemented that day. Conversely, if decreases in R-R were present, lower-intensity training or rest was prescribed, and after four weeks, maximum load and VO_{2peak} significantly increased in heart rate variability-guided training group (Kiviniemi et al., 2007).

Although we did not assess this concept of day-to-day changes in heart rate variability, we highlight the importance of monitoring heart rate variability following exercises that are repeated often, such as in cycling, running or specific compound resistance exercise movements. Overtraining does not have a specific diagnostic criteria, rather it is a "diagnosis of exclusion" (Carrard et al., 2022). It has been hypothesized that heart rate variability can be used as a marker of athletes approaching overtraining status, as imbalances within the autonomic nervous system are thought to manifest as a result. Studies examining the autonomic effects of overtraining have shown a variety of results; however, the majority of them reveal decreased heart rate variability in athletes who are overtrained (Mourot et al., 2004; Plews et al., 2013). Disagreement between investigations are likely due to the inconsistency in overtraining and specific diagnostic criteria separating the presence of functional overreaching, non-functional overreaching, overtraining, and overtraining syndrome (Singh et al., 2018). Mourot et al. (2004) compared to athletes that

were diagnosed with overtraining syndrome, revealing the overtrained group had a significant predominance towards sympathetic activity and a lower overall heart rate variability compared to non-overtrained and sedentary groups (Mourot et al., 2004). Uusitalo et al. (2000) also found an increase in sympathetic activity through increased R-R interval in overtrained subjects (Uusitalo et al., 2000)

Conclusions and Limitations

This investigation has revealed an acute reduction in parasympathetic tone following aerobic exercise, which was not statistically present following resistance exercise. In contrast to previous research, this finding suggests that the acute disruption of parasympathetic signaling is more prevalent following continuous aerobic exercise carried out at a moderate-high intensity when compared to the intermittent nature of resistance exercise of a similar relative intensity. At first glance, this interpretation would suggest that aerobic exercise, and not resistance exercise, elicits a more strenuous stimulus on the modulation of heart rate after exercise. Importantly, however, we report a prolonged reduction of R-R interval following resistance exercise until 60-minutes, which suggests sympathetic influence following the resistance exercise session may persist and compete with the return of vagal tone back to baseline levels.

We recognize that this investigation is not without its limitations, and our ability to accurately assess the presence of sympathetic influence during the acute recovery period is a primary concern. As discussed earlier, it is technically challenging to discern between sympathetic and parasympathetic contribution using low frequency power, particularly when the contribution from the two arms of the autonomic nervous system is not clearly defined within it. Thus, we chose to limit our interpretation to measurements of high frequency while controlling respiratory rate to limit any influence of breathing on vagal modulation. Taken together, future

research should investigate the presence of a sustained reduction to parasympathetic tone after resistance exercise, as high frequency power tended to be reduced for longer, relative to the rapid return to baseline observed following aerobic exercise.

We also believe this work sets the foundation for examining heart rate variability measurements after exercise that is physiologically (performance bias) or psychologically preferred. Examining the relationship of parasympathetic withdrawal following exercise that is preferred or biased seems intuitive, but importantly highlights the risk associated with repeated exercise and overtraining potential. Exercisers that tend to do more similar exercises because they are better at them, or simply enjoy them more, run the risk for greater strain in response to high workloads. In this controlled study we ensured participants maintained 70% of their maximal workload at VO_{2peak} during the aerobic exercise session by measuring their expired gasses throughout exercise. Regardless, individuals with a stronger aerobic preference or training history tended to produce a greater overall workload that may influence the less favorable return of parasympathetic tone after exercise. Similarly, and to a greater extent, this effect was observed in persons who preferred resistance exercise. When taken together, these responses highlight the potential for overtraining to occur more quickly with repeated exercise use, or conversely, highlight cross-training methods to reduce the likelihood of overtraining progression.

In conclusion, we believe this work adds to the literature supporting the use of resistance exercise for combating the progression of cardiovascular disease, or at the very least it suggests that resistance exercise is not more deleterious than moderate-high intensity aerobic exercise. We suggest future work examine more closely the relationship between resistance exercise, exercise adherence, and the prevalence of cardiovascular disease within at-risk populations.

Finally, we support previous findings that highlight the use of acute heart rate variability to determine the presence of a greater exercise strain present following different forms of exercise, and potentially extend support for heart rate variability-guided training paradigms to limit the progression of overtraining in athletes.

Appendix A

Exclusion Criteria

1. Activity levels Tier 1 (recreationally active) or below as defined by McKay et al. (2022).
2. Non-English speaking.
3. Prior diagnosis of cardiovascular disease.
4. Prior diagnosis of diabetes, sleep apnea or obesity.
5. Prior diagnosis of asthma, emphysema, COPD, or cystic fibrosis.
6. Prior diagnosis of autonomic disorders.
7. Prior history of pelvic/abdominal surgery.
8. Self-reported smoking, nicotine, or recreational drug use.
9. Ongoing medical therapy (other than birth control).
10. Ongoing use of OTC/Rx antihistamines.
11. Allergies or hypersensitivities to local anesthetics, skin disinfectants, adhesives, or medications.
12. Currently pregnant, breastfeeding, or desiring to become pregnant in the next 6 months.
13. Mobility restrictions that interfere with physical activity.
14. Previous or ongoing use of performance enhancing drugs.
15. Systolic ≥ 130 measured during screening visit.
16. Diastolic ≥ 80 measured during screening visit.

Appendix B

Inclusion Criteria

1. Activity levels Tier 2 and Tier 3 as defined by McKay, et al. (2022).
2. Training History: average of ≥3 exercise sessions per week and ≥3 months of recent training.
3. Age: 18-35.
4. Back Squat 1-RM: 1.25x-3.0x Body weight (BW) estimated from pre-screening questionnaire then assessed during screening visits.
5. $VO2_{peak}$: Males: 45mL/min/Kg-63mL/min/kg; Females: 32mL/min/kg-46mL/min/kg determined during screening visits.

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