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# Heavy Domain Exercise Delays Recovery of Linear Measures of Heart Rate Variability Independent of Heart Rate

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Supplementary materials:

[https://osf.io/rau7x/?view\\_only=1464ed216f3f4c0b9fab6217014815dd](https://osf.io/rau7x/?view_only=1464ed216f3f4c0b9fab6217014815dd)

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## ABSTRACT

**Introduction:** Previous research has shown blunted recovery of heart rate variability (HRV) following acute exercise in the heavy domain. It is unknown whether this occurs independent of heart rate changes or whether nonlinear HRV analysis methods, such as detrended fluctuation analysis (DFA  $\alpha 1$ ) and sample entropy (SampEn), show similar patterns. **Methods:** Ten distance runners (7 male, 3 female) of varying training statuses completed a graded exercise test to determine maximal aerobic capacity ( $\dot{V}O_{2MAX}$ ) and ventilatory thresholds. Participants completed two, one-hour runs in the moderate (MOD) or heavy (HVY) domain on separate days. Before and after exercise, heart rate (HR) and heart rate variability (HRV) were measured using a chest strap HR monitor, and blood was drawn to measure cortisol. The following day, participants completed high-intensity intervals and a 3,000m time trial. Linear mixed models were used to compare the effect of exercise domain on recovery before and after correcting for HR and the effect of exercise domain on endurance performance. **Results:** HVY delayed the recovery of linear HRV measures for the first 20 minutes after exercise with no differences at subsequent timepoints. Recovery of SampEn ( $p = 0.447$ ) and DFA  $\alpha 1$  ( $p = 0.064$ ) were not different between HVY and MOD. After correcting for recovery HR and other covariates, HVY still impaired linear measures of HRV. However, endurance performance and blood cortisol levels were not different between HVY and MOD ( $p > 0.05$ ). **Conclusions:** Nonlinear measures of HRV were not impaired to the same extent as linear measures following HVY exercise. Differences in recovery HRV between exercise domains are still significant after correcting for recovery HR. Further research is needed to better understand these findings in the context of chronic training and their effects on endurance performance.

Keywords: Heart rate correction, endurance performance, cortisol, exercise domain, ventilatory threshold

## INTRODUCTION

To maximize sport performance, endurance athletes undergo large training volumes, predominantly in the moderate domain, below the first ventilatory threshold<sup>1-3</sup>. Given these large training volumes, it is important for coaches, athletes, and scientists to understand how different exercise intensities affect athletes and their recovery. Invasive measures of stress, such as blood cortisol, show a clear and marked elevation following high-intensity exercise<sup>4,5</sup>. However, the minimal exercise intensity that increases blood cortisol has been reported to range anywhere from 60% to 80%  $VO_{2MAX}$ <sup>4,6,7</sup>. Heart rate variability (HRV) is a non-invasive measure of cardiac parasympathetic activity<sup>8</sup>, which has led to its use as a proxy for stress by endurance athletes and their coaches<sup>8</sup>. Unlike blood cortisol, post-exercise suppression of HRV (indicating higher stress) is consistently greater following exercise in the heavy domain in trained<sup>9,10</sup> and well-trained athletes<sup>11</sup>.

Following submaximal exercise, both heart rate (HR) and HRV return to baseline with a probable mechanistic link between their recoveries<sup>12</sup>. However, HR and HRV are not linearly related<sup>13,14</sup>, so the differences in post-exercise HRV between moderate and heavy domain exercise could be due to differences in HR itself rather than HRV<sup>15,16</sup>. Further, previous research has focused on time and frequency domain measures of HRV. Nonlinear measures of HRV, such as the alpha-1 exponent of detrended fluctuation analysis (DFA  $\alpha_1$ ) and sample entropy, could better capture the changes in cardiac parasympathetic activity in the dynamic

environment of exercise recovery<sup>17</sup>. Previous research applying DFA  $\alpha_1$  and sample entropy to exercise recovery shows acute suppression following exercise<sup>17,18</sup>, but these methods have yet to be applied to exercise recovery between exercise domains.

The primary aim of this study was to investigate the effect of HR correction on differences between moderate and heavy domain exercise on the recovery of HRV in endurance athletes through linear and non-linear measures. Secondary aims were to explore the effect of exercise domains on blood cortisol and next-day endurance performance, assessed by a high-intensity workout and 3,000m time trial. We hypothesized heavy domain exercise would lead to greater post-exercise suppression of linear and non-linear measures of HRV than moderate domain exercise, even after correcting for HR.

## **METHODS**

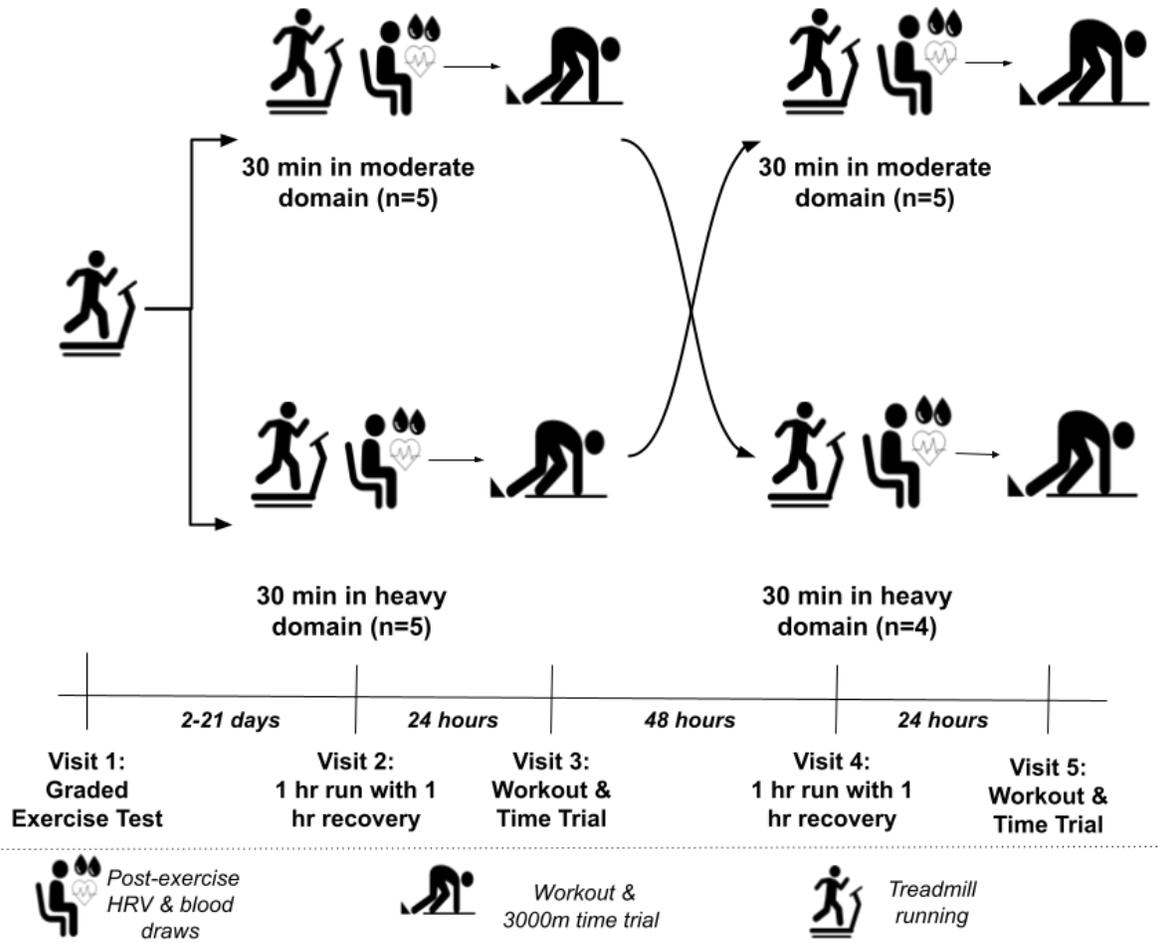
### **Study Design**

Participants visited the Human and Sport Performance lab (HSPL) at the University of Minnesota on five separate days (Figure 1). At the first visit, participants gave their informed consent and completed a graded exercise test to determine  $VO_{2MAX}$ , the first and second ventilatory thresholds (VT1 and VT2, respectively). Participants returned to the lab after three to twenty-one days for four more visits. Visits 2 and 4 consisted of one-hour runs followed by seated recovery for one hour. Visits 3 and 5 consisted of a brief workout followed by a fifteen-

minute break and a 3-km time trial. The exercise conditions for Visits 2 and 4 were completed in a randomized order with a crossover design.

A power analysis was conducted using the *simpr* package<sup>19</sup> within R (version 4.1.2, R Core Team; Vienna, Austria) to ensure 80% power at an alpha level of 0.05. To detect significant differences within 10 minutes after the end of exercise, an *a priori* analysis using estimations from Figure 1 in Seiler et al.<sup>11</sup> indicated eight individuals would be needed. This was based on estimations from their data in Figure 1, where there was a mean difference between exercise conditions of 60% of baseline morning HRV and a standard deviation of 35% at five minutes after exercise. The present study was not powered to detect significant differences in blood cortisol or endurance performance, so secondary aims were conducted in an exploratory manner. Therefore, ten endurance athletes (n=3 female) were recruited via printed or electronic flyer from the Twin Cities area to participate in this study.

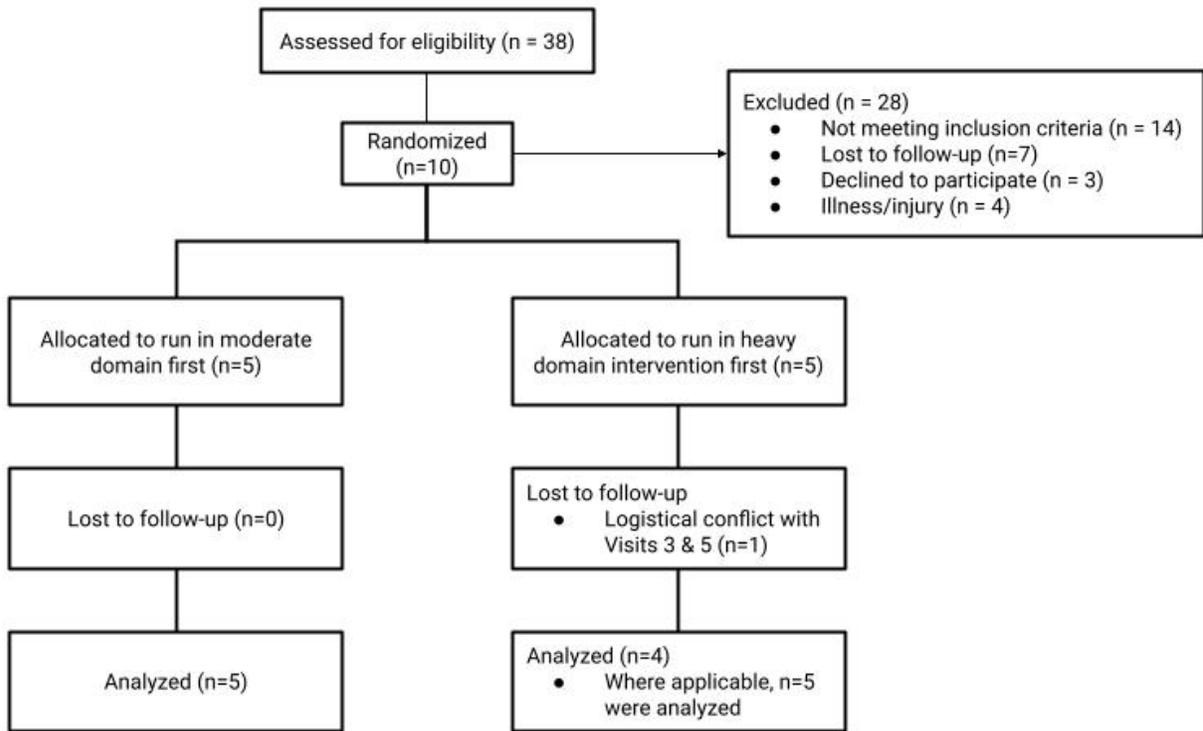
Figure 1: Experimental Design



## Participants

Participants were screened for inclusion criteria and training habits using a Qualtrics survey (Seattle, Washington, USA). Inclusion criteria included either an average of six or more hours of running or seven or more hours of aerobic exercise per week (swimming, cycling, etc.) with at least two hours of running. Participants were also required to have participated in at least one race between 1.5 km and 42.2 km in the last three months. Given the COVID-19 pandemic and limited race opportunities, self-reported time trial efforts were also considered. Female participants were naturally menstruating but were not tested to confirm a particular hormonal profile; all female participants self-reported eumenorrhea<sup>20</sup>. All participants were between 18 and 40 years old, free of chronic disease, had received at least two COVID-19 vaccine doses, and were not taking any medications known to alter autonomic function<sup>21</sup>. Throughout recruitment and testing, participants were blinded to treadmill speed and incline. Participants were not informed of previous research surrounding impaired HRV after exercise in the heavy domain, but they were told the study sought to characterize recovery after submaximal exercise. A CONSORT diagram for participant recruitment and allocation is included in Figure 2. Approval was obtained from the University of Minnesota Institutional Review Board prior to any recruitment (STUDY00013454).

Figure 2: CONSORT Flow Chart



## Graded exercise testing

Prior to arrival at the HSPL, participants were asked to refrain from strenuous exercise or nicotine/tobacco use for at least 24 hours and from caffeine use for at least 12 hours. After obtaining informed consent, participants were weighed via electronic scale (Etekcity Personal Scale; Anaheim, CA, USA), and height was measured via stadiometer (ACCUSTAT; Genentech, San Francisco, CA, USA). They were then fitted with a validated<sup>22</sup> chest-strap heart rate monitor (Polar H10; Polar Electro, Kempele, Finland) and neoprene face mask. Prior to each testing

session, the metabolic cart (Ultima cart; Medgraphics, Minneapolis, MN, USA) was calibrated with a three-liter syringe and standard gases.

Throughout testing, breath-by-breath expired gases were recorded using a metabolic cart and associated software (Breeze, 8.6.0.56; Medgraphics, St. Paul, MN, USA). Throughout testing, R-R intervals were recorded continuously using the chest strap heart rate monitor to a mobile app (Elite HRV; Austin, TX, USA), which has been validated for measuring HRV<sup>23,24</sup>. Briefly, the graded exercise testing protocol consisted of a five minute warmup at either 8.0-8.9 kph (females) or 9.7 kph (males) and a 1% incline<sup>25</sup>. Following a five-minute standing rest, participants completed a ramp treadmill test to volitional exhaustion (see *Supplemental Materials: Graded exercise testing*). Ventilatory thresholds were determined using a combination of visual and automated detection, as neither method has been shown to be superior on its own<sup>26</sup>. The times at VT1 and VT2 were adjusted using methods outlined by Keir et al.<sup>27</sup>, and the resulting speeds and heart rates at VT1 and VT2 were used to prescribe intensities for all subsequent visits.

## Experimental Visits

Visits 2 and 4 consisted of five minutes of walking and 55 minutes of running followed by one hour of seated recovery. Participants arrived in the morning after eating a small breakfast, which was recorded in a food log and standardized across their visits. Upon arrival,

participants were asked to void their bladder before sitting in a quiet room. A five-minute HRV measurement was completed using a chest strap HR monitor as described above. Following the HRV measurement, blood was drawn from the antecubital vein (see *Supplemental Materials: Blood collection and processing*). Participants were weighed using an electronic scale before fitting with a neoprene mask. Expired gases and R-R intervals were recorded continuously as described above. For the warmup stage, all participants walked at 3.0 mph on a treadmill for five minutes before running for 15 minutes at 1.0 mph less than the speed at VT1 ( $v_{VT1}$ ). At this point, the neoprene face mask was removed. For the next 30 minutes, participants completed one of two experimental conditions, and speed was adjusted to maintain a target HR. We used HR to adjust exercise intensity as it has been shown to track well with oxygen consumption<sup>28</sup> and is easier for coaches and athletes to measure. We did not measure oxygen consumption beyond the 20-minute warmup to maintain ecological validity and limit participant discomfort.

The target HR during the experimental condition was determined according to visit type. For the visit in the moderate domain, the target HR was 5-7 bpm less than the HR at VT1 ( $HR_{VT1}$ ). For the visit in the heavy domain, the target HR was at least two bpm above  $HR_{VT1}$  but less than the mean HR between  $HR_{VT1}$  and the HR at VT2 ( $HR_{VT2}$ ). Speed was adjusted as needed to maintain the target HR, and rating of perceived exertion (RPE) was assessed every

five minutes<sup>29</sup>. After 30 minutes had passed, the speed was decreased to 1.0 mph less than vT1 for a cool-down period. Additional decreases in speed were used as needed to match the HR during the warmup period. During the cool down, participants were given 150 mL of cold water to mitigate dehydration while limiting the effect of water intake on HRV<sup>30,31</sup>; all participants drank the water as requested.

At the end of exercise, participants were directed immediately to a quiet room. They were instructed to remain seated, breathe normally, and minimize movement for one hour while watching a documentary<sup>32</sup>. Blood draws were completed at 10 and 60 minutes after the end of exercise, as recommended by Daly et al.<sup>33</sup>. HRV was measured continuously throughout exercise and during recovery. There was a 48-hour washout period between Visit 3 and Visit 4. Participants were encouraged to maintain routine exercise habits during this time but to refrain from any strenuous exercise or low-intensity running longer than one hour.

### **Workout, Time Trial, and Home Measures**

Visits 3 and 5 were each completed the day after Visits 2 and 4, respectively, and they were done at the same time of day. Participants arrived at the lab postprandial after a small breakfast, which was the same for all visits. After voiding the bladder and sitting for at least three minutes, a five-minute HRV recording was obtained followed by a blood draw.

Participants were weighed before fitting with a neoprene face mask. They ran for five minutes

at 1.0 mph less than vVT1 before running three intervals at vVT2. Each interval was three minutes long and separated by three minutes of walking at 3.0 mph. After the last interval, participants ran for five minutes at 1.0 mph less than vVT1 to cool down. Data collection throughout the workout was the same as detailed above and included ventilatory measures, HR, and RR intervals. At the end of each stage, rating of perceived exertion (RPE) was recorded<sup>29</sup>. Following the completion of the workout, participants were given 15 minutes to consume water and complete desired warmup activities, which were standardized between visits.

The 3,000m time trial was completed on an indoor 200m track. Verbal encouragement was scripted and delivered by the same investigator at each visit. Lap splits were recorded every 200m and given to participants at 800m and 1600m. Due to the COVID-19 pandemic, some participants were required to wear face masks while running. Face masks were made by the same company and in the same style for each participant.

Participants also completed a five-minute HRV measurement from their homes upon waking using the EliteHRV app and a chest strap HR monitor, as described above. They also completed the Short Stress and Recovery Score (SRSS) questionnaire, an eight-item questionnaire suitable for daily monitoring<sup>34</sup>. These were each completed every morning,

starting two days before Visit 2 and continuing through one day after Visit 5 (see *Supplemental Materials: At-home measurements*).

## Data analysis

The RR intervals from before and after exercise and from home recordings were exported from EliteHRV and organized in R. RR intervals collected during a blood draw were excluded. The remaining RR intervals were then imported into Kubios (version 3.5.0, University of Kupio, Finland) for artifact correction and variability analysis. All resting and exercise recovery data were analyzed in five-minute segments, and each five-minute segment was analyzed in the time and nonlinear domains. These measures included heart rate (HR), root mean square of successive differences (RMSSD), standard deviation of NN intervals (SDNN), sample entropy (SampEn), and the short-term, alpha one exponent of detrended fluctuation analysis (DFA $\alpha$ 1). Heart rate measurements during exercise from Visits 2 – 5 were also processed in Kubios to obtain HR but were not used for variability analyses. Breath-by-breath data collected during the workout visit was averaged using mid-5-of-7 averaging. Due to the shorter stage length of the workout, only the last 30 seconds of each interval and recovery stage were averaged for analysis. At each stage, RER and VO $_2$  were recorded. Data collected during the recovery between intervals were excluded.

## Statistical Analysis

Unless otherwise stated, all data were analyzed in R (version 4.1.3). Prior to analysis, normality, homogeneity, and linearity were visually confirmed. A natural logarithm was used to correct deviations from normality in both resting and recovery RMSSD, recovery HR, cortisol concentrations, and RER during the workout. A natural logarithm did not correct the skew in DFA  $\alpha_1$  from recovery after exercise, so a cube root transformation was used instead. Cohen's  $f^2$  was used to measure the effect size for linear models, and Cohen's  $d$  was used for pairwise comparisons. The alpha level was set *a priori* at 0.05.

Linear mixed models were used to answer the research questions using the lmerTest package<sup>35</sup>. To account for the repeated-measures nature of the data, participant ID was included as a random effect, and models were constructed to determine the effect of exercise domain and time (recovery time, workout stage, etc.) on outcome variables of interest. Models were run with and without relevant covariates, which included recovery HR, resting HRV, participant sex, and  $VO_{2MAX}$ . Pairwise comparison using the Holm correction for multiple comparisons were used when applicable.<sup>36</sup> A paired t-test was used to compare time trial time between exercise conditions. For full details on statistical models, see *Supplemental Materials: Linear mixed models*.

## Results

Data are presented as mean  $\pm$  standard deviation. P values and effect sizes (Cohen's  $f^2$  or Cohen's  $d$ ) are included in parentheses following model coefficients or pairwise comparisons. Ten participants (n=3 female) completed the study (Table 1). Using the participant classification framework proposed by McKay et al.<sup>37</sup>, our participants were trained (n = 8) or highly trained (n=2), with three of the trained individuals achieving performances within 30% of the world record times.

We successfully manipulated exercise intensity to achieve the desired experimental condition during the hour run. There were no differences in measures of exercise intensity between visits during the warmup or cooldown stages (*Supplemental Materials: Validation of Experimental Condition*). As intended, exercise intensity was higher during heavy domain exercise for HR ( $161 \pm 10$  vs.  $140 \pm 11$  bpm;  $p < 0.001$ ;  $f^2 = 1.513$ ), RPE ( $13 \pm 2$  vs.  $11 \pm 3$ ;  $p = 0.001$ ;  $f^2 = 0.308$ ), and speed ( $8.0 \pm 1.4$  vs.  $6.5 \pm 1$  mph;  $p < 0.001$ ;  $f^2 = 1.416$ ).

Table 1: Participant Characteristics

	All Participants (n=10)	Females (n=3)	Males (n=7)
Age (yr)	28.3 ± 5.0	29.2 ± 8.6	27.9 ± 3.5
Height (cm)	173.9 ± 9.8	161.7 ± 8.6	179.1 ± 3.4
Weight (kg)	68 ± 10.2	56.7 ± 8.2	72.9 ± 6.6
BMI (kg*m <sup>-2</sup> )	22.4 ± 1.8	21.7 ± 2.3	22.7 ± 1.6
VO <sub>2</sub> MAX (mL/kg/min)	52.6 ± 7.8	44.4 ± 8.1	56.1 ± 4.5
VT1 (%VO <sub>2</sub> MAX)	69.3 ± 5.1	70.0 ± 4.8	69.0 ± 5.5
VT2 (%VO <sub>2</sub> MAX)	84.5 ± 6.0	84.5 ± 5.1	84.4 ± 6.7
Average 3,000m time (sec)	659.9 ± 101.3	767.3 ± 84.6	606.2 ± 56.2
Resting HR (bpm)	64.5 ± 8.3	71.9 ± 5.3	60.8 ± 7.1
Resting SDNN (ms)	62.0 ± 22.3	56.7 ± 25.8	64.6 ± 22.5
Resting RMSSD (ms)	67.7 ± 37.5	61.1 ± 46.9	71.0 ± 36.5
Resting Sample Entropy	1.60 ± 0.30	1.40 ± 0.40	1.70 ± 0.2
Resting DFA α1	1.03 ± 0.25	1.02 ± 0.42	1.03 ± 0.18

*Body mass index (BMI); maximal aerobic capacity (VO<sub>2</sub>MAX); first ventilatory threshold (VT1); second ventilatory threshold (VT2)*

## Recovery of HR, LnRMSSD, and SDNN

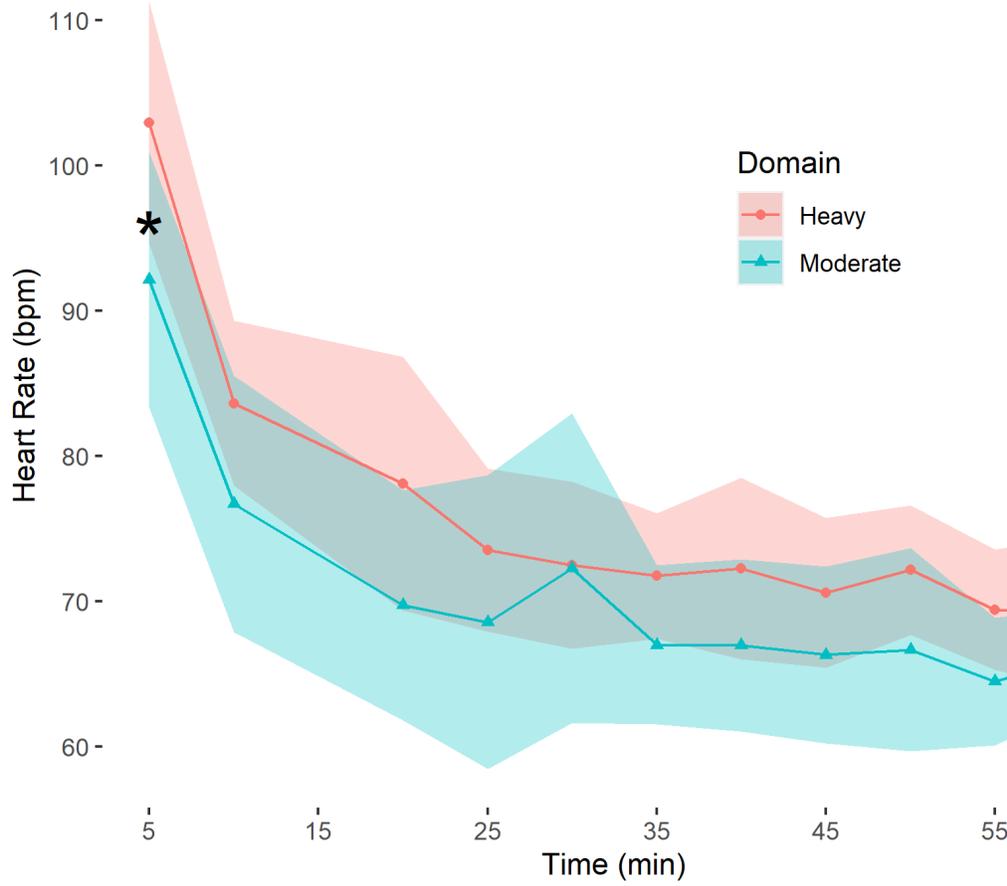
Recovery of HR had a large main effect for time ( $p < 0.001$ ;  $f^2 = 0.643$ ) and exercise domain ( $p = 0.014$ ;  $f^2 = 0.048$ ) but not their interaction ( $p = 0.572$ ;  $f^2 = 0.002$ ) (Figure 3 and Table 2). At five minutes post-exercise, pairwise comparisons indicated HR was higher after exercise in the heavy domain ( $p = 0.011$ ;  $d = 0.074$ ); no other timepoints were significantly different between exercise domains ( $p > 0.05$ ). When resting HR, sex, and  $VO_{2MAX}$  were included as covariates in the model, exercise domain still impacted recovery HR ( $p = 0.014$ ;  $f^2 = 0.048$ ). The following variables also significantly predicted recovery HR: time ( $p < 0.001$ ;  $f^2 = 0.658$ );  $VO_{2MAX}$  ( $p = 0.029$ ;  $f^2 = 0.774$ ); and resting HR ( $p = 0.007$ ;  $f^2 = 1.221$ ). Participant sex ( $p = 0.430$ ;  $f^2 = 0.068$ ) and the interaction between time and exercise domain ( $p = 0.566$ ;  $f^2 = 0.003$ ) were not significant predictors of recovery HR.

Recovery of LnRMSSD had significant main effects for time ( $p < 0.001$ ;  $f^2 = 0.500$ ), exercise domain ( $p < 0.001$ ;  $f^2 = 0.110$ ), and their interaction ( $p = 0.0485$ ;  $f^2 = 0.031$ ) (Figure 4 and Table 2). Pairwise comparisons showed LnRMSSD was higher after exercise in the moderate domain at five ( $p = 0.025$ ;  $d = 0.062$ ) and 20 ( $p = 0.015$ ;  $d = 0.070$ ) minutes after exercise. After addition of resting LnRMSSD, recovery HR, sex, and  $VO_{2MAX}$ , exercise domain ( $p = 0.005$ ;  $f^2 = 0.645$ ) and the interaction between time and exercise domain ( $p = 0.017$ ;  $f^2 = 0.046$ ) remained significant predictors of LnRMSSD during recovery. Resting LnRMSSD ( $p <$

0.001;  $f^2 = 2.413$ ) and recovery HR ( $p < 0.001$ ;  $f^2 = 1.234$ ) were also significant, while time ( $p = 0.143$ ;  $f^2 = 0.017$ ), sex ( $p = 0.114$ ;  $f^2 = 0.329$ ), and  $VO_{2MAX}$  ( $p = 0.632$ ;  $f^2 = 0.027$ ) were not significant in the second model.

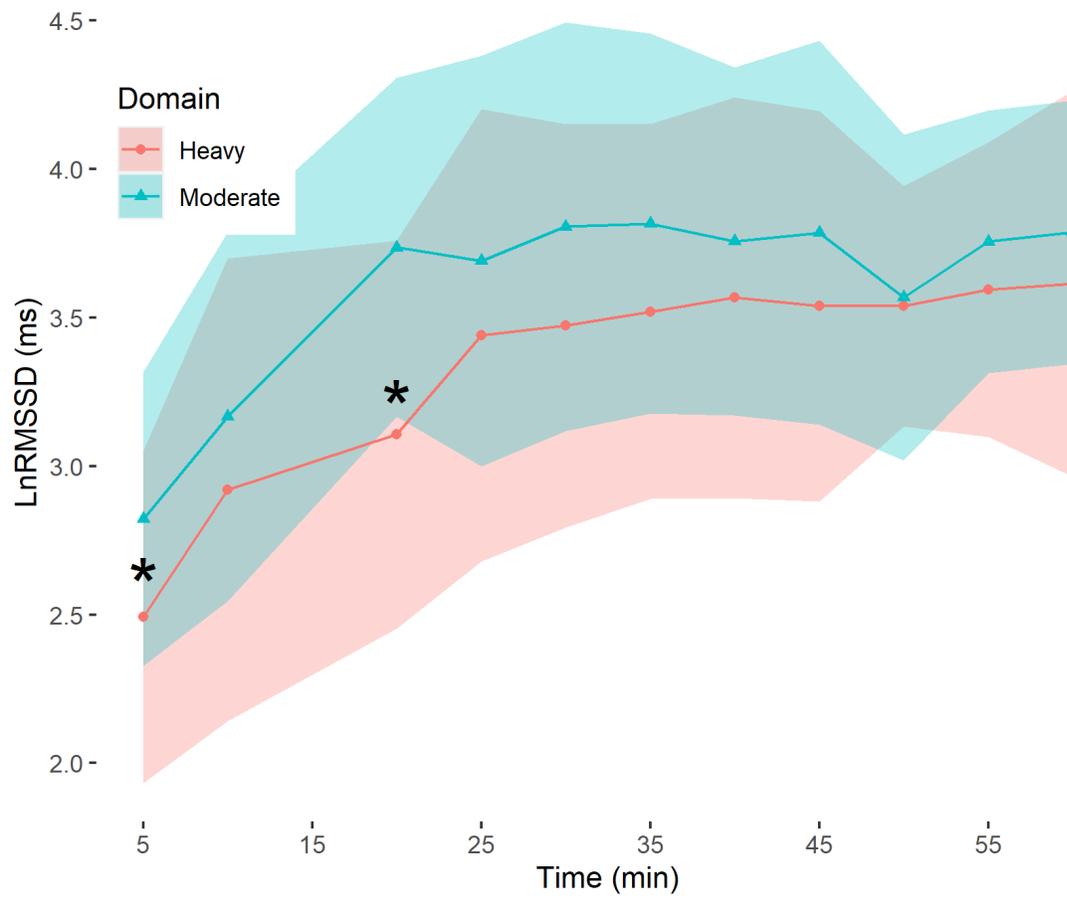
Similar results were observed for recovery of SDNN (Table 2 and Figure S1). There were significant main effects for time ( $p < 0.001$ ;  $f^2 = 0.307$ ), exercise domain ( $p = 0.003$ ;  $f^2 = 0.073$ ), and their interaction ( $p = 0.018$ ;  $f^2 = 0.045$ ). Pairwise comparisons showed no differences at any timepoints (all  $p > 0.065$ ). Exercise domain ( $p = 0.041$ ;  $f^2 = 0.033$ ) and the interaction between exercise domain and time ( $p = 0.013$ ;  $f^2 = 0.049$ ) remained significant after addition of sex,  $VO_{2MAX}$ , resting SDNN, and recovery HR. Resting SDNN ( $p = 0.016$ ;  $f^2 = 0.920$ ) and recovery HR ( $p < 0.001$ ;  $f^2 = 0.377$ ) were also significant predictors, while sex ( $p = 0.088$ ;  $f^2 = 0.400$ ), time ( $p = 0.174$ ;  $f^2 = 0.014$ ), and  $VO_{2MAX}$  ( $p = 0.951$ ;  $f^2 < 0.001$ ) were not.

Figure 3: Recovery of Heart Rate by Exercise Domain



\*  $p < 0.05$ ; means are shown with solid symbols with shading showing the 95% CI.

Figure 4: Recovery of LnRMSSD by Exercise Domain



\*  $p < 0.05$ ; means are shown with solid symbols with shading showing the 95% CI.

## Recovery of Nonlinear Measures

Exercise domain impacted nonlinear measures to a lesser extent during recovery (Table 3; Figures S2 and S3). While there was an effect of time on sample entropy ( $p < 0.001$ ;  $f^2 = 0.124$ ), there was no effect of exercise domain ( $p = 0.447$ ;  $f^2 = 0.004$ ) or an interaction between time and exercise domain ( $p = 0.586$ ;  $f^2 = 0.002$ ). After the addition of sex,  $VO_{2MAX}$ , resting sample entropy, and recovery HR, the effect of time was no longer significant ( $p = 0.302$ ;  $f^2 = 0.008$ ), and exercise domain ( $p = 0.276$ ;  $f^2 = 0.009$ ) and the time-by-domain interaction ( $p = 0.314$ ;  $f^2 = 0.008$ ) remained non-significant. Recovery HR was a significant covariate in the adjusted model ( $p < 0.001$ ;  $f^2 = 0.933$ ), while  $VO_{2MAX}$  ( $p = 0.478$ ;  $f^2 = 0.004$ ), resting sample entropy ( $p = 0.183$ ;  $f^2 = 0.014$ ), and sex ( $p = 0.311$ ;  $f^2 = 0.008$ ) were not.

There was a significant effect of time on DFA  $\alpha_1$  during recovery ( $p < 0.001$ ;  $f^2 = 0.198$ ) without a significant effect for exercise domain ( $p = 0.064$ ;  $f^2 = 0.027$ ) or an interaction between domain and time ( $p = 0.934$ ;  $f^2 < 0.001$ ). After adding sex,  $VO_{2MAX}$ , resting DFA  $\alpha_1$ , and recovery HR as covariates to the model, recovery HR had a small but significant effect on DFA  $\alpha_1$  during recovery ( $p < 0.001$ ;  $f^2 = 0.286$ ). Exercise domain ( $p = 0.393$ ;  $f^2 = 0.006$ ), time ( $p = 0.543$ ;  $f^2 = 0.003$ ), their interaction ( $p = 0.725$ ;  $f^2 = 0.001$ ), resting DFA  $\alpha_1$  ( $p = 0.072$ ;  $f^2 = 0.540$ ),  $VO_{2MAX}$  ( $p = 0.232$ ;  $f^2 = 0.204$ ), and participant sex ( $p = 0.421$ ;  $f^2 = 0.084$ ) were all non-significant. At 24 hours after exercise, there were no differences between the domains for HR

( $p = 0.553$ ;  $d = 0.082$ ), LnRMSSD ( $p = 0.937$ ;  $d = 0.217$ ), SDNN ( $p = 0.838$ ;  $d = 0.155$ ), sample entropy ( $p = 0.356$ ;  $d = -0.093$ ), or DFA  $\alpha_1$  ( $p = 0.311$ ;  $d = 0.678$ ).

*Table 2: Recovery of HR and Linear Measures of HRV At Select Timepoints*

	HR		LnRMSSD		SDNN	
Time	H	M	H	M	H	M
10 min	84 ± 6	77 ± 9	2.9 ± 0.8	3.2 ± 0.6	30.1 ± 14.6	34.9 ± 10.6
30 min	73 ± 6	72 ± 11	3.5 ± 0.7	3.8 ± 0.7	45.9 ± 20.3	53.4 ± 18.3
60 min	69 ± 5	66 ± 3	3.6 ± 0.7	3.8 ± 0.4	49.3 ± 23.5	53.9 ± 17.8
24 hours	66 ± 8	66 ± 7	3.9 ± 0.5	4.0 ± 0.4	53.3 ± 15.4	55.5 ± 15.1

*Statistical comparisons have been omitted from this table for brevity. Moderate (M); Heavy (H)*

*Table 3: Recovery of Nonlinear Measures of HRV At Select Timepoints*

	Sample Entropy		DFA $\alpha_1$	
Time	H	M	H	M
10 min	0.98 ± 0.35	1.12 ± 0.31	1.45 ± 0.28	1.38 ± 0.29
30 min	1.15 ± 0.22	1.08 ± 0.63	1.35 ± 0.38	1.17 ± 0.34
60 min	1.03 ± 0.24	1.12 ± 0.17	1.22 ± 0.42	1.17 ± 0.25
24 hours	1.58 ± 0.26	1.55 ± 0.31	1.08 ± 0.29	1.10 ± 0.25

*Moderate (M); Heavy (H)*

## Cortisol and Next-day Endurance Performance

There was no significant effect of exercise domain on blood cortisol ( $p = 0.206$ ;  $f^2 = 0.063$ ).

There was no difference between resting cortisol and cortisol measured at ten minutes ( $p = 0.467$ ;  $f^2 = 0.009$ ), one hour ( $p = 0.324$ ;  $f^2 = 0.039$ ), or 24 hours ( $p = 0.324$ ;  $f^2 = 0.042$ ) after exercise (Table S5). During the interval workout (visits 3 and 5), there was no effect of exercise domain on  $VO_2$  as a percentage of  $VO_{2MAX}$  ( $p = 1.000$ ;  $f^2 = 0.001$ ), RER ( $p = 1.000$ ;  $f^2 = 0.004$ ), HR ( $p = 1.000$ ;  $f^2 = 0.017$ ), or RPE ( $p = 0.824$ ;  $f^2 = 0.023$ ) (Tables S3 and S4). The time to complete a 3,000m time trial was not affected by exercise domain ( $p = 0.493$ ;  $d < 0.001$ ) (Figure S4). There was no effect of exercise domain ( $p = 1.000$ ;  $f^2 = 0.003$ ) or the interaction between lap number and exercise domain ( $p = 1.000$ ;  $f^2 < 0.001$ ) on the difference between each 200m lap split and the average lap time during the time trial. Measures of perceived stress and recovery were not statistically analyzed but are reported in the Supplemental Materials (Figures S5 and S6).

## Discussion

This study aimed to compare acute recovery and next-day endurance performance after exercise in the moderate and heavy domains. Our findings support the concept of lower cardiac parasympathetic activity immediately following exercise in the heavy domain, even after controlling for resting HRV and recovery HR. The present study suggests that exercise in the

heavy domain transiently delays the recovery of cardiac parasympathetic activity with minimal effects on next-day endurance performance or circulating cortisol.

The differences in post-exercise HR and HRV between exercise conditions align with the findings of previous studies. A previous study<sup>11</sup> with identical exercise durations in a similar population found HR and LnRMSSD were lower for 15 minutes after exercise above VT1 compared to exercise below VT1. Despite the lower  $VO_{2MAX}$  and lower training volume of participants in the present study, we found similar differences, and these differences extended through a comparable time course after exercise. Similarly, Parekh and Lee<sup>38</sup> reported lower SDNN after exercise at 80% of  $VO_2$  reserve compared with an isocaloric exercise bout at 50% of  $VO_2$  reserve; despite the slight differences in exercise intensity and duration, comparable findings were observed in the present study.

Michael et al.<sup>39</sup> also found differences in post-exercise LnRMSSD through 15 minutes when comparing exercise below VT1 with exercise bouts between VT1 and VT2 and above VT2. Notably, these changes were still present after dividing LnRMSSD by RR interval length, which has been proposed as one way to account for the effect of HR on HRV<sup>15,16</sup>. Our study validates these findings, as exercise domain impacted both SDNN and LnRMSSD after the addition of recovery HR to the linear models. Our study extends these differences through 20 minutes after exercise, where there was a difference in LnRMSSD but not HR. It should be noted that

the effect size for recovery HR on LnRMSSD during recovery was large ( $f^2 = 1.234$ ), reinforcing the importance of adjusting for HR when analyzing HRV.

Unlike the linear measures of HRV, nonlinear measures of HRV were not significantly affected by exercise domain. Nonlinear measures of HRV during recovery from exercise remain poorly understood and could follow a different pattern and time course compared to more traditional linear measures. Further, our power analysis did not consider these nonlinear measures, which have less previous research than time domain measures. Higher exercise intensities than the ones employed in this study do not seem to cause further suppression of nonlinear measures beyond the suppression already seen with low-intensity exercise<sup>18</sup>. The resting sample entropy of our participants is higher than has been reported for untrained college students<sup>18</sup>. Similarly, DFA  $\alpha_1$  values were higher at rest than in sedentary subjects before and after 8 weeks of endurance exercise training<sup>17</sup>. Exercise also suppressed sample entropy, which was not seen in a subset of active participants in a previous study<sup>40</sup>. Altogether, our participants demonstrate a high degree of cardiac complexity at rest, which is quickly restored after exercise.

Examination of the fractal behavior of their heart rate shows a signal with pink noise at rest and return to this state after 24 hours. A closer look at the acute recovery after exercise shows elevated DFA  $\alpha_1$  after exercise in the heavy domain, suggesting a more “Brownian” state

with less complexity. However, this was not significantly different from recovery after exercise in the moderate domain. Given the limited research on nonlinear measures of HRV after exercise and the small effect sizes compared to traditional linear measures, future research may require a larger sample size to observe significant differences compared to linear measures. Despite their purported advantage in analyzing nonstationary data, recovery HR significantly impacted both sample entropy and DFA  $\alpha_1$ . This aligns with previous studies showing the impact of RR interval length and nonstationary data on sample entropy and DFA  $\alpha_1$ <sup>41-43</sup>.

We found no significant effect of exercise domain on circulating cortisol. Previous studies have found a variety of relative intensities that may increase circulating cortisol. Increases in cortisol have been observed after 30-60 minutes of exercise at 60%<sup>44</sup>, 76%<sup>45</sup>, and 80% of  $VO_{2MAX}$ <sup>7</sup>, while others have shown no differences between exercise at 50% and 70% of  $VO_{2MAX}$ <sup>46</sup> or 65% and 80% of  $VO_{2MAX}$ <sup>47</sup>. Our findings suggest that exercise in the heavy domain does not necessarily increase circulating cortisol. However, direct comparison to previous studies is challenging given the absence of oxygen consumption data during exercise. Our sample size is also smaller than previous studies showing differences between intensities. As this was a secondary aim, our study was not sufficiently powered to detect cortisol differences between exercise domains. Given our small sample size, it remains unclear whether the

minimum intensity required to increase circulating cortisol is a percentage of  $VO_{2MAX}$  or a physiologic threshold (VT1).

Exercise in the heavy domain did not impair next-day endurance performance compared to exercise in the moderate domain. This is similar to a previous study<sup>10</sup>, which found no differences in RPE, RER, or  $VO_2$  in a submaximal running test the day after exercise in the moderate and heavy domains. We also found no evidence of impaired endurance performance or pacing changes following exercise in the heavy domain. Due to the nature of the study, we are unable to determine whether this is a physiologic adaptation due to exercise training or an inherent trait of these participants.

The role of exercise in the heavy domain for endurance athletes remains a subject of debate in the literature<sup>48,49</sup>. Some have argued for a polarized approach, suggesting that exercise in the severe domain is required to elicit enough stress to improve trained endurance athletes<sup>49</sup>. Others have argued for a pyramidal approach, which they state is more widely practiced by elite athletes with comparable efficacy to polarized training<sup>48</sup>. The null findings in this study suggest that exercise in the heavy domain may be well-tolerated by endurance athletes without negatively affecting next-day endurance performance, though caution is needed given the small sample size. This may support the advantages recently highlighted in the literature surrounding the success of pyramidal training for improving endurance

performance for runners<sup>50</sup> and half-Ironman triathletes<sup>51</sup>. However, neither study assessed HRV nor measures of the hypothalamic-pituitary-adrenal axis, limiting our ability to make direct comparisons.

Our study is not without limitations. The small overall sample size and limited number of female participants limits our ability to make comparisons between males and females. Thirty seconds of maximal cycling appears to perturb DFA  $\alpha_1$  during recovery more in women than men, and recovery of RMSSD occurred faster for men than women<sup>52</sup>. There is conflicting evidence as to whether differences in cardiac autonomic recovery after exercise between men and women are solely due to differences in  $VO_{2PEAK}$ <sup>53,54</sup>. Participants in the present study were within ten bpm of VT1 for both exercise bouts, which may limit our ability to ensure the entire bout was above VT1. The difference of ~20 bpm between conditions in the present study is smaller than what was used in the study by Seiler et al.<sup>11</sup>. Nevertheless, our main findings were comparable to theirs, and we still noted significant differences in HR, speed, and RPE between the exercise bouts.

## **Conclusion**

Our study contributes to the growing body of evidence that heavy domain exercise suppresses the acute reactivation of cardiac parasympathetic activity after exercise when linear measures of HRV are used, even after controlling for heart rate during recovery. We also show that circulating cortisol, physiologic markers of submaximal endurance performance, and maximal endurance performance are relatively unaffected by heavy domain exercise the previous day in trained runners. These findings may have implications for using pyramidal training, where exercise in the heavy domain would be emphasized. Studies with a larger sample size may be needed to better understand nonlinear measures of HRV after exercise. Further research involving chronic exercise in the heavy domain is required to confirm this application and to determine whether differences exist between males and females.

## **Contributions**

Contributed to conception and design: NAF, ASH, CJL

Contributed to acquisition of data: NAF, ASH

Contributed to analysis and interpretation of data: NAF, CJL

Drafted the article: NAF

Revised the article: ASH, CJL

Approved the submitted version for publication: NAF, ASH, CJL

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## **Data and Supplementary Material Accessibility**

Data, R scripts, and supplemental materials can be viewed here:

[https://osf.io/rau7x/?view\\_only=1464ed216f3f4c0b9fab6217014815dd](https://osf.io/rau7x/?view_only=1464ed216f3f4c0b9fab6217014815dd)

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