

Exercise tolerance through the severe and extreme intensity domains

by

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Abstract

Background and Aim: The power-duration relationship accurately predicts exercise tolerance for constant power exercise performed in the severe intensity domain. At intensities above critical power (CP), the power-duration relationship establishes a hyperbolic curve. However, the prediction of exercise tolerance is currently unclear for work rates within the extreme intensity domain (durations <2min). We hypothesized that the power-duration relationship deviates from a linear 1/time relationship for WRs within the extreme intensity domain.

Methods: Six men completed nine bilateral knee-extension tests on non-consecutive days and then performed 3 exercise tests in the severe intensity domain (S1-S3; $T_{lim} \geq 2-15$ min) and 4 in the extreme domain at 60%, 70%, 80%, and 90% 1RM ($T_{lim} < 2$ min), in random order. Twitch force (Q_{tw}), maximal voluntary contraction (MVC), and voluntary activation (VA) were measured on the right vastus lateralis before and after <80s) each test; EMG was measured on the right vastus lateralis throughout each test. T_{lim} were plotted as a function of 1/Time. T_{lim} for the extreme intensities were compared to the predicted T_{lim} of the slope of the S1-S3 regression.

Results: The r^2 for the severe domain 1/time model was 0.99 ± 0.007 . T_{lim} for exercise at 60% 1RM was not different than the predicted T_{lim} , however, T_{lim} for exercise at 70–90% 1RM was shorter than the predicted T_{lim} ($p < 0.05$). Post hoc analysis of the extreme domain (70–90% 1RM) revealed a significant linear relationship, suggesting a W' within the extreme domain (W'_{ext}). T_{lim} of exercise at 60% 1RM was not different from the predicted value of the 1/Time relationship of the extreme domain. Q_{tw} and MVC were significantly decreased following exercise at S1-S3 and 60% 1RM, while no changes existed in Q_{tw} or MVC following exercise at 80 and 90% 1RM. Further, no changes were found in VA following any exercise intensity.

Conclusion: These data suggest that exercise tolerance in the extreme domain is limited by different factors than in the severe domain. However, there is a separate but measurable W'_{ext} . Further, the factors limiting exercise in the extreme domain must be those from can be recovered by the time post-exercise measurements were made.

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Chapter 1 - Introduction

Within the severe-intensity domain, the power/duration relationship exhibits a hyperbolic relationship, with task failure occurring much sooner at higher compared to lower intensities. (26, 28, 35). The asymptote of this relationship has been termed critical power (CP), and represents the lower limit of the severe intensity domain (6, 25, 26, 35, 36). The curvature constant of the power-duration relationship has been termed W' and represents a finite work capacity above CP (26, 28), which allows exercise tolerance (T_{lim}) in the severe domain to be highly predictable. Though the exact mechanisms limiting W' still remain elusive, W' has been associated with the depletion of intramuscular energy stores, such as phosphocreatine (PCr) (19, 23), glycogen (24), and energy stores, and the accumulation of metabolites, including inorganic phosphate (Pi) and hydrogen ions (H^+) (10, 18, 19, 36). Due to the hyperbolic nature, this relationship can be expressed using a linear model by plotting intensity as a function of exercise tolerance (T_{lim}) as $1/T_{lim}$, where the Y-intercept is CP and the slope of the regression is W' .

The relationship between exercise intensity and time to exhaustion (T_{lim}) has been extensively studied in an attempt to identify the mechanisms of exercise intolerance (3-5, 8, 12, 34). In order to estimate the location of the fatigue during exercise, researchers have investigated central (i.e. proximal to the neuromuscular junction) and peripheral (i.e. distal to the neuromuscular junction) fatigue (3, 4, 9, 20). Following sustained maximal contractions, central fatigue may account for only up to 30% of force decline (4, 20), suggesting intramuscular milieu (i.e. peripheral fatigue) is largely responsible for maximal exercise tolerance. Further, Bigland-Ritchie (3) found the decline of MVC force following isometric voluntary contractions at 50 % maximal voluntary contraction (MVC) was primarily caused by peripheral fatigue with little to

no central fatigue present (3). This is consistent with the work of Yoon *et al.* (37), who showed that though central fatigue was minimal during exercise at 80% MVC, it appeared to be responsible for the force decline during exercise at 20% MVC. Burnley (11) suggested that CP may represent a threshold in which central fatigue was responsible for T_{lim} below CP, while peripheral fatigue was largely responsible for T_{lim} during intensities above CP. This could be due to the ability to reach a steady-state, where metabolite clearance (e.g. Pi, H^+ , Lactate, etc.) matches metabolite production below CP, whereas metabolite production outweighs metabolite clearance above CP ultimately leading to exercise cessation.

Historically, 3-4 bouts of exercise eliciting T_{lim} ranging from 2 – 20 min have been used to determine CP and therefore the lower boundary of the severe intensity domain (6, 15, 28). Further, the severe intensity is unique in that it is the only exercise domain within which $\dot{V}O_{2max}$ can be reached, as $\dot{V}O_2$ will reach steady-state at lower intensities (18). Additionally, Hill *et al* (18) recognized the need for a supra-severe exercise domain, where exercise intensity was so great that $\dot{V}O_{2max}$ could not be reached before exhaustion and coined it the ‘extreme’ domain, where T_{lim} would typically be reached in less than ~ 2 min. Previously, exhaustive exercises with high resistance, low repetitions (~75-85% 1RM) have been shown to better predict one repetition maximum (1RM) than low resistance, high repetition (~20-60% 1RM) exercises for untrained populations as well as specific athletes (13, 29). Interestingly, during these high-resistance, low-repetitions exercises, exhaustion is reached in less than ~ 2 min and elicits a curve similar to that observed in the severe intensity domain (13, 29), where the power/duration relationship has been characterized using a number of exponential equations (13). Whether or not the T_{lim} of the severe intensity domain predicts the T_{lim} of the extreme intensity domain has yet to be investigated. If the extreme domain does indeed exhibit a

relationship as that of the severe domain, it would suggest that T_{lim} within the extreme domain may be dictated by similar mechanisms. It has been suspected that there is an inherent change of fatiguing profiles occurring in the extreme domain compared to the severe intensity domain which is associated with exercise cessation before reaching $\dot{V}O_{2max}$ (18).

Therefore, the first aim of the current study was to determine and compare the power/duration relationship throughout the severe and extreme intensity domains. The second aim was to quantify the relative contributions of peripheral and central fatigue throughout these intensity domains. We hypothesized that during dynamic knee extension exercise 1) T_{lim} for a given force or power in the extreme intensity domain (T_{lim} less than 2 min) would be shorter than predicted by the severe intensity power/duration relationship, 2) throughout the severe and extreme domains, the contribution of peripheral fatigue would change with intensity, whereas 3) the contribution of central fatigue would be constant and not different following exercise.

Chapter 2 - Methods

Subjects: Six healthy men (mean \pm S.D.; 22.0 \pm 3.1 yr; 72.5 \pm 6.5 kg; 178 \pm 2 cm) participated in this study. All participants were free from cardiovascular, pulmonary, and metabolic disease as determined by a medical history questionnaire. Prior to participation in this study, subjects were informed of all procedures, and associated potential risks and benefits. Written informed consent was obtained from all participants prior to participation. Subjects were instructed to refrain from vigorous exercise 24 hours, alcohol consumption 12 hours, and food and caffeine 2 hours prior to each session. Subjects were instructed to maintain current exercise habits in order to avoid any training or detraining effect. All research components were reviewed and approved by the Institutional Review Board of Human Subjects at Kansas State University, Manhattan, KS.

Experimental Design: Subjects visited the laboratory a minimum of 9 sessions, with at least 48 hours between sessions. Subjects were first familiarized to all testing procedures and equipment prior to testing. All exercise tests were performed on an iso-lateral knee extension machine (MTSLE Iso-Lateral Leg Extension Machine, LifeFitness, Rosemont, IL, USA) that was customized to perform traditional bilateral knee extension exercise. The subjects were seated on the knee extension machine with a hip flexion angle of 90° and a knee flexion angle of 80°. Seat position was replicated for each exercise session. Following familiarization, the first session consisted of a 1RM test, defined as the heaviest weight lifted through the entire pre-determined range of motion (75°). The subjects began with a warm-up of 8 to 10 repetitions at 9.09 kg. Initial weight was selected following feedback pertaining to exercise history. Each subsequent increment was selected based on the rating of perceived exertion of the subject. The minimum

increase in weight was 4.54 kg due to the design of the exercise machine. No more than 2 attempts were allowed at a single resistance. A minimum of 5 minutes of rest was required between each attempt (22, 29).

In order to make comparisons to peak power (P_{peak}), the subjects performed a conventional incremental resistance test (6) to volitional fatigue to determine P_{peak} during the second visit to the laboratory. Subjects performed knee extension exercise at a 50% duty cycle (1.5 s contraction; 1.5 s relaxation) to the cadence of a pre-recorded audio cue. Initial resistance was set to 9.09 kg and was increased by 4.54 kg every minute until task failure. Subjects paused the contraction cycle during the last 5 s of each minute to allow resistance to be increased. Task failure was determined when the subject failed to maintain contraction pace or complete the full range of motion for 3 consecutive contractions.

During each subsequent visit, subjects performed constant-load tests to T_{lim} at 60, 70, 80, and 90% 1RM on separate visits and in random order. Following the tests on subsequent days, subjects performed a minimum of 3 constant-load exercise tests (S1, S2, and S3) at intensities predicted to elicit a T_{lim} of 2-15 min, on separate days and in random order. T_{lim} of S1 - S3 were then used to calculate CP via the power-duration relationship. If exercise continued for >20 min, the exercise intensity was considered to be below CP, the test was terminated, and the subject returned after 48 h to perform a test at a higher work rate. Power was found using the equation:

$$P = (W*d) / \text{Contraction Time}$$

where 'w' is the product of the mass of the resistance (kg) and the gravity constant (i.e. 9.8 m/s^2), 'd' is the fixed distance that the weight moved (0.345 m), and 'Contraction Time' was the total time from beginning of one contraction to the next consecutive contraction.

Neuromuscular Function: Neuromuscular function testing was conducted similar to previous protocols used in our laboratory (7) on the right leg prior to and following each constant load exercise test. The right ankle was secured to a force transducer (LBG1, BLH Electronics, Waltham, MA, USA). Ankle height was adjusted for each subject such that a 90° angle of pull was maintained. The height was recorded and replicated for all future sessions. Force was sampled at 1000 Hz and displayed on a computer screen (LabVIEW, National Instruments, Austin, TX, USA). The vastus lateralis was stimulated using a high-voltage constant-current electrical stimulator (DS7AH, Digitimer, Welwyn Garden City, UK). Paired stimuli (doublets) were delivered at 400 V with 100 μ s square-wave pulse durations and a 10 ms pulse interval. Adhesive electrodes (4 x 6 cm) were used to electrically stimulate the right vastus lateralis via the femoral nerve. The anode was attached to the gluteal fold and the cathode was positioned over the approximate location of the femoral nerve (2), located by palpation of the femoral artery proximal to the femoral artery bifurcation. Before beginning each exercise protocol, the placement of the cathode that produced the greatest force development with electrical stimulation was determined and used for pre- and post-exercise testing. Maximal stimulation was assessed prior to each exercise bout. Stimulation intensity was initiated at 50 mA and was increased in 25 mA increments until the measured force and compound muscle action potential (M-wave) no longer increased. The stimulator current was then increased an additional 30% to ensure the stimuli were supramaximal. Prior to each exercise test, subjects performed a series of 6, 3 s maximal voluntary contractions (MVCs), beginning every 30 s. Doublet muscle stimulations were delivered 5 s prior to each MVC, 1.5 s into the MVC, and 5 s after each MVC to obtain measurements of unpotentiated, superimposed, and potentiated doublet forces, respectively. MVC was determined as the greatest force attained prior to the superimposed muscle doublet

stimulation. This neuromuscular assessment was completed a second time < 80 s following exercise cessation.

Electromyography: Surface EMG measurements were obtained during each session using a commercially available system (Trigno EMG, Delsys Inc., Boston, MA, USA). Each EMG sensor contained four electrodes (5 x 1 mm) arranged in a 2 x 2 orientation to make single differential measurements. The belly of the right vastus lateralis was identified and placement of the sensor was marked with indelible ink to ensure repeatability of placement. The sensor was secured using an adhesive film. The EMG data were collected at a sampling rate of 1,000 Hz and band-pass filtered (13-400 Hz) using a fifth-order Butterworth filter. The EMG signal corresponding to each muscle contraction was detected using previously developed (in house) software (MATLAB R2011a, The Mathworks, Natick, MA, USA). The amplitude characteristics were described using the root mean squared (RMS) to provide an index of muscle activation and motorneuron firing rate. The frequency characteristics were described via median power frequency (MedPF) to provide an index of the muscle action potential conduction velocity. The EMG data were analyzed using binned averages of 3 contractions.

Statistical Analysis: The traditional CP and W' were determined by a linear regression using the $1/T_{lim}$ of S1 – S3 as a function of resistance (kg). Actual T_{lim} for 60 – 90% 1RM were then compared to the T_{lim} predicted by the severe intensity regression. In addition, a linear regression was applied to $1/T_{lim}$ for the 70 – 90% 1RM work rates. Actual T_{lim} for 60% 1RM, and S1 – S3 were compared to the T_{lim} predicted by the second linear regression. A two-way ANOVA with repeated measures was used to test for differences in time of contraction using EMG burst length

time across all work rates and between the average of the first 5 contractions compared to the average of the last 5 contractions (Intensity & First/Last). A two-way ANOVA was used to test for day-to-day differences in the pre-exercise value of the MVC (Intensity & Day). A one-way ANOVA with repeated measures (Fisher LSD) was used to test the change from baseline among intensities for Q_{tw} , MVC, and %VA. Differences were considered statistically significant when $p < 0.05$. Data were reported as means \pm standard deviation unless otherwise noted.

Chapter 3 - Results

There were no significant day-to-day differences in MVC ($p=0.13$). The mean pre-exercise coefficient of variation for MVC was 11.1 with a range of 7.4 – 18.8. MVC was $44.2 \pm 15.7\%$ 1RM (MVC measured on single leg knee extension; 1RM was performed as bilateral knee extension). 1RM was 108 ± 21 kg. P_{peak} was 45.5 ± 5.8 kg. S1 was $44.3 \pm 6.9\%$ 1RM, S2 was $33.5 \pm 4.4\%$ 1RM, and S3 was $25.8 \pm 2.7\%$ 1RM. The average EMG burst time of the first 5 contractions was not different from the average EMG burst time of the last 5 contractions for any subject ($p=0.46$), and was independent of intensity ($p=0.38$). Overall contraction time, and thus duty cycle, constant across work rates within each subject i.e., within each subject, there was a constant relationship between force and power. We chose to present the data as power.

T_{lim} for Severe and Extreme Domains: Mean \pm SD T_{lim} for each intensity is shown in Figure 1, while Table 1 provides the values. For each subject, $1/T_{\text{lim}}$ for each severe intensity (S1 - S3) was plotted as a function of resistance to estimate CP (y-intercept) and W' (slope of regression). CP was 19.6 ± 2.6 kg ($18.5 \pm 3.7\%$ 1RM; $43.4 \pm 7.2\%$ P_{peak}), while W' was 42.9 ± 10.8 kJ. T_{lim} 60% 1RM (85.0 ± 17.7 s) was not statistically different ($p=0.39$) from the T_{lim} predicted from the severe domain (100.5 ± 12.3 s) 1/Time model (Figure 1); however, T_{lim} for 70 – 90% 1RM were significantly shorter ($p<0.05$) than that predicted from the 1/Time model. Post hoc analysis of the extreme domain (70 - 90% 1RM) revealed a significant linear relationship between resistance and T_{lim} ($r^2 = 0.99$), with a W' of the extreme domain (W'_{ext}) of 10.4 ± 3.3 kJ. T_{lim} of 60% 1RM (85.0 ± 17.7 s) was not significantly different ($p=0.47$) from the $1/T_{\text{lim}}$ predicted from the

extreme domain 1/Time model (85.0 ± 16.2 s), while T_{lim} of S1 – S3 could not be predicted from the extreme domain 1/Time model.

Neuromuscular Function: The post-exercise changes in potentiated twitch force (Q_{tw}) are shown in Figure 2 for each intensity. The Q_{tw} significantly decreased following each severe intensity exercise test (S1 – S3) ($p < 0.01$). In addition, Q_{tw} declined following exercise at 60% 1RM and 70% 1RM ($p < 0.01$). However, there was no significant decline in Q_{tw} following exercise at 80% or 90% 1RM ($p > 0.05$), suggesting that task failure was not the result of peripheral factors at these intensities. Across intensities, the decrease in Q_{tw} was significantly less following exercise at 80 and 90% 1RM when compared to Q_{tw} following S1 - S3 and 60% 1RM ($p < 0.01$). In turn, the decline of Q_{tw} following exercise at 70% 1RM was significantly different from the decline of Q_{tw} following S1 and S2 intensities ($p < 0.01$).

Changes in MVC post-exercise for each intensity are shown in Figure 3. MVC decreased significantly following S1 – S3 and 60% 1RM ($p < 0.05$), however, the decrease in MVC following S2 was significantly different from all other intensities ($p < 0.05$). MVC did not change post-exercise ($p = 0.76$) following exercise at 70, 80, or 90% 1RM. Further, MVC following S1-S3 and 60% reached significantly lower values ($p < 0.05$) than MVC following 70 – 90% 1RM. Figure 4 illustrates post-exercise values of % VA for each intensity. The % VA showed no significant change pre- to post-exercise across intensities.

Root mean square (RMS) and Median power frequency (MedPF) responses during each exercise test are shown in Figures 5 & 6; respectively. RMS increased and MedPF decreased throughout each intensity without a plateau ($p < 0.01$). End exercise RMS following 90% 1RM

and S1 were different from end exercise S3 ($p < 0.01$). End exercise MedPF was not different among tests.

	Resistance (kg)			T_{lim} (s)		
	Mean	±	SD	Mean	±	SD
90% 1RM	97.7	±	19.2	27.1	±	5.8
80% 1RM	87.1	±	15.9	36.6	±	9.0
70% 1RM	75.8	±	15.4	54.6	±	13.3
60% 1RM	64.4	±	13.3	85.0	±	17.7
S1	47.7	±	8.5	154	±	29
S2	36.4	±	6.4	269	±	38
S3	28.0	±	4.5	468	±	86

Table 1: Times to Task Failure

Mean ± SD resistance and time to task failure (T_{lim}) for all intensities. S1 – S3 = severe intensity exercises ($T_{lim} = > 2$ min).

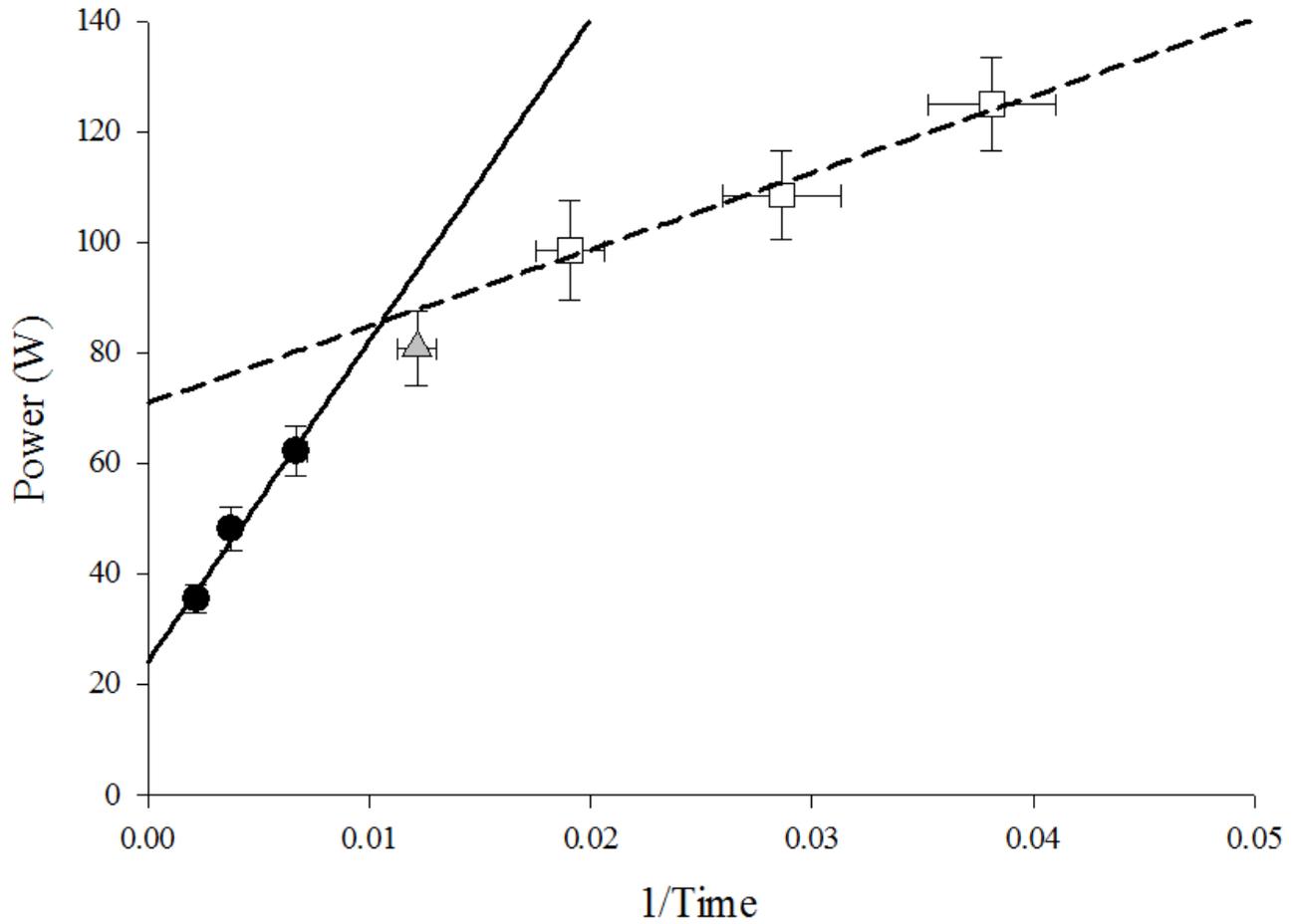


Figure 1: Severe and Extreme Power-Duration Relationships

Severe and extreme intensity power/duration relationships shown by linear transformation. S1 – S3 (●) regression shown by solid line. 70% – 90% 1RM (□) regression shown by dashed line. T_{lim} of 60% 1RM (▲) was not significantly different from the T_{lim} predicted by either S1 – S3 regression ($p=0.39$) or 70% - 90% 1RM regression ($p=0.47$).

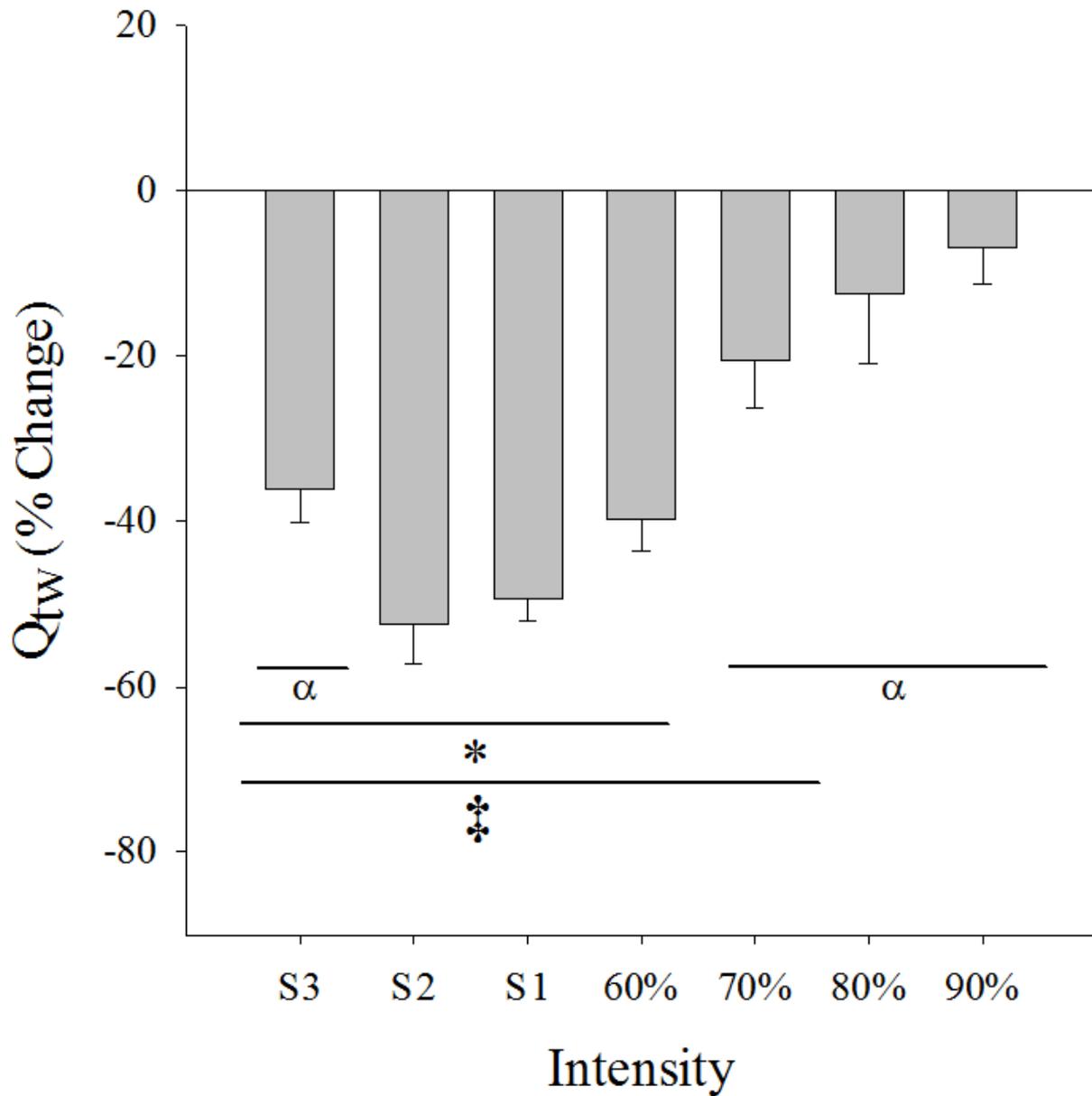


Figure 2: Twitch Force Pre- to Post- Exercise

Percent change in potentiated twitch (Q_{tw}) post-exercise for all intensities (Mean \pm SE). α = different from S2 ($p < 0.01$); * = different from 70%, 80%, and 90% 1RM ($p < 0.01$); ‡ = different from zero ($p < 0.01$). Q_{tw} following exercise at 80% and 90% did not significantly decrease ($p = 0.34$).

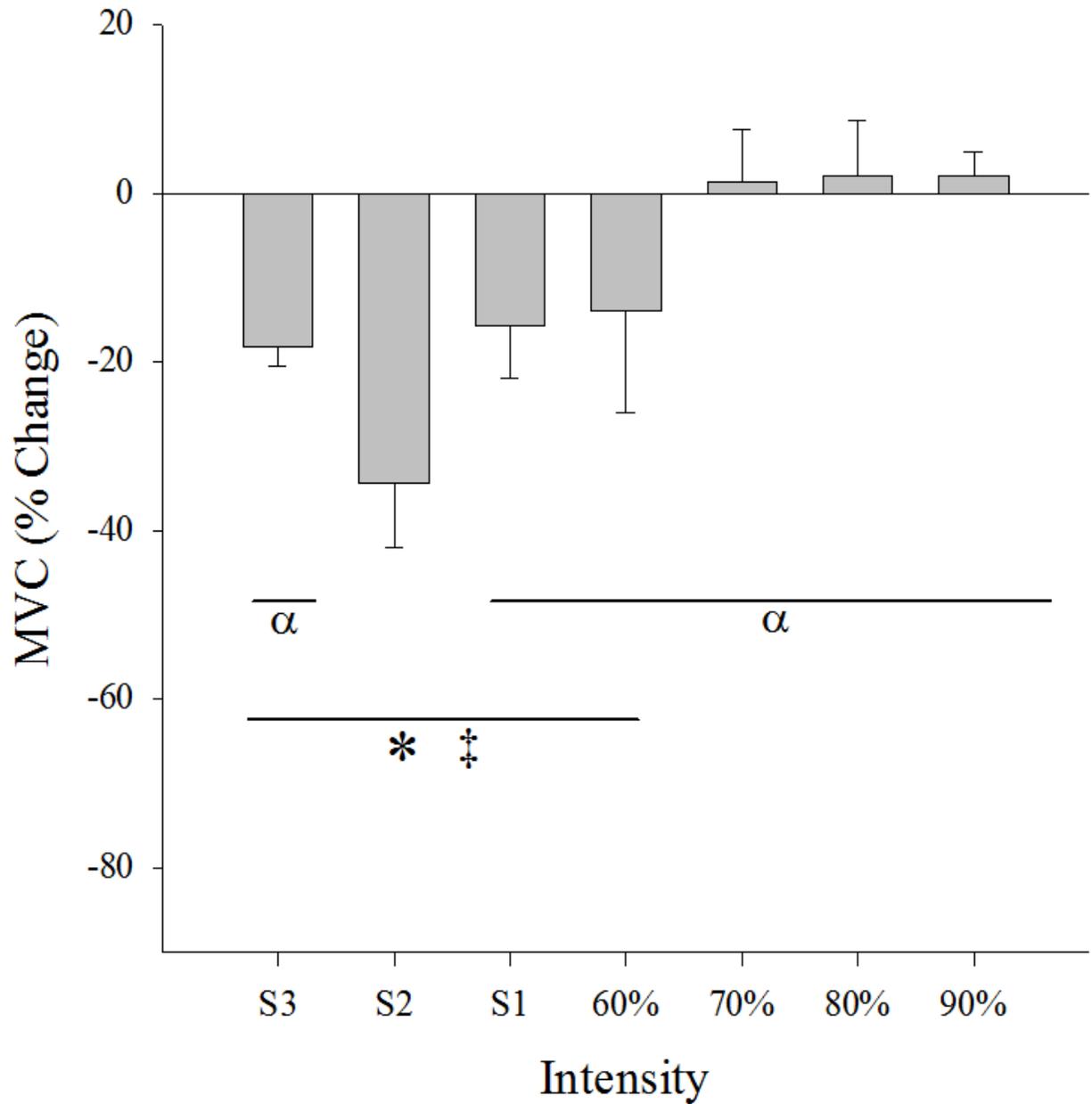


Figure 3: Maximal Voluntary Contraction Pre- to Post- Exercise

Percent change in maximal voluntary contraction (MVC) post-exercise for all intensities (Mean \pm SE). α = different from S2 ($p < 0.05$); * = different from 70%, 80%, and 90% 1RM ($p < 0.05$); ‡ = different from zero ($p < 0.05$). MVC following exercise at 70%, 80%, and 90% did not significantly decrease ($p = 0.76$).

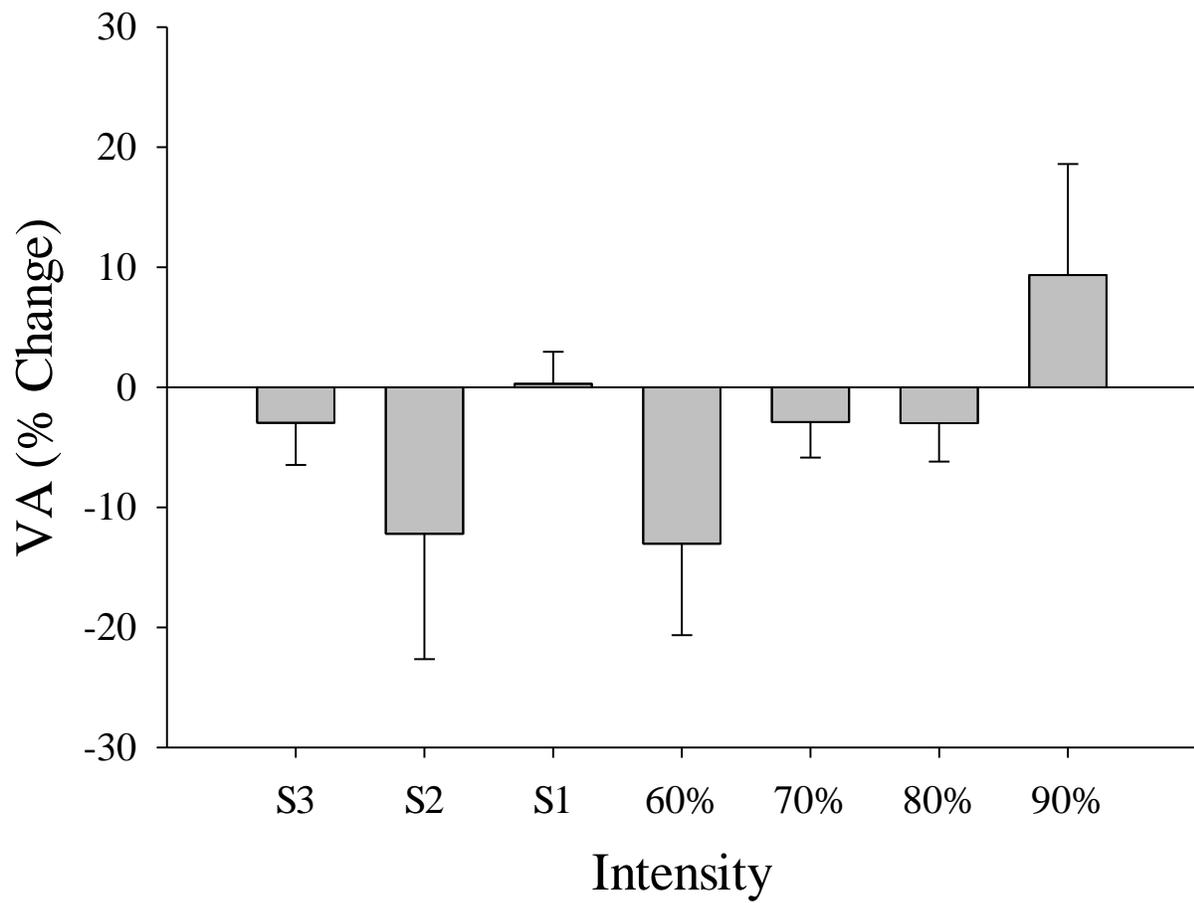


Figure 4: Voluntary Activation Pre- to Post- Exercise

Percent change in voluntary activation (VA) post-exercise for all intensities (Mean \pm SE). No differences were detected in the percent change pre- to post- exercise or the percent change among intensities.

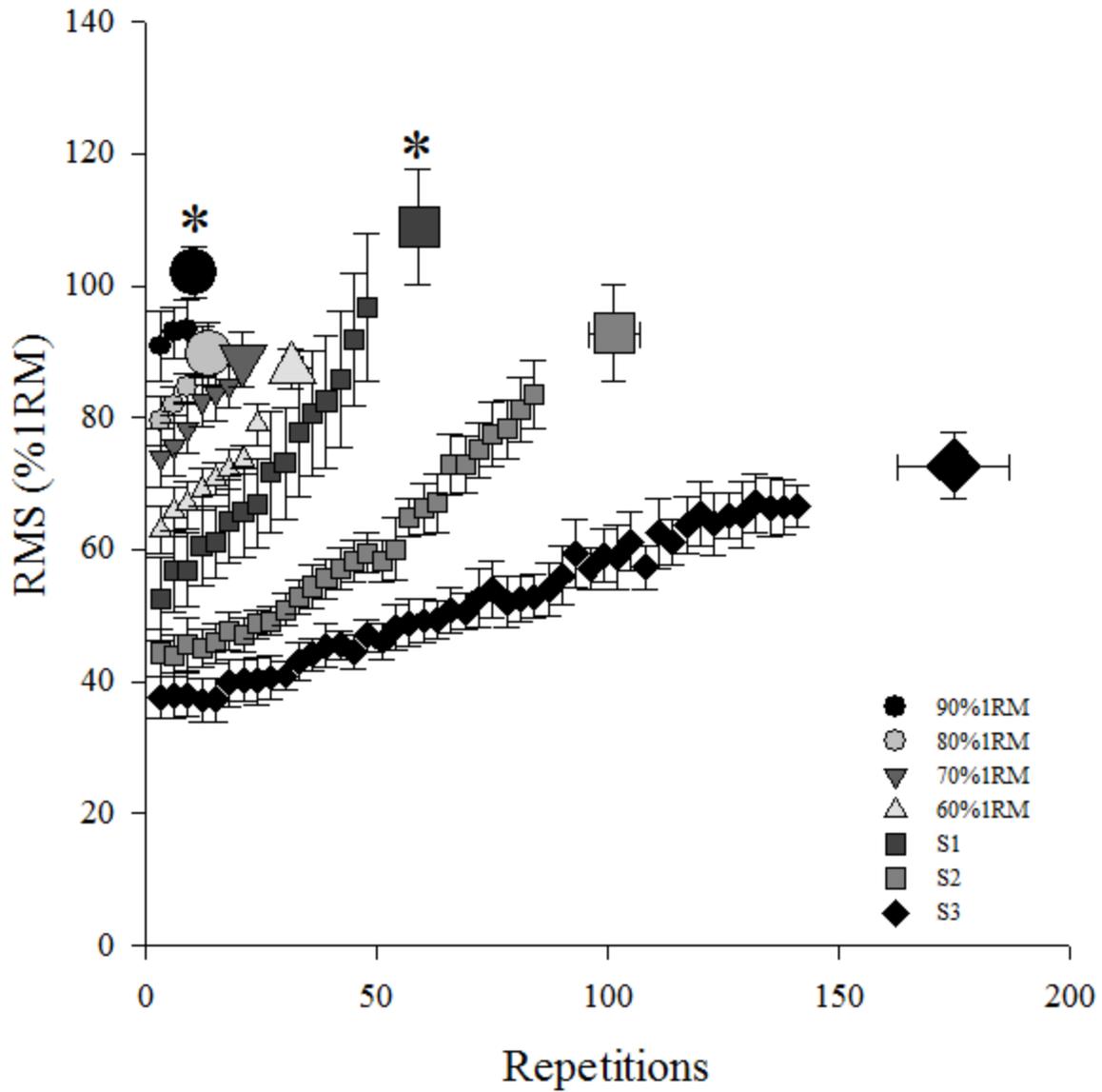


Figure 5: Root Mean Square throughout Exercise

Changes in root mean square (RMS) as a percent of 1RM throughout each intensity and at end exercise. Data were averaged into 3 contraction bins. RMS increased throughout each intensity so that end exercise was significantly greater than at the beginning of exercise ($p < 0.05$). * = end exercise different from S3.

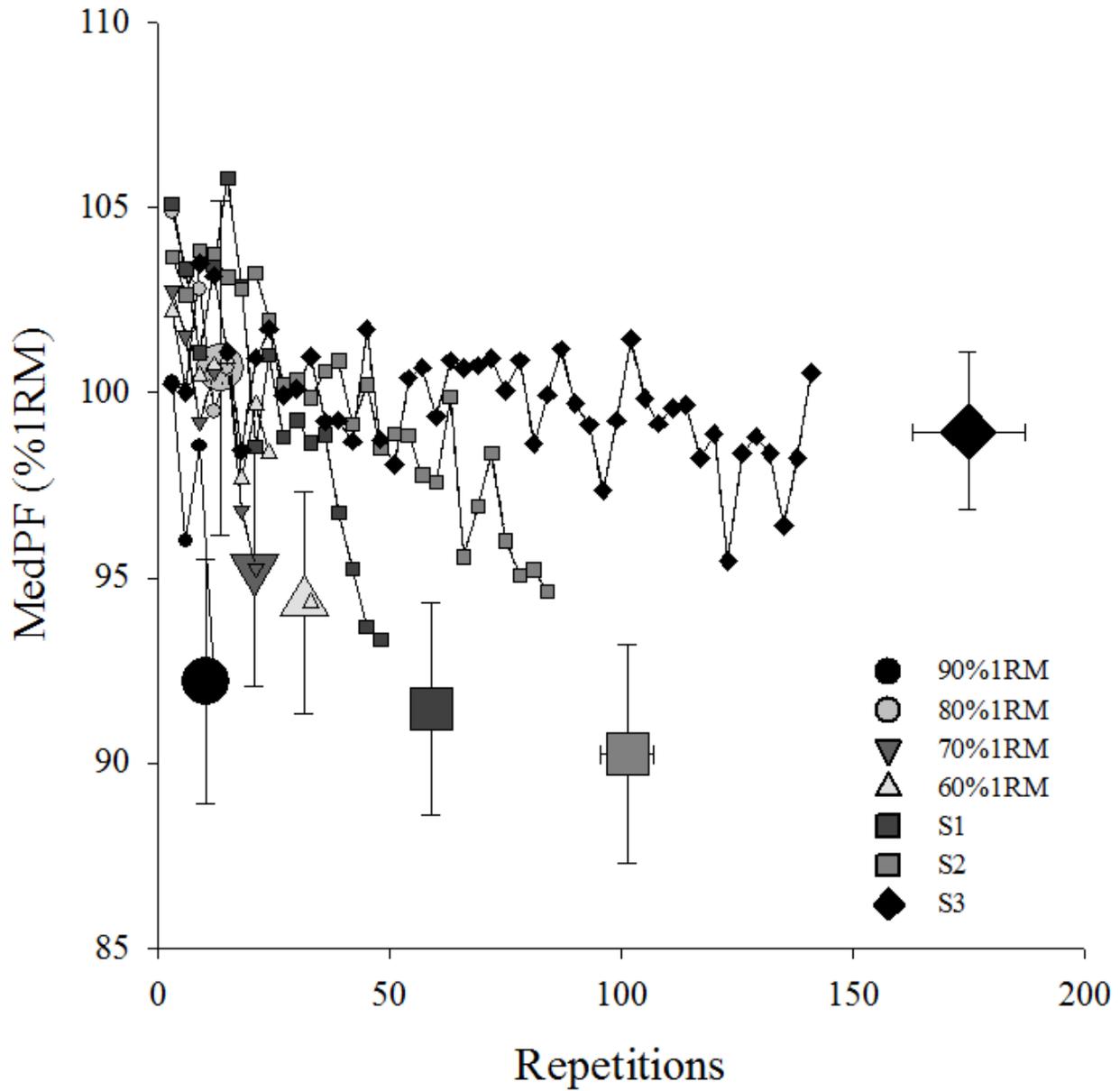


Figure 6: Median Power Frequency throughout Exercise

Changes in median power frequency (MedPF) as a percent of 1RM throughout each intensity and at end exercise. Data were averaged into 3 contraction bins. MedPF decreased throughout each intensity so that end exercise was significantly less than at the beginning of exercise ($p < 0.05$). There were no differences among tests at end exercise.

Chapter 4 - Discussion

Consistent with our hypotheses, the major findings of this study were 1) that T_{lim} within the extreme intensity domain were not predicted via the $1/Time$ linear regression model from the severe intensity T_{lim} , 2) peripheral fatigue was intensity-dependent, while 3) central fatigue did not appear to be a limiting factor to exercise tolerance in either the severe or extreme domain. Further, we found that EMG reached similar maximal (RMS) or minimum (MedPF) values between intensities above CP, independent of intensity above CP.

Tlim Prediction: Time to task failure as a function of work rate or power in the severe intensity domain has been shown to have a hyperbolic relationship or a linear relationship when plotted as a function of $1/Time$ (6, 25, 26, 28, 35, 36). This suggests that the mechanisms of fatigue limiting exercise tolerance in this domain are not dependent on power. It has been suggested that CP is susceptible to changes in aerobic energy pathways (10, 31) while W' is affected by anaerobic energy pathways (23, 24). Vanhatalo *et al.* (36) showed that breathing hyperoxic gas increased CP, but decreased W' , while Simpson *et al.* (31) showed breathing hypoxic gas decreased CP while having no effect on W' . Further, glycogen depletion decreased W' while CP remained unchanged (24). Jones *et al.* (19) found that at task failure at varying intensities in the severe intensity domain, PCr, Pi, and H^+ were not different among tests. These data suggest that W' is susceptible to anaerobic energy stores and/or metabolite accumulation balance. However, our data suggest these mechanisms are not responsible for task failure at all intensities above CP. Above 60% 1RM, we found a breakpoint in the $1/Time$ model (Figure 1). At intensities 70 – 90% 1RM, a separate linear model was necessary to describe the $1/Time$ relationship. Similar to

the intensity-duration relationship of the severe intensity domain, this suggests that exercise tolerance in the extreme domain is limited or determined by mechanisms consistent within this domain, and would therefore be a unique W' of the extreme domain (W'_{ext}). Hill (18) hypothesized that this difference would be due to T_{lim} being too short to reach $\dot{V}O_{2max}$ in the extreme domain, compared to the severe domain where $\dot{V}O_{2max}$ should be reached by task failure. Hill (18) further hypothesized that the upper threshold of the severe domain would occur at intensities that limit T_{lim} to less than 2 min. This hypothesis is consistent with our data in that although T_{lim} for 60% 1RM was approximately 2 min, this T_{lim} was not different than the expected T_{lim} predicted by the slope of S1 – S3. However, at greater intensities, T_{lim} became much shorter than expected, showing a different linear relationship above 60% 1RM.

Peripheral Fatigue: To investigate potential changes in the characteristics of fatigue development throughout the severe and extreme domains, we measured changes in potentiated twitch force (Q_{tw}), maximal voluntary contraction (MVC), and voluntary activation (VA) pre- to post-exercise to distinguish the source of fatigue being either proximal or distal to the neuromuscular junction. Decreases in Q_{tw} have been used as evidence of peripheral fatigue. Previously, post-severe intensity exercise bouts, Q_{tw} has been shown to reach similar values (10, 11). Our data are consistent with these previous data; we show that decreases in Q_{tw} following exercise at S1-S3 and 60% 1RM reached significantly lower values compared to 70%, 80%, and 90% 1RM. This is further evidence that there are likely metabolic differences dictating exercise (in)tolerance between severe intensity exercise up to 60% compared to 70% to 90% 1RM.

It has been shown that within the severe domain, by-products of muscle contractions, such as blood lactate, Pi, and H^+ continue to rise before task failure is met and exercise is

stopped. Although blood lactate and H^+ do not reach steady-state during severe intensity exercise, their specific roles in fatiguing processes that directly cause exercise intolerance are questionable. Pi accumulation, mainly arising from the breakdown of PCr, has been shown to decrease force production by directly affecting the cross-bridge function while also reducing myofibrillar Ca^{2+} sensitivity (27). These intramuscular processes are consistent with our findings that Q_{tw} significantly decreased following severe intensity exercise.

However, the decrease in Q_{tw} was not different among post-exercise measurements following intensities at 70%, 80%, and 90% 1RM. During these short-duration exercises ($T_{lim} < 60$ s), it is likely that PCr and anaerobic glycolysis are the primary energetic pathways. Although PCr resynthesis has been shown to be rapid at exercise cessation (half depletion restored after ~30 s (16)), lactate removal is relatively slower (half accumulated lactate removed after ~ 10 min (30)). Further, no significant decline in Q_{tw} , VA, or MVC following exercise at 80% and 90% 1RM. This begs the question as to why exercise could not be sustained if force output was not significantly decreased. It may be that the fatigue which developed was sufficient to prevent additional contractions but, due to short exercise durations, the muscle was able to recover to elicit the same force/power as before exercise. However, it has been shown that peripheral fatigue takes minutes to recover from following exercise cessation (14), suggesting that mechanisms allowing for quick recovery would be located proximal to the neuromuscular junction.

Central Fatigue: Consistent with our third hypothesis, the MVC and %VA data suggest central fatigue is not a limiting factor in exercise tolerance above critical power. The present study showed no change in VA following severe or extreme exercise intensities. This is consistent with

previous studies. Bigland-Ritchie (4) reported little to no central fatigue development in half of the subjects (4/9) following a 60 s sustained MVC. In the remaining subjects, central fatigue accounted for at most 30% of the decrease in force production. They further noted that, with familiarization, most of these subjects could increase VA, minimizing the degree of central fatigue. This is consistent with the work of Burnley *et al.* (9) who showed that of the ~71% reduction in torque, ~26% was associated with reduction in VA following a 5 minute all-out knee extension test. Indeed, CP has been suggested to be the boundary below which fatigue is primarily determined by central factors, whereas above CP fatigue factors are primarily peripheral (32, 33).

It has been suggested that the mechano- and metabo-sensitive Group III/IV afferents play a protective role for exercising muscle. Amann *et al.* (1) proposed that Group III and IV afferents provide feedback that may reduce central motor drive with increases in muscle metabolite accumulation, leading to a de-recruitment of the exercising muscle and an inability to sustain exercise. Using fentanyl to block Group III and IV muscle afferents, Amann *et al.* (1) showed increased iEMG and greater power output during the first half of a 5 km self-paced cycling race compared to control and placebo groups. This suggested diminished afferent feedback led to an increase in power output as metabolite accumulation went 'unseen' by the CNS.

Martin *et al.* (21) showed that central fatigue can recover quickly. These investigators found that force of elbow extensors continuously decreased following sustained MVCs induced via corticospinal stimulation (CMEP) and brachial plexus stimulation (M_{\max}). However, any change in force via M_{\max} would also be reflected in the change in CMEP. Thus, when normalizing CMEP to M_{\max} , the change in CMEP recovered within 15 s following exercise cessation. This quick recovery of central mechanisms may shed light as to why exercise could

not continue despite no recorded change in MVC or VA following exercise at 80 and 90% 1RM. Post-exercise measurements in the current study were made as quickly as possible, and began 30 s following exercise cessation. However, this may have been enough time for those central factors that limited exercise to recover and therefore remain undetected.

Contraction Characteristics: Hill (17) showed that as force increases, the muscle velocity of contraction decreases. Further, as muscle fatigues at a constant work rate, time for tension development also increases. Together, this would suggest that time of contraction would increase with heavier workloads and throughout exercise bouts. In order to enhance ecological validity, we did not control for duty cycle in the present study. However, we did control for relaxation time in order to ensure the muscle would have constant time between contractions for blood flow (6). Interestingly, we found that within each subject, time of contraction (determined by length of EMG bursts) did not statistically change within individual exercise bouts or across bouts. This suggests that individuals have an intrinsic contraction time that does not vary with heavier loads. Although time of contraction was not different, towards end exercise, the rate of metabolite accumulation may increase with longer duration contractions. Therefore, if the subject increased their contraction time, the time for metabolite clearance would become a smaller proportion of the duty cycle, and an earlier task failure would be expected (6). Another explanation may be an altered concentric-eccentric cycle. As duty cycle was not controlled for, it could be that concentric contraction was lengthened followed by a much shorter or non-existent eccentric cycle which was undetected by EMG.

Experimental Considerations: EMG burst time reflects work done by the muscle. Though relaxation time was kept constant, contraction time was not. This was purposefully done to quantify any volitional changes in duty cycle from beginning to end exercise as well as differences from lighter to heavier loads. However, the average burst time for the first five contractions was not different from the average burst time of the last five contractions for any intensity ($p=0.46$) or among intensities ($p=0.38$) suggesting overall duty cycle did not change. We were unable to distinguish the concentric and eccentric portions of the contractions with EMG. Therefore, it may be possible that the concentric portion of the contraction gradually increased while the eccentric contraction was reduced. However, because total contraction times were constant and relatively short, we believe the any potential changes in concentric-eccentric balance would have minimal effect.

Conclusions: In conclusion, the current data show that the power-duration relationship of the severe intensity domain does not describe all intensities above CP. At $\sim 60\%$ 1RM, a different W' (W'_{ext}) became evident which described the power-duration relationship of the extreme intensity domain. Further, the mechanism(s) of fatigue that limit exercise tolerance in the extreme domain must be that which can be fully recovered in ~ 30 s. This likely means it originates centrally, and not within the muscle itself.

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