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Original Research

Performance Fatigability at Task Failure and Beyond: Distinct Patterns of Recovery Following Constant Load Versus Intermittent Cycling Exercise

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Abstract

International Journal of Exercise Science 18(7): 971-983, 2025. https://doi.org/10.70252/MJTH7186 The total work completed during an exercise session is often assumed to be the primary factor influencing the kinetics of post-exercise recovery. However, the duration of work and rest periods during high intensity interval training (HIIT) have been shown to impact the magnitude of physiological stress and could also impact post-exercise recovery. The aim of this study was to characterize the recovery time course of neuromuscular function following work-to-rest matched HIIT protocols with different work interval durations and conducted to task failure. Participants (n=12, n=6 females) completed a ramp incremental exercise test to determine peak power output (PPO). In a randomized order, participants completed 3 cycling protocols at 90% PPO: (i) 3 min work, 3 min passive rest HIIT ($HIIT_{3min}$), (ii) 1 min work, 1 min passive rest HIIT ($HIIT_{1min}$), and (iii) constant load (CL) cycling. Femoral nerve electrical stimuli during maximal voluntary contractions (MVC) of isometric knee extension were performed at baseline, task failure (TF), and TF+1min, TF+4min, and TF+8min to delineate the time course of neuromuscular function recovery. MVC force declined to the same level following the three conditions at TF and demonstrated a partial recovery within TF+8min (time effect: $P<0.001$). The evoked muscle twitch force declined more following the CL compared to $HIIT_{1min}$ at TF ($P = 0.013$) and showed a faster recovery within TF+8min ($P<0.024$). Voluntary activation decreased at TF in the $HIIT_{1min}$ but not in $HIIT_{3min}$ or CL (interaction effect: $P<0.023$) and fully recovered within TF+8min. Central and peripheral components of neuromuscular function demonstrate distinct time courses of recovery between CL exercise and work:rest matched HIIT protocols with different work interval durations.

Keywords: Neuromuscular recovery, high-intensity interval training, exercise performance, dynamic contractions, task failure

Introduction

The development and recovery of performance fatigability, which is defined as a decline in an objective measure of performance such as maximal voluntary force production capacity or time to task failure,[1] is modulated by the modality, density, volume, and intensity of the exercise

stimulus.[2–8] High-intensity interval training (HIIT) is a popular exercise paradigm[9] that allows the exerciser to complete a larger amount of work in the severe intensity domain in comparison to constant load exercise.[10] Therefore, HIIT exercise prescription has been utilized in the investigation of performance fatigability. For instance, Chidnok et al,[11] demonstrated that reducing the duration of passive recovery (48 s vs. 30 s vs. 18 s) in between 60 s high-intensity single-leg knee-extension bouts could aggravate intramuscular perturbations (e.g., reduced muscle [PCr] reconstitution between work intervals) and accelerate the attainment of the limit of tolerance. In another study, Davies et al,[12] used a constant work:rest ratio of HIIT intervals and demonstrated that, at the same power output, extending the duration of work intervals increased metabolic disturbances within the exercising muscles. These metabolic responses are likely responsible for the exacerbated performance fatigability observed during HIIT with longer intervals, as demonstrated by greater declines in maximal voluntary contraction (MVC) force output and muscle contractile function [9]. Despite these observations, however, no study to date has reported the effects of different work interval durations on the recovery profile of performance fatigability measures. Understanding the effect of HIIT characteristics (e.g., work and rest interval durations) on the extent of performance fatigability and the subsequent rate of recovery following HIIT protocols may have applications in optimizing the exercise training and recovery prescription in a periodized program aimed at inducing a specific fitness adaptation.[3,13–15]

Previous literature has investigated the kinetics of performance fatigability development and recovery following different exercise tasks,[8,16–19] however, no study has explored the effects of different HIIT work interval durations on the recovery of neuromuscular responses. In this context, exercise tasks that have been investigated include single leg concentric extension/flexion time trial,[20] sustained unilateral vs. bilateral isometric maximal voluntary knee extensions,[6] different intensity-duration of cycling protocols including 30-s all out, 10-min at severe-intensity, and 90-min at moderate-intensity tasks,[7,8] different recovery interventions (passive, active and electromyostimulation) following a treadmill run to volitional exhaustion,[17] and 90-min continues or intermittent running tasks.[21] However, as mentioned earlier, despite robust methodologies and novel findings, none of these studies delineated the recovery kinetics of neuromuscular functions at the central (i.e., processes at or above the presynaptic terminal of the neuromuscular junction) and peripheral levels (i.e., processes distal to the presynaptic terminal of the neuromuscular junction) determining voluntary and evoked muscle force output following HIIT protocols using different duration of work intervals (despite using the same work-to-rest ratio).

Thus, the purpose of this study was to characterize the time course of central and peripheral neuromuscular responses during post-task failure recovery following work-to-rest ratio and power output matched HIIT protocols differing in work interval duration (3:3 min vs. 1:1 min work:recovery). Considering that prior investigations, albeit not in HIIT exercise, have shown that the recovery of muscle twitch force was faster following shorter vs. longer time trials (e.g., 1-min vs. 10-min time trial),[22] and following a 30 s all out sprint compared to 10 min of cycling in the severe intensity domain,[8] we hypothesized that increasing the HIIT interval duration (resulting in a shorter time to task failure) and associated decline in voluntary and evoked force

output would be accompanied by an accelerated recovery of neuromuscular function relative to task failure.

Methods

Participants

Twelve healthy and recreationally active participants (age: 26.1 ± 5.3 years, height: 172.0 ± 10.3 cm, weight: 71.2 ± 11.4 kg; n=6 male participants [peak rate of oxygen consumption, $\dot{V}O_{2peak}$ = 3.28 ± 0.41 L·min^{-1}]; n=6 female participants [$\dot{V}O_{2peak}$ = 3.44 ± 0.32 L·min^{-1}]) were recruited using convenience sampling. The sample size required was estimated using G*Power software (version 3.1.9.2), with data from a previous investigation that explored neuromuscular fatigability development during two work-matched high-intensity intermittent exercises.[23] Participants completed a Physical Activity Readiness Questionnaire[24] and provided written, informed consent prior to participation. Participants were excluded from the study if (1) they reported having any neuromuscular and/or cardiorespiratory conditions that would negatively impact their capacity to exercise at high intensities; or (2) were not recreationally active, which for the purposes of this study was defined as engaging in at least 3 structured exercise sessions per week. We did not control for the female menstrual cycle but did note that, coincidentally, all female participants in this study were regularly menstruating, monophasic oral contraceptive users (tested during the 3 week active pill phase) or had a hormonal intrauterine device. These protocols were approved by the University of Calgary Conjoint Health Research Ethics Board (REB21-0629) and testing was conducted based on the Declaration of Helsinki (without registration). This research was carried out fully in accordance to the ethical standards of the *International Journal of Exercise Science*.[25]

Protocol

All exercise testing was performed on a semi-recumbent cycle ergometer that allowed near instantaneous assessment of neuromuscular function.[26] Prior to the experimental sessions, participants attended the laboratory to be familiarized with the experimental set up, the cycle ergometer, and the neuromuscular assessment protocol and to complete a ramp incremental test (20 W·min^{-1}) to establish peak power output (PPO) and $\dot{V}O_{2peak}$ values. Next, in a randomized order, with each session separated by at least 48 hours, participants completed three experimental sessions: (i) HIIT with a work:passive recovery duty cycle of 1 min:1 min ($HIIT_{1min}$), (ii) HIIT with a work:passive recovery duty cycle of 3 min:1 min ($HIIT_{3min}$), and (iii) continuous cycling at a constant-load trial (CL). The CL was used as a control condition to investigate the effect of increased metabolic perturbations without the interruptions of rest intervals applied during HIIT protocols. Whereas, previous literature[12] and pilot testing supported the use of a 1 min: 1 min ratio and a 3 min: 3 min because shorter and longer work intervals, despite being matched for their work-to-rest ratio, elicit less and more metabolic perturbations, respectively. In each experimental condition, participants cycled at a power output of 90 % of PPO (work phases and entire CL condition), and at a self-selected cadence between 80 and 90 rpm until task failure was reached. Task failure was defined as the

participants being unable to maintain a cadence of 60 rpm for 10 s despite strong verbal encouragement from the researcher.

Neuromuscular assessment.

With a knee angle of 90 degrees, the participant's dominant leg, as determined by the leg they would use to kick a ball,[27] was used for all neuromuscular assessments. Neuromuscular assessments were conducted on a validated semi-recumbent cycle ergometer that has previously been established as valid and reliable for the quantification of neuromuscular function via interpolated twitch technique [26]. Briefly, the horizontal force output during knee extension was measured using a pedal mounted on the ergometer crankshaft (PowerForce analysis system, Model PF1.0.0; Radlabor GmbH, Freiburg, Germany) at a sampling frequency of 500 Hz, processed [28], and displayed on a monitor positioned in front of the participant.

Peripheral nerve stimulation was delivered using an electrical simulator (DS7A; Digitimer, Welwyn Garden City, United Kingdom), with the cathode electrode (Kendall MediTrace) placed within the femoral triangle on the femoral nerve, and the anode electrode (Durastick Plus; DJO, Global, Vista, CA) placed on the gluteal fold. For each session, the stimulation intensity was progressively increased until an intensity that was 130% of the intensity needed to elicit a maximal twitch force from a single stimulus was determined and used all subsequent stimulation. The supramaximal stimulation intensity (mean ± SD) was 129.2 ± 26.5 mA for CL, 132.6 ± 34.4 mA for $HIIT_{3min}$, and 129.4 ± 27.0 mA for $HIIT_{1min}$.

The neuromuscular assessment consisted of a high frequency doublet stimulation (100 Db) superimposed on a MVC of isometric knee extension (MVC), followed 3 s later by three stimuli that were evoked every 3 s in a rested state (i) 100 Db stimuli, (ii) a low-frequency stimulus (10 Db), and (iii) a single twitch. Using this protocol, neuromuscular function was assessed at baseline (as the mean of two trials), immediately (within 1-2 s) post-task failure, and at 1-, 4-, and 8- min post-task failure to quantify the time course of recovery.

Electromyography (EMG) signals for the vastus lateralis (VL) and rectus femoris (RF) muscles were measured continuously. To decrease signal impedance, the target area for electrode placement (on the muscle belly) was identified, shaved, and cleaned with an alcohol swab. A self-adhesive Ag/AgCl surface electrodes (Kenall MediTrace; Covidien LLF, Mansfield, MA) was then placed on the target area. EMG recording was conducted at 2000Hz using PowerLab (16/3-ML800/P; ADInstruments) and the data was processed and analyzed as previously described.[28]

Data analysis.

Low frequency fatigue (LFF) was defined as the ratio between resting Db10 and Db100 stimuli (Db10:100). The MVC force and the VL maximal root mean square (RMS) EMG were defined as their maximum values during the 500 ms prior to the superimposed 100 Db stimulus. The maximal RMS was then normalized to the maximal M-wave peak to peak amplitude during the

single twitch (RMS·M_{max}^{-1}). Voluntary activation (VA) was calculated using the modified interpolated twitch technique formula (Huang et al. 2010).

$$VA(\%) = 100 - \frac{D\ x \frac{F_B}{F_{max}}}{F_{Pt}}\ x\ 100, \qquad [1]$$

Where, D is defined as the difference between MVC force and 100 Db stimulus force, F_B is the MVC force immediately prior to superimposed stimuli, F_{max} represents the maximal MVC force, and F_{Pt} describes the force evoked from the resting 100 Db stimulus.

For the purposes of data analysis and presentation, all data at task failure and during recovery is calculated on an individual participant basis as the percent change from the baseline value for that metric. Although not used for analysis, raw data is also provided in the supplementary material (Supplementary Table 1).

Statistical Analysis

GraphPad Prism (GraphPad Software V9.4, San Diego, CA) was utilized to conduct all statistical analyses. Dependent variable normality and sphericity was assessed by way of Shapiro-Wilk and Mauchly tests, respectively; Greenhouse-Geisser correction was implemented in cases where the assumption of sphericity was violated. Two-way repeated measures ANOVA (alpha level = 0.05) with Bonferroni post hoc analysis was used to assess the recovery of neuromuscular responses for 3 conditions (i.e., $HIIT_{1min}$, $HIIT_{3min}$, CL) for 4 time points (i.e., Task Failure [TF], TF+1min, TF+4min, TF+8min). Effect size was measured as partial eta squared η_p^2 for the ANOVA comparison (i.e., small < 0.02; medium 0.02-0.26, large > 0.26)[29] and Hedges' g for post hoc comparisons (i.e., small < 0.02, 0.02 ≤ medium < 0.8, large ≥ 0.8).[30] Descriptive analyses are presented as the mean ± standard deviation.

Results

With respect to task duration, task failure was shorter in the $HIIT_{3min}$ (7.9 ± 2.1 min of work) and CL (4.4 ± 1.0 min of work) conditions compared to $HIIT_{1min}$ (50.8 ± 3.4 min of work), however, no difference was observed between $HIIT_{3min}$ and CL.

Neuromuscular recovery responses for $HIIT_{1min}$, $HIIT_{3min}$, and CL conditions are presented as a percent of the baseline value for TF, TF+1min, TF+4min, and TF+8min timepoints (Figure 1). For MVC force, a main effect of time was found ($F_{3,33}$ = 9.13, P < 0.001, η_p^2 = 0.453) without condition or interaction effects indicated that this measure was greater at TF+8min than at TF (P < 0.001, g = 8.46) and TF+1min (P = 0.002, g = 2.99) (Figure 1A).

With respect to twitch force, an interaction effect was found ($F_{6,66}$ = 9.73, P < 0.001, η_p^2 = 0.469). This showed that CL twitch force was lower than $HIIT_{1min}$ at TF (P = 0.013); however, initial twitch force recovery (in relation to task failure values) of CL occurred at TF+4min (P < 0.001, g = 1.02), whereas initial recovery of $HIIT_{3min}$ did not occur until TF+8min (P < 0.024, g = 0.454)

and no recovery was observed for $HIIT_{1min}$. At TF+8min, CL twitch force was greater than both $HIIT_{1min}$ and $HIIT_{3min}$ ($P = 0.024$, $g = 0.402$; $P < 0.001$, $g = 0.611$) (Figure 1B).

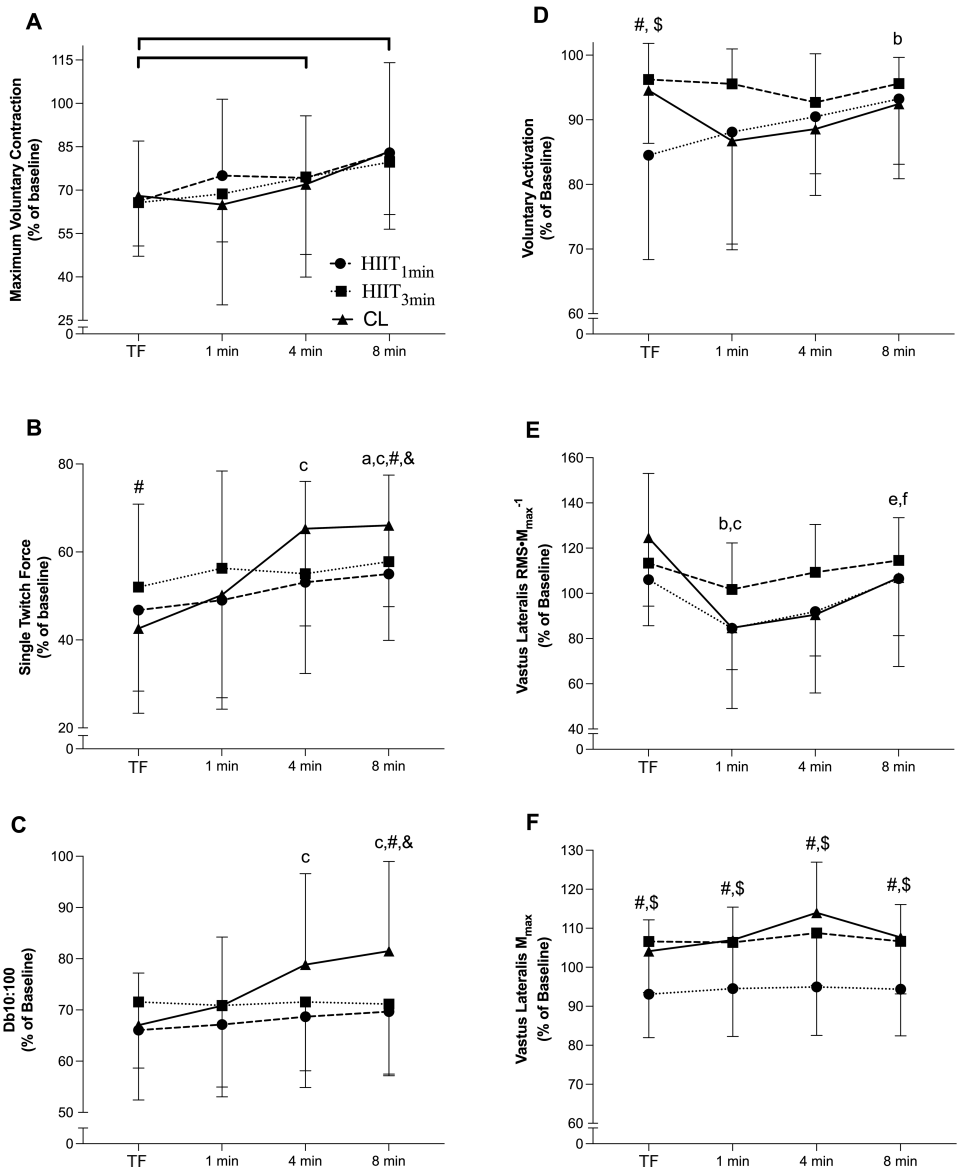

Figure 1. Recovery of Neuromuscular Function, as a Percent of Baseline, measured at 1, 4, and 8-minutes Post Task Failure. Maximal voluntary isometric force (A); single twitch force (B); Db10:100 (the ratio representing low frequency fatigue, LFF) (C); voluntary activation (D); vastus lateralis root mean square normalized to muscle compound action potential (RMS·Mmax-1) (E); and vastus lateralis muscle compound action potential (M_{max}). All above variables are presented as a percentage of their pre-exercise, baseline value. Experimental conditions included high intensity interval training (HIIT) protocol consisting of a 1-min on-phase and a 1-min rest phase ($HIIT_{1min}$), HIIT protocol consisting of a 3-min on-phase and a 3-min rest phase ($HIIT_{3min}$), and constant-load cycling trial (CL). a, $HIIT_{3min}$ is different than $HIIT_{3min}$ at task failure (TF); b, $HIIT_{1min}$ is different than $HIIT_{1min}$ at task failure; c, CL is different than CL at task failure; e, $HIIT_{1min}$ is different than $HIIT_{1min}$ at task failure plus 1 min; f, CL is different than CL at task failure plus 1 min; #, CL is different than $HIIT_{1min}$; &, CL is different than $HIIT_{3min}$; $, $HIIT_{1min}$ is different than $HIIT_{3min}$.

Regarding LFF, an interaction effect was found ($F_{6,66}$ = 3.840, P = 0.002, η_p^2 = 0.259) and showed that the initial recovery of CL occurred at TF+4min (P < 0.001, g = 0.761), and no recovery of $HIIT_{1min}$ or $HIIT_{3min}$ was present within 8 min (P > 0.391) (Figure 4E). At TF+8min, the Db10:100 ratio for CL was larger than for $HIIT_{1min}$ (P = 0.003, g = 0.452) or $HIIT_{3min}$ (P < 0.001, g = 0.557) (Figure 1C).

There was an interaction effect for VA ($F_{6,66}$= 2.650, P = 0.023, η_p^2 = 0.194), where $HIIT_{1min}$ demonstrated a lower VA than $HIIT_{3min}$ and CL at TF (P < 0.001, g = 0.904; P = 0.016, g = 0.731). Also, the initial recovery of $HIIT_{1min}$ occurred at TF+8min (P < 0.050, g = 0.602) whereas no significant recovery for $HIIT_{3min}$ or CL was present by TF+8min (P > 0.999). At TF+8min, no differences were presented between conditions (P > 0.999) (Figure 1D).

An interaction effect for RMS·M_{max}^{-1} of the VL muscle ($F_{6,66}$ = 2.316, P = 0.043, η_p^2 = 0.174) demonstrated that $HIIT_{1min}$ and CL decreased to a lower level at TF+1min compared to TF (P = 0.047, g = 1.03; P < 0.001, g = 1.12). From TF+1min, the initial recovery of $HIIT_{1min}$ (P = 0.0357, g = 0.926) and CL (P = 0.031, g = 0.550) occurred at TF+8min and no recovery of $HIIT_{3min}$ was present by TF+8min (P>0.999). Additionally, at TF+8min, no differences were observed from TF (P > 0.999) and no differences were present between conditions (P > 0.999) (Figure 1E).

With respect to VL muscle M_{max}, an interaction effect was observed ($F_{6,66}$ = 2.840, P = 0.0162, η_p^2 = 0.205) where at TF, $HIIT_{1min}$ was lower than $HIIT_{3min}$ and CL (P < 0.001, g = 1.03; P < 0.001, g = 1.05). However, for all conditions, no difference between TF and TF+8min was noted and at TF+8min, HIIT1min was lower than HIIT3min (P < 0.001, g = 0.895) and CL (P < 0.001, g = 1.19) (Figure 1F).

Discussion

This study explored the acute recovery of the central and peripheral neuromuscular responses to power output and work-to-rest ratio matched HIIT protocols (i.e., $HIIT_{1min}$ vs. $HIIT_{3min}$) with different work interval durations. A power out-matched constant load cycling exercise to task failure (CL condition) was also tested to study the effect of power output specific metabolic perturbations without the influence of rest intervals applied during the HIIT protocols. The major findings of this study were that, although MVC showed a similar recovery profile following the three experimental conditions, the peripheral and central neuromuscular subcomponents of performance fatigability demonstrated condition-dependent recovery kinetics. Specifically, CL resulted in more rapid recovery of muscle contractile function (i.e., peripheral component) compared to both $HIIT_{1min}$ and $HIIT_{3min}$. This was contrary to our hypothesis which stated that $HIIT_{3min}$ would demonstrate more rapid recovery of voluntary and evoked muscle contraction force in comparison to shorter work duration HIIT ($HIIT_{1min}$). Additionally, we noted that $HIIT_{1min}$ demonstrated greater depression and faster recovery of VA compared to the other conditions. Overall, these results suggest that both the development and recovery of central and peripheral neuromuscular functions during HIIT and constant load exercise needs to be taken into consideration. These findings may have implications in how practitioners optimize the prescription of exercise sessions within a periodized program.

This work expands our knowledge of the impact of the training session design on the recovery of neuromuscular functions at post-exercise. Previous work has addressed the impact of power output and duration of exercise on recovery kinetics,[6,8,11,17,20,21] but the impact of different HIIT work interval durations per se is less studied. Nevertheless, our results suggest that sustaining the same power output without (i.e., CL) or with less frequent rest intervals (i.e., $HIIT_{3min}$) was accompanied by more rapid recovery of twitch force in comparison to exercise conducted for a longer duration (i.e., $HIIT_{1min}$). On the other hand, only longer duration exercise (i.e., $HIIT_{1min}$) resulted in a marked reduction in the central neuromuscular indices of performance fatigability (i.e., voluntary activation) at task failure, which recovered within 8-min. These are indeed novel findings because none of the prior studies had intensity matched conditions where the effect of different work intervals was explored on recovery kinetics of neuromuscular responses.

While a decrease in muscle contractile function has been shown to be closely associated with metabolic perturbations in the muscle during severe intensity exercise,[4,31–33] the measurement techniques used in the current study can not elaborate on discrete underpinning mechanisms facilitating faster recovery of muscle contractile function following CL and $HIIT_{3min}$ compared to the $HIIT_{1min}$ protocols. Considering that longer work interval HIIT (i.e., $HIIT_{3min}$) and CL exercise tasks have a physiological profile reflective of the severe domain,[28] it is plausible that task failure in these trials is accompanied by a progressive accumulation of metabolic by-products that impair muscle contractile function and the depletion of anaerobic capacity (also referred to as W`). However, an augmented accumulation of metabolites during $HIIT_{3min}$ and CL does not seem to corroborate with the faster recovery of muscle contractile function in these two conditions. Therefore, a more rapid recovery in $HIIT_{3min}$ and CL conditions could be associated with faster replenishment of anaerobic sources. On the other hand, shorter work interval HIIT (i.e., $HIIT_{1min}$), despite involving a severe intensity of exercise, has a physiological profile more similar to that generally observed in the heavy domain.[12,28] Accordingly, plausible explanations for the delay recovery of resting twitch force in $HIIT_{1min}$ condition could be glycogen depletion[34] and/or the inhibition of excitation-contraction coupling by reactive oxygen species.[35,36] Indeed, there are multiple lines of evidence indicating that reactive oxygen species play a greater inhibitory role during longer exercise tasks.[37] Of note, the influence of factors such as precipitation of Ca^{2+} caused by P_i reducing Ca^{2+} release from the sarcoplasmic reticulum[38] should not be overlooked, as this may influence recovery of muscle contractility. However, our data demonstrates no differences in LFF (i.e., taken as proxy for reduced Ca^{2+} release) at task failure. Therefore, further mechanistic studies are required to elucidate the contribution of factors such as glycogen depletion, release of reactive oxygen species, and accumulation of metabolites such as P_i modulating Ca^{2+} release and Ca^{2+} sensitivity on the recovery kinetics of muscle contraction function after different HIIT protocols.

When considering the central neuromuscular component of performance fatigability, the results indicate that although $HIIT_{1min}$ VA was depressed to a larger extent than the other conditions at TF, VA recovered to the same extent in all conditions within 8 minutes post TF. Previous literature has stated that reductions in VA following exercise is often a function of exercise duration, where in longer tasks VA is mitigated to a greater extent,[8] and this argument is supported in the present data. In general, considering that the recovery profiles of

neuromuscular function underlying performance fatigability distinctly reflect HIIT work interval duration, these findings may impact current HIIT exercise prescription methodologies and should be investigated further with different work interval durations and through the modulation of other HIIT characteristics.

Understanding the patterns of neuromuscular function recovery following HIIT is interesting to gain insight into the mechanisms of fatigability. For instance, based on our findings, the presence of distinct recovery patterns in muscle contractile function between HIIT and CL in the presence of similar performance fatigability at task failure and a similar work accomplished between $HIIT_{3min}$ and CL suggests that the repeated metabolic fluctuations during HIIT have a longer lasting effect on muscle contractile function than continuous protocols. This has important implications because it is often assumed that, upon task failure following severe-intensity exercise, the rate of neuromuscular performance recovery primarily depends on the characteristics of the recovery phase (e.g., intensity, duration).[39] Adding onto this evidence, here we show that despite similar work accomplishment and performance fatigability at task failure between $HIIT_{3min}$ and CL, recovery of the neuromuscular system depends on the characteristics (i.e., density) of the fatiguing exercise. These findings have implications for the planning and prescription of various interval style training sessions across a periodized plan. Specially, the findings of this study suggest that practitioners cannot rely exclusively on the exercise duration and intensity as indicators of the recovery needed between training sessions. By considering exercise duration, intensity, and density together, and by quantifying fatigue and recovery responses in the minutes/hours following exercise, practitioners are likely to gain important contextual information that can inform the prescription of subsequent training sessions. To this end, future research should adopt a similar methodology in different exercise modalities where various densities of training are prescribed in resistance training (e.g., cluster sets) or sport-specific training (e.g., sports relying on repeated bouts of high intensity, such as ice hockey) with little consideration for the associated magnitude and rate of performance fatigability recovery.

The recovery was quantified from task failure to 8 minutes post task failure. This provides detailed insights into the acute recovery kinetics of longer and shorter HIIT work intervals relative to CL cycling. Understanding the acute recovery kinetics may be important in informing recovery in protocols that require multiple exercise bouts completed in relatively rapid succession. However, we did not quantify recovery using a longer timeframe that included evaluating neuromuscular function hours after exercise. This is a promising area for future research as this data could provide important additional information that is applicable to most structured training programs. In addition, we evaluated recovery of neuromuscular function following two HIIT protocols with distinct work interval durations. Quantification of recovery following other popular structures of HIIT (i.e., 0.5:1min work-to-rest intervals) would add to the body of knowledge in this area.

This study demonstrates that despite no condition-dependent changes occurring during the MVC recovery, the central and peripheral components of neuromuscular function demonstrated distinct time courses of recovery between CL exercise and work-to-rest ratio matched HIIT protocols with different work interval durations. Collectively, elucidating the influence of

different HIIT characteristics on the amount of work that can be accumulated and the subsequent recovery time course of neuromuscular function following exercise may have implications to understanding the mechanisms and magnitude of physiological adaptation following exercise and using this information to prescribe exercise within a periodized program. Future research should build off the findings of the present study and investigate neuromuscular recovery kinetics within a longer time frame following task failure and consider the impact of multiple consecutive exercise bouts on recovery kinetics.

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Supplementary Table 1.

Condition	Time	MVC (N)	Single Twitch (N)	Db10:100	VA (%)	VL RMS·M_{max}^{-1} (mV)	RF RMS·M_{max}^{-1} (mV)	VL M_{max} (mV)	VL M_{max} (mV)
$HIIT_{1min}$	BL	449.6 ± 173.5	151.9 ± 51.0	1.00 ± 0.05	94.0 ± 3.9	0.04 ± 0.01	0.05 ± 0.02	15.7 ± 5.7	8.4 ± 2.7
	TF	282.0 ± 125.0	74.2 ± 30.0	0.72 ± 0.14	79.3 ± 15.1	0.04 ± 0.01	0.04 ± 0.01	14.4 ± 4.7	7.5 ± 2.6
	TF+1min	313.4 ± 130.0	82.0 ± 17.5	0.71 ± 0.17	82.7 ± 17.6	0.03 ± 0.01	0.04 ± 0.01	14.5 ± 4.7	7.5 ± 2.7
	TF+4min	323.0 ± 157.0	80.4 ± 40.7	0.72 ± 0.14	85.0 ± 8.8	0.04 ± 0.01	0.04 ± 0.01	14.6 ± 4.9	7.4 ± 2.8
	TF+8min	345.2 ± 140.9	85.0 ± 40.0	0.72 ± 0.14	87.4 ± 8.5	0.04 ± 0.01	0.05 ± 0.01	14.6 ± 5.0	7.8 ± 2.6
$HIIT_{3min}$	BL	459.3 ± 156.5	154.9 ± 58.2	1.04 ± 0.09	95.1 ± 3.9	0.04 ± 0.01	0.05 ± 0.03	16.5 ± 5.8	7.9 ± 3.2
	TF	295.9 ± 130.5	68.7 ± 27.6	0.69 ± 0.16	91.5 ± 6.1	0.04 ± 0.02	0.06 ± 0.03	17.4 ± 5.7	8.1 ± 3.0
	TF+1min	317.0 ± 132.6	71.1 ± 32.6	0.70 ± 0.18	90.8 ± 4.8	0.04 ± 0.01	0.04 ± 0.01	17.3 ± 5.4	8.2 ± 3.0
	TF+4min	345.2 ± 162.63	76.80 ± 28.7	0.72 ± 0.16	88.1 ± 6.5	0.04 ± 0.01	0.05 ± 0.02	17.6 ± 5.3	8.4 ± 3.1
	TF+8min	368.8 ± 155.2	81.9 ± 31.3	0.73 ± 0.15	90.9 ± 4.6	0.04 ± 0.01	0.04 ± 0.01	17.3 ± 5.4	8.4 ± 3.0
CL	BL	455.0 ± 186.8	146.1 ± 52.8	1.02 ± 0.05	95.6 ± 4.0	0.04 ± 0.01	0.06 ± 0.02	18.0 ± 6.1	8.7 ± 2.7
	TF	303.71 ± 144.3	58.5 ± 27.9	0.68 ± 0.12	90.2 ± 6.4	0.056 ± 0.01	0.06 ± 0.03	18.5 ± 5.9	8.3 ± 2.9
	TF+1min	281.9 ± 152.2	68.1 ± 36.4	0.72 ± 0.15	82.9 ± 15.7	0.03 ± 0.01	0.04 ± 0.01	18.9 ± 5.6	8.8 ± 2.9
	TF+4min	328.5 ± 196.2	88.6 ± 31.2	0.80 ± 0.18	84.7 ± 10.0	0.04 ± 0.01	0.04 ± 0.02	20.2 ± 6.0	9.3 ± 3.0
	TF+8min	373.7 ± 188.4	91.2 ± 30.3	0.82 ± 0.17	88.2 ± 9.7	0.04 ± 0.01	0.05 ± 0.01	19.3 ± 6.5	9.2 ± 3.1