Intensity-dependent tolerance to exercise after attaining $\dot{V}O_2_{\text{max}}$ in humans

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Although this relationship is difficult to determine at the extremes of muscular exercise, such as short (e.g., <1–2 min) all-out efforts greater than ~135% of the work rate (WR) associated with maximal oxygen consumption ($\dot{V}O_2_{\text{max}}$) (10) or for durations of ≥60 min (5), for durations between these limits, the hyperbolic relationship appears to provide a good characterization of the physiological response. The curvature constant of the hyperbola ($\dot{W}$) is mathematically equivalent to a constant amount of work, i.e., the product of WR (above CP) and time (6, 19, 22), and has been suggested to be notionally equivalent to an energy store consisting of oxygen, high energy phosphates, and a source related to anaerobic glycolysis (17, 19). However, it may also relate to the buildup of fatigue-inducing metabolites or conditions to a critical level, such as low intramuscular pH and/or high intramuscular Pi concentration ([Pi]).

The CP threshold has also been shown to be a close correlate of the highest metabolic rate that is associated with pulmonary oxygen uptake ($\dot{V}O_2$), and acid-base status and blood lactate concentration ([La]) being maintained at a constant level (10, 22). As such, CP has been suggested to characterize the transition between, what Whipp (30) has termed, the heavy- and very heavy-intensity exercise domains. Above CP, in the very heavy-intensity domain, $\dot{V}O_2$ and [La] increase inexorably, terminating at fatigue with $\dot{V}O_2$ attaining its maximum (e.g., Refs. 21, 22) and depletion of (or attainment of the critical metabolite accumulation determining) $\dot{W}$'. Therefore, Fukuba and Whipp (6) have hypothesized that continued exercise after exhaustion (i.e., with $\dot{W}'$ depleted) would only be possible if the power output were reduced to a level below CP, where predominantly aerobic energy transfer can occur, i.e., permitting $\dot{W}'$ to be partially restored or replenished. Conversely, it is suggested that if at the point of fatigue (where fatigue is used in the sense of a level of fatigue relationship has an asymptote on the power axis termed the critical power (CP; or fatigue threshold).
that limits exercise tolerance) the power output were reduced to a level greater than CP, then continued exercise would not be possible because repulsion of W′ would not occur in a domain where obligatory increases in V̇O₂, [La], and [Pi] were evident. We were interested in testing this hypothesis by determining whether, after induction of limiting fatigue with an initial intense exercise bout, it was possible to continue to exercise at a reduced WR if the rate was either above or below CP.

METHODS

Exercise tests. Six healthy male subjects (age 22 ± 2.7 yr; height 183 ± 1.6 cm; body mass 85 ± 5.2 kg; Table 1) from the Medical School population volunteered to take part in the study after providing informed consent, as approved by the Local Research Ethics Committee (in accordance with the Declaration of Helsinki). After familiarization with the laboratory and procedures, each subject performed 7 exercise tests on an electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands); each test was performed on a different day. Subjects initially performed an incremental ramp test (25 W/min, incremented at a rate of 5 W every 12 s) to the limit of tolerance. This allowed the V̇O₂ max and the lactate threshold (θL) to be estimated independently by two experimenters using standard noninvasive pulmonary gas exchange criteria (3, 32).

Exercise tests were then performed to exhaustion at three different high-intensity constant-load WRs, chosen to span a range of fatigue times of ~4–10 min. The subjects were required to maintain a pedal cadence between 60 and 80 rpm for all constant-load tests. The time to exhaustion were measured to the nearest second with a stopwatch and used to estimate each subject’s CP by using a three-point linear regression and extrapolation of the time-1 vs. WR relationship. The slope of this relationship provides the W′, and its WR intercept corresponds to CP (e.g., Refs. 9, 22). All tests were preceded by (3–4 min) and followed by (6 min) a 20-W control period. All constant-load tests were assigned in a randomized sequence.

Finally, each subject completed three subsequent tests consisting of an initial exhaustive WR (WRexh; targeted from the power-duration relationship to exhaust the subject in ~6 min), followed by, at the point of fatigue (when subjects were no longer able to maintain a pedal cadence of at least 60 rpm), an abrupt reduction in the WR to each of three intensities for a 20-min target period; 1) 110% CP (very heavy), 2) 90% CP (heavy), and 3) 80% CP (moderate). Each subject performed the experiments requiring the recovery intensities of very heavy, heavy, and moderate in a randomized sequence and separated by at least 48 h. If the subject was unable to maintain the recovery exercise for the required target of 20 min, the fatigue time was recorded and the WR was reduced to 20 W for the remainder of the 20 min.

Equipment. The subjects breathed through a mouthpiece connected to a low-dead space (90 ml), low-resistance (<1.5 cmH₂O at 3 l/s) turbine volume transducer (Interface, Irvine, CA) for the measurement of inspiratory and expiratory volume. Respired gas was continuously sampled (at 1 ml/s) from the mouthpiece and analyzed by mass spectrometry (QPS9000, Morgan Medical, Gillingham, UK) for the concentrations of oxygen, carbon dioxide, and nitrogen. Before each experiment, calibration was made from two precision-analyzed gas mixtures chosen to span the range of respired gas concentrations; this calibration was verified at the end of each experiment. The time delay between the gas concentrations and the volume signals was measured by passing a bolus of known gas mixture through the system by using a low dead-space solenoid valve (2). The electrical signal outputs were digitally converted every 20 ms and sampled by computer for the calculation of V̇O₂, carbon dioxide output (V̇CO₂), and minute ventilation (V̇E) by using the algorithms of Beaver et al. (1, 2).

Analyses. Editing of data was performed from V̇O₂ in the time domain to exclude occasional aberrant breaths caused by swallowing, coughing, sighing, etc., which were considered not to reflect the underlying response, i.e., values greater than three standard deviations from the local mean were omitted (15). All breath-by-breath V̇O₂ responses were interpolated on a second-by-second basis and time averaged by using discrete 10-s time bins to produce a standard weighted response.

Times to fatigue were compared by using ANOVA and post hoc Neuman-Keuls tests. Differences between the six square-wave exhaustive exercise bouts were considered significant if P < 0.05. The dispersion about the mean is expressed as ± standard deviation.

RESULTS

Incremental ramp exercise. V̇O₂ increased linearly throughout the incremental ramp test (after a delay due to the V̇O₂ time constant), up to V̇O₂ max (averaging 4.5 ± 0.3 l/min). The slope of the V̇O₂ response as a function of WR (or gain) averaged 12.3 ± 0.6 ml·min⁻¹·W⁻¹, falling within the expected normal limits for cycle ergometer exercise (28). θL averaged 2.7 ± 0.3 l/min, which corresponded to 60.0 ± 4.6% of V̇O₂ max (Table 1).

Constant-load tests to estimate CP. In the subsequent exhaustive constant-load tests, V̇O₂ max was not significantly different from that obtained during the incremental ramp test (4.5 ± 0.4 l/min). As expected, the time to fatigue was inversely correlated (P < 0.05)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>V̇O₂ max, l/min</th>
<th>θL, l/min</th>
<th>θL, % V̇O₂ max</th>
<th>CP, % Δ*</th>
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<tr>
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<tr>
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<td>22</td>
<td>185</td>
<td>81</td>
<td>4.6</td>
<td>3.1</td>
<td>67.4</td>
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<tr>
<td>5</td>
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<tr>
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<td>0.3</td>
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<td>4.6</td>
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</table>

V̇O₂ max, maximal oxygen uptake; θL, lactate threshold; CP, critical power. Δ* is defined at the difference between the work rate at θL and V̇O₂ max (see Ref. 21 for further discussion).

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to power output in all subjects. An example from subject 1 is shown in Fig. 1A. With the use of the time to fatigue at each of these WRs, a linear regression and extrapolation of the power vs. time−1 relationship was used to calculate CP (the abscissa intercept) and W′ (the slope of the linear fit) for each subject (an example from subject 1 is shown in Fig. 1B). The 95% confidence limits of the fit (dotted lines in Fig. 1B) were also calculated, and the fit was deemed acceptable if the confidence of CP estimation was within ±5%, which was the case in all but one instance. In this case, a fourth constant-load test was performed at another WR to improve the accuracy of CP estimation. The CP threshold occurred at 246 ± 24 W, which was equivalent to 41.8 ± 9.0% of the change in WR range (defined as the difference between the WRs at θL and VO2,max taken from the ramp incremental test, c.f., Özyener et al. (21)) (Table 1). The W′ value averaged 20.9 ± 2.35 kJ or 246 ± 30 J/kg for the six subjects. 

Intensity-dependent recovery tests. WRexh, predicted from the power-duration relationship, to fatigue the subject in ~6 min averaged 305 ± 22 W (Table 2). Subjects completed an average of 348 ± 40 s at WRexh before fatigue (there being no significant difference between each of the 3 bouts). The subsequent WRs for the three experimental conditions (110 and 90% CP, and 80% θL) are given in Table 2. When the WR was reduced to 110% CP (very heavy-intensity domain; a reduction in WR averaging 34 ± 7 W), exercise was sustained, on average, for only 30 ± 12 s beyond the point of initial fatigue (i.e., after exhaustion at WRexh; Table 2). Even though WR was reduced by an average of 11 ± 2%, subjects were still unable to sustain a significant duration of exercise. This was calculated to be equivalent to only 3.5 ± 1.7% of W′ (on the basis of the WR above CP multiplied by the recovery duration expressed as a percentage of W′). Examples of this response are shown in Fig. 2, A and B.

In contrast, when WR was dropped to 80% θL (the moderate-intensity domain; a reduction in WR averaging 170 ± 22 W), all subjects completed the 20-min target period, as expected (Table 2; Fig. 2A). This corresponded to a 56 ± 4% reduction in WR.

Interestingly, however, only two of six subjects completed the target 20 min at the 90% CP WR (the heavy-intensity exercise domain; a reduction in WR averaging 83 ± 5 W) with the remaining four subjects fatiguing before the 20-min target. The exercise duration at 90% CP was highly variable and averaged 785 ± 400 s (range: 188–1,200 s; Table 2). The drop in WR from WRexh averaged 27 ± 2%. Two examples of the VO2 response are shown in Fig. 2, A and B. Figure 2A provides an example of a subject who could not sustain the 20-min target exercise period, and Fig. 2B provides an example of a subject who was able to sustain the 20 min of exercise after initial fatigue at WRexh. Figure 3 illustrates the times to fatigue for each subject (after the initial fatiguing WRexh bout) at the three different exercise intensities.

The four subjects who were unable to sustain exercise for the 20-min target period all fatigued at a value of VO2 less than the maximum (e.g., subject 1 in Fig. 2A). In all cases, standard respiratory measurements revealed that metabolic demands were falling at the time of fatigue. The responses of VO2, VCO2, and VE were falling in all cases. In these four subjects, VO2 at fatigue averaged 4.1 ± 0.4 l/min, or 89 ± 8% of their VO2,max, VCO2 averaged 3.9 ± 0.3 l/min, or 73 ± 5% of VCO2,max (of the WRexh constant load), and VE averaged 113 ± 9 l/min, or 77 ± 10% of VO2,max (of the WRexh constant load). Figure 4, A and B, shows examples of VCO2 and VE in subjects 1 and 2, respectively (subject 1 fatigued before the 20-min target; subject 2 achieved the 20-min target). Heart rate, however, was observed to have a secondary rise during the 90% CP recovery exercise bout, causing the four subjects who fatigued in recovery to attain a heart rate of 100 ± 3% of their maximum heart rate (HRmax), compared with the two subjects who achieved the 20-min target who attained

Fig. 1. A: superimposed oxygen consumption (VO2) responses from 3 constant-load exhaustive exercise tests (subject 1); B: estimate of critical power (CP) from the power-to-time−1 relationship obtained from the exercise tests depicted in A. Extrapolation of best-fit line (with 95% confidence bands; dotted lines) to abscissa allows CP estimation. VO2,max, maximal VO2.
96 ± 2% of HRmax (2 individual examples are shown in Fig. 4, A and B).

ANOVA and post hoc (Neuman-Keuls) tests revealed that, although there were no differences between exercise durations at WR_{exh} on the three tests, there was a significant difference between the exercise duration during the recovery from a fatiguing bout (WR_{exh}) within each of the moderate, heavy, and very-heavy exercise intensity domains (P < 0.05). Figure 5 shows a summary of the group mean responses. The mean power duration curve and the CP and \( \hat{\theta}_L \) thresholds are shown, and the arrows depict the mean durations in recovery after depletion of W during WR_{exh}.

**DISCUSSION**

Despite an abrupt reduction in the WR at the point of fatigue, a significant amount of exercise could not be

<table>
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<tr>
<th>Subject</th>
<th>WR_{exh}, W</th>
<th>110% CP, W</th>
<th>90% CP, W</th>
<th>80% ( \hat{\theta}_L ), W</th>
<th>110% CP, s</th>
<th>90% CP, s</th>
<th>80% ( \hat{\theta}_L ), s</th>
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<td>266</td>
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<td>5</td>
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<td>209</td>
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<td>26</td>
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<td>254</td>
<td>208</td>
<td>135</td>
<td>43</td>
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<tr>
<td>Mean</td>
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<td>271*</td>
<td>222*</td>
<td>135*</td>
<td>30†</td>
<td>785†</td>
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<td>9</td>
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</table>

WR_{exh}, exhaustive work rate. *Significant difference (P < 0.05) between each of the other work rates. †Significant difference (P < 0.05) between each of the other recovery durations.
maintained if this WR was above a subject's CP. The results are, therefore, in accordance with the suggestions of Fukuba and Whipp (6) and have broad implications for the demarcation of the domains of exercise intensity in humans. First, they suggest that the physiological underpinnings of the concept of CP are valid and remain so even during and after a fatiguing bout of exercise, i.e., once WR is depleted, exercise cannot continue unless the WR is reduced into a domain where “wholly aerobic” energy transfer may allow exercise to be sustained. The very heavy-intensity domain has been demonstrated to result in a continuously increasing metabolic rate when WR is abruptly increased (e.g., Refs. 21, 22). Second, therefore, the results of the present study demonstrate that this intensity domain is associated with a progressively increasing drive to metabolism that is independent of the prior energy state of the muscle, i.e., despite a reduction in WR, subjects were unable to meet the energy demands of the task. As such, it appears that functional recovery within the very heavy-intensity domain (with the use of the definition of Whipp (30)) is not possible; that is, V̇O₂ was not able to fall, and it is inferred, therefore, that [La] would also not reduce (c.f., Ref. 22), and hence pH would be likely to continue to fall, high-energy phosphate stores, e.g., phosphocreatine concentration ([PCr]), would continue to decrease.

Fig. 3. Times achieved for each subject during recovery exercise at each of 80% WR (moderate), 90% CP (heavy), and 110% CP (very heavy).

Fig. 4. Responses of carbon dioxide output (VCO₂; top), minute ventilation (Ve; middle), and heart rate (bottom) in 2 subjects during the 90% CP exercise bout. Both subjects exercised to the limit of tolerance at WRexh. bpm, Beats/min. A: subject who did not achieve the 20-min target recovery period at 90% CP. B: subject who did achieve the 20-min target.
oxygen deficit, however, W' (17). Because of its relationship with the oxygen, and stores of muscle glycogen related to anaerobic capacity or maximum oxygen deficit (9), to be related to the anaerobic work performed by Monod and Sherrer (18), and Moritani et al. (19), and Smith (9, 11) in humans. The amount of work, 246 kJ or 96% of HRmax, required for exercise to continue at a supra-CP WR a proportion of W' must remain. If W' were depleted during an exercise bout then a reduction in the WR to a level below CP is required for W' to be replenished. In a practical sense [similar to Fukuba and Whipp (6)], this suggests that an athlete who performs with such an ill-considered pace strategy that W' is depleted before the end of the race would have to not only reduce their WR to continue but reduce it to a sub-CP level.

The determinants of W' are presently poorly understood but have been suggested by Knuttgen (14), Monod and Sherrer (18), and Moritani et al. (19), and in review by Hill (9) to be related to the anaerobic work capacity or maximum oxygen deficit (9, 16). These reflect energy stores, such as PCr (and ATP), stored oxygen, and stores of muscle glycogen related to anaerobic glycolysis (17). Because of its relationship with the oxygen deficit, however, W' may also reflect an inverse function of this, such as the attainment of maximum rates of aerobic function (attainment of VO2 max), or even a buildup of fatigue-inducing metabolites such as H+ and P1. What is interesting about the CP concept is that it predicts that fatigue will ensue as soon as W' is depleted, an event that occurs only above CP; these data add that W' may also not be replenished in this intensity domain despite reducing the WR, i.e., the flux of W' is unidirectional above CP.

W' in the present study averaged 20.90 ± 2.35 kJ or 246 ± 30 J/kg, consistent with values reported by Hill and Smith (9, 11) in humans. The amount of work actually performed by the subjects in the very heavy-intensity domain after initial fatigue corresponds to only a small proportion of this value (3.5 ± 1.7%). However, the ~30-s tolerance duration at the supra-CP intensity may actually reflect a tolerance time of, or close to, zero were the subject (and W') actually exhausted by the fatiguing ride. That is, the duration of the recovery period was determined from the point at which the subject could no longer maintain a pedal cadence of >60 rpm, at which point the WR was dropped and the subject was instructed to again increase the pedal cadence; this recovery then continued until the subject could again no longer maintain 60 rpm. It was, however, necessary to allow the subjects some time at the new, reduced WR to determine whether they could actually sustain the required exercise (during which time they received verbal encouragement from the experimenters and the pedal cadence was in some cases continually falling throughout). This time amounted to ~10 s. Thus we had to allow the subjects sufficient W' reserve to be able to bring the cycling cadence back up to the target level.

It was not surprising that all the subjects were able to continue exercise in the moderate-intensity domain (below the \( \theta_f \)). This necessitates a greatly reduced rate of ATP provision and, therefore, VO2 requirement. This domain does not require a significant proportion of energy derived from anaerobic mechanisms and, therefore, allows for the clearance of fatigue-inducing substances.

What was more surprising, however, was that four of six subjects were unable to maintain exercise in the heavy-intensity domain for the required 20-min target; the overall average was ~13 min. The hypothesis, based on the models of Fukuba and Whipp (6), suggested that subjects would be able to sustain exercise in this intensity domain after initial fatigue. Although we cannot rule out the possibility that the wide variability observed between the recovery duration tolerated at this intensity may, at least in part, be due to day-to-day variability in performance, we believe this to be unlikely on the basis of both the magnitude of the differences and the different response profiles in those who attained the target recovery time and those who did not. Although each recovery bout was not repeated, the initial WRexh was undertaken on three occasions that yielded an average coefficient of variation for constant load performance of 10%. This value is far lower than the ~50% coefficient of variation for the duration of the 90% CP recovery bout. The mechanism of this fatigue is unclear. The four subjects fatigued at a VO2 averaging 89 ± 8% of their maximum but a heart rate of 100 ± 3% of their HRmax, compared with the two subjects who achieved the target at 86 ± 4% VO2 max and 96 ± 2% of HRmax (2 individual examples are shown in Figs. 2 and 4). It is possible, therefore, that...
the heart rate in the four subjects who fatigued reflects a condition, presumably (although not absolutely certainly) in the contracting muscles, that is not tolerable. This is despite a reduction in metabolic demand and oxygen pulse inferred from the \( VO_2 \) and heart rate responses (e.g., Fig. 4A). The falling oxygen pulse is consistent with stroke volume and/or the arteriovenous oxygen content difference being lower than that required for still-elevated metabolic demands. Further work is required to elucidate the mechanism(s) of this novel feature. We assume that the four subjects who could not recover sufficiently to meet the 20-min target would have been successful had the WR been lower in the domain.

\( W' \) has previously been demonstrated to be dependent (at least in part) on the glycogen storage status of the subject (17). Thus exercise designed to deplete \( W' \) will also reduce muscle glycogen content in the contracting units, but the degree to which this occurred in our subjects is unknown. It is well established that during WRs that induce fatigue in \( \sim 10 \) min, the average muscle glycogen content remains high at the point of exhaustion (e.g., Ref. 25). At WR ranges that demand between \( \sim 60 \) and \( 85\% \) \( VO_{2\max} \) (on the basis of the linear WR-\( VO_2 \) relationship from the ramp incremental test), muscle glycogen is almost completely depleted at fatigue (e.g., Ref. 4). Newsholme et al. (20), for example, have suggested that muscle glycogen limitation might even limit maximum performance at durations of \( \sim 30 \) min by using plausible estimates of muscle glycogen storage and utilization rates (13). Our subjects exercised for a total of \( \sim 19 \) min on average, of which \( 6 \) min was at a WR that resulted in \( 100\% \) of \( VO_{2\max} \) and an additional \( 13 \) min where \( VO_2 \) declined, but only to a value of \( \sim 88\% \) \( VO_{2\max} \). It is, therefore, possible that fatigue ensued in some subjects during the \( 90\% \) CP bout, because of the differences in muscle glycogen availability, but not in others. Furthermore, this degradation may be exacerbated in different muscle fiber types (26) and, therefore, may be dependent on an individual’s fiber-type profile and recruitment pattern during the exercise. Another possible mechanism for the differences observed is on the basis of lactate transport in the different individuals; this too, however, may be exacerbated by a different fiber-type profile (e.g., Ref. 8). Because the fiber-type characteristics of these subjects were not known, we hypothesized that parameters of aerobic fitness, such as \( VO_{2\max} \) or \( \theta_1 \), which are related to fiber-type proportion, or, conversely, anaerobic parameters, such as \( W' \), might provide a basis on which to distinguish between subjects. We were, however, unable to find any correlation between the tolerable duration of recovery at the \( 90\% \) CP WR and these parameters, but, of course, the subject number is small.

At the extremes, subject 5 exercised for a total of \( \sim 21 \) min before fatiguing (i.e., \( 365 \) s at WRexh and \( 916 \) s at \( 90\% \) CP), whereas subject 3 managed only \( \sim 7 \) min (\( 294 \) s at WRexh and \( 118 \) s at \( 90\% \) CP), which would appear to be too short a duration to deplete muscle glycogen stores by using the assumptions of Newsholme et al. (20). Another explanation, therefore, is that high-intensity exercise itself altered CP. CP has previously been shown to be dependent on state of training (7, 23) and on the inspired oxygen fraction, being increased in hyperoxia (31) and decreased in hypoxia (19, 31). In contrast, glycogen depletion affected \( W' \) only and not the value of CP (17). It is possible that if the fatiguing exercise itself (WRexh) affected the value of CP, then the recovery exercise may have been at a level \( \geq 90\% \) CP. We believe that this is unlikely, at least to within \( \pm 10\% \) of CP because exercise was not well tolerated at \( 110\% \) CP but was better tolerated at \( 90\% \) CP. It is not possible, however, to establish CP immediately after fatigue by using presently available techniques.

In conclusion, therefore, our findings demonstrate that functional recovery after fatiguing exercise is highly intensity dependent, and this has implications for the optimization of training strategies for athletic performance.

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DISCLOSURES

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REFERENCES


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