Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the “critical power”

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1Sport and Health Sciences, College of Life and Environmental Sciences, St. Luke’s Campus, University of Exeter, Devon, United Kingdom; 2Peninsula National Institute for Health Research Clinical Research Facility, St. Luke’s Campus, University of Exeter, Devon, United Kingdom; 3Human Performance Laboratory, Health Studies, Physical Education and Human Performance Sciences, Adelphi University, Garden City, New York; and 4Teachers College, Department of Biobehavioral Sciences, Columbia University, New York, New York

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Chidnok W, Fulford J, Bailey SJ, DiMenna FJ, Skiba PF, Vanhatalo A, Jones AM. Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the “critical power”. J Appl Physiol 115: 243–250, 2013. First published May 2, 2013; doi:10.1152/japplphysiol.00334.2013.—We tested the hypothesis that muscle high-energy phosphate compounds and metabolites related to the fatigue process would be recovered after exhaustion during recovery exercise performed below but not above critical power (CP) and that these changes would influence the capacity to continue exercise. Eight male subjects completed single-leg, knee-extension exercise to exhaustion (for ~180 s) on three occasions, followed by a work-rate reduction to severe-intensity exercise, heavy-intensity exercise (<CP), or a 10-min passive recovery period, in random order. The muscle metabolic responses to exercise were assessed using 31P magnetic resonance spectroscopy. There was a significant difference in the muscle metabolic responses between the heavy (~CP) and passive (<CP) conditions (at least 10 min and 39 ± 31 s, respectively; P < 0.05). During passive recovery and <CP recovery exercise, muscle phosphocreatine concentration ([PCR]) increased rapidly after the exhaustion point, reaching ~96% and ~76% of baseline values, respectively, after 10 min (P < 0.05). Moreover, pH increased abruptly, reaching 7.0 ± 0.0 and 7.0 ± 0.2, respectively, after 10 min recovery (P < 0.05). However, during >CP recovery exercise, neither muscle [PCR] nor pH recovered, reaching ~37% of the initial baseline and 6.6 ± 0.2, respectively. These results indicate that the muscle metabolic dynamics in recovery from exhaustive >CP differ according to whether the recovery exercise is performed below or above the CP. These findings confirm the importance of the CP as an intramuscular metabolic threshold that dictates the accumulation of fatigue-related metabolites and the capacity to tolerate high-intensity exercise.

Critical power; W’; exercise tolerance; constant work rate; fatigue

The physiological responses during constant work-rate (CWR) exercise are highly predictable depending on the exercise-intensity domain in which an individual is exercising (12, 21, 29, 30). The asymptote of the hyperbolic relationship between power output (P) and the time to exhaustion (Tlim) during high-intensity exercise [critical power (CP)] marks the boundary between the heavy (<CP)- and severe-intensity exercise domains [see (13) for review; 17, 18]. The CP therefore represents an important physiological threshold that approximates the so-called maximal lactate steady state (22, 24) and is considered to represent the highest sustainable rate of oxidative metabolism (13, 19). During sustained >CP, a slowly developing oxygen consumption (V̇O2) “slow component” will eventually result in the attainment of maximal V̇O2 (V̇O2max), with the Tlim attained shortly thereafter (9, 12, 21, 30). In the >CP, tolerance can be closely predicted based on the hyperbolic relationship between P and Tlim (13, 17, 18). The curvature constant of the power-duration hyperbola (W’) represents a fixed amount of work that can be performed above CP (13, 17, 18). This constancy of W’ for the entire range of supra-CP work rates means that Tlim during any high-intensity exercise coincides with the complete depletion of a fixed capacity for work above CP (W-_CP) (5, 8, 13, 17), the physiological determinants of which are uncertain (8, 13a, 26). Consistently low values of muscle phosphocreatine concentration ([PCR]) and pH have been reported at the limit of tolerance during CWR exercise above CP (14, 26).

It has been suggested that once the W’ has been exhausted and the Tlim attained during supra-CP exercise, the work rate must be reduced below the CP for W’ to be reconstituted and for exercise to be continued (7). Coats and coworkers (7) asked subjects to complete severe-intensity CWR to Tlim (attained in ~6 min) and then immediately reduced the work rate to 80% of the gas exchange threshold (GET), 90% CP, or 110% CP; the subjects then attempted to complete 20 min of exercise. It was reported that all six subjects completed the 20-min target time at 80% GET, only two subjects completed the 20-min target time at 90% CP, whereas none of the subjects completed the 20-min target time at 110% CP (mean ± SD exercise time: 30 ± 12 s). The authors interpreted these findings as evidence that the W’ recovers in an intensity-dependent manner following supra-CP exercise with important implications for exercise tolerance (7). Consistent with this, we have reported that recovery intervals between repeated severe-intensity work bouts enable the finite W’ to be restored, with the magnitude of this reconstitution related to the intensity of the recovery interval (6). Specifically, recovery work rates below CP allow for a partial recharge of W’, whereas “recovery” work rates above CP continue to deplete W’, albeit at a slower rate than is observed during the work interval (6, 23). However, the intramuscular bases for this intensity-dependent W’ recovery have yet to be investigated.

Therefore, the purpose of the present study was to use 31P-magnetic resonance spectroscopy (31P-MRS) to investigate the mechanistic bases for the intensity-dependent changes in the reconstitution of the W’ and exercise tolerance immediately.
following exhaustive severe-intensity exercise. We did this by assessing the responses of intramuscular phosphorus-linked metabolites and pH during recovery exercise performed at different intensities following severe-intensity CWR to exhaustion. We hypothesized that recovery exercise <CP would be sustainable for an appreciable duration (at least 10 min) after the exhaustive exercise and that muscle [PCr] and pH would be recovered significantly. We also hypothesized that exercise tolerance would be severely limited during recovery exercise >CP as a consequence of an inability to recover [PCr] and pH.

METHODS

Subjects. Eight male subjects (mean ± SD: age 23 ± 5 years, stature 1.78 ± 0.03 m, body mass 76.7 ± 8.2 kg) volunteered and gave written, informed consent to participate in this study, which was approved by the University of Exeter Research Ethics Committee. The subjects were all recreationally active and were familiar with the experimental procedures used in the study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h and having abstained from food, alcohol, and caffeine for the preceding 3 h. Testing was conducted at the same time of day (±2 h) for each subject, and laboratory visits were separated by at least 48 h.

Experimental overview. The study was conducted in two parts. The power–duration relationship was first established in the laboratory during single-leg, knee-extension exercise for each subject from four separate exercise bouts. From this relationship, the CP and W’ were estimated. Subsequently, with the use of the same ergometer, subjects performed the single-leg, knee-extension exercise to exhaustion within a magnetic resonance scanner. Muscle high-energy phosphate compounds and metabolites [PCr, ADP, inorganic phosphate (Pi), and pH] were assessed continuously at a severe-intensity CWR, estimated to result in exhaustion in 3 min (P; 26 ± 3 W; <153% of CP). Immediately following exhaustion, subjects underwent a 10-min passive recovery period, or the work rate was reduced to a >CP (21 ± 4 W) or <CP (13 ± 5 W) single-leg, knee-extension exercise. These conditions were presented in random order.

Part I: derivation of the power–duration relationship and estimation of CP and W’. The subjects initially completed four severe-intensity CWR single-leg, knee-extension exercise bouts at different work rates to determine the hyperbolic power–Tlim relationship. The work rates for the trials were selected to yield a range of Tlim, varying from ~2 min for the shortest trial to ~12 min for the longest trial (26). The exercise bouts were completed on separate days and presented in random order. Subjects were placed in a prone position and secured to the ergometer bed with Velcro straps at the thigh, buttocks, and lower back to minimize extraneous movement during the exercise protocol. The ergometer consisted of a nylon frame secured on top of the bed close to the subject’s feet and a base unit placed at the distal end of the bed. The subject’s right foot was connected to a rope running along the top of the frame to the base unit, on which a mounted pulley system allowed brass-weight plates to be lifted and lowered. Exercise was performed at the rate of 40 contractions/min, with the subject lifting and lowering the weight over a distance of ~0.22 m, in accordance with a visual cue presented on a monitor and an audible cue timed to the bottom of the down stroke. A shaft encoder (type BDK-06; Baumr, Swindon, UK) was fitted within the pulley system to record the distance traveled by the load, alongside a nonmagnetic load cell (type F250; Novatech Measurements, St Leonards On Sea, East Sussex, UK) to record applied force, which allowed the calculation of work rate.

During all exercise tests, the subjects were verbally encouraged to continue exercising for as long as possible. The Tlim, which was recorded to the nearest second, was defined as the time at which the subject could no longer keep pace with the required rate of P. Subjects were not informed of the work rates or their performance until the entire project had been completed. Individual CP and W’ estimates were derived from the prediction trial by least-squares fitting of the following regression models: nonlinear P vs. time (T)

\[ T = \frac{W}{(P - CP)} \]  
linear work (W) vs. time model

\[ W = CP \cdot T + W' \]  
linear P vs. 1/time model

\[ P = \frac{1}{T} \cdot W' + CP \]

The parameter estimates from Eqs. 1–3 were compared to ensure goodness of fit, and the model with the lowest standard error of the estimate (SEE) was chosen for further analysis (10). The 95% confidence intervals for the estimation of CP were used to calculate the work rates that were just below and just above the CP. We reasoned that this approach would provide reasonable assurance that the work rates were truly below and above the CP for all subjects.

Part II: 31P-MRS assessment of muscle metabolic responses to high-intensity exercise. After completion of the predictive trials for estimation of the CP and W’, the subjects reported to the MRS laboratory at the Peninsula Magnetic Resonance Research Unit (Exeter, UK) on three separate sessions. Exhaustive severe-intensity exercise was performed with simultaneous measurement of muscle metabolic responses by 31P-MRS using a 1.5 T superconducting magnetic resonance scanner (Intera; Philips, Amsterdam, the Netherlands) and using the same ergometer as for part I. To collect the 31P data during the exercise protocol within the scanner, a 6-cm 31P transmit/receive surface coil was placed within the ergometer bed, and the subject was positioned such that the coil was centered over the quadriceps muscle of the right leg. Initially, fast-field echo images were acquired to determine correct positioning of the muscle relative to the coil. Placement of cod-liver oil capsules, which yield high-intensity signal points within the image adjacent to the coil, allowed its orientation relative to the muscle volume under examination to be assessed. A number of pre-acquisition steps were carried out to optimize the signal from the muscle under investigation. Tuning and matching of the coil were performed to maximize energy transfer between the coil and the muscle. An automatic shimming protocol was then undertaken within a volume that defined the quadriceps muscle to optimize homogeneity of the local magnetic field, thereby leading to maximal signal collection.

Subjects were required to exercise to Tlim at a severe-intensity CWR, predicted to result in P1 (with the use of the CP and W’ derived from part I; P = [W/Tlim] of 180 s (T50) + CP). Immediately following exhaustion, subjects either: 1) continued to perform exercise at an above CP (>CP) or below CP (<CP) work rate, with exercise continued for 10 min or as long as possible, or 2) ceased exercise completely for a 10-min period. The three tests were undertaken on separate days in random order. During the entire exercise and recovery periods, 31P data were acquired every 1.5 s with a spectral width of 1,500 Hz. Phase cycling with four phase cycles was used, leading to a spectrum acquired every 6 s. The subsequent spectra were quantified by peak fitting with the assumption of prior knowledge, using the advanced method for accurate, robust, and efficient spectral (AMARES) fitting algorithm in the jMRUI (version 3) software package. Spectra were fitted with the assumption that Pi, PCr, ATP, and phosphodiester peaks were present. In all cases, relative amplitudes were corrected for partial saturation due to the repetition time relative to T1 relaxation time. The T1 saturation was corrected via a spectrum consisting of 48 individually acquired spectra that were acquired with a long relaxation time before the beginning of data acquisition.

Intracellular pH was calculated using the chemical shift of the Pi peak relative to the PCr peak (25). The [PCr] and Pi concentration
Table 1. Work rates and limit of tolerance during exhaustive severe-intensity constant work-rate (CWR) exercise and subsequent resting or exercising [heavy- and severe-intensity exercise (<CP and >CP, respectively)] recovery

<table>
<thead>
<tr>
<th></th>
<th>Resting</th>
<th>&lt;CP</th>
<th>&gt;CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>CWR exercise bout, W</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tlim, during CWR exhaustive exercise, s</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>W−CP, during CWR exhaustive exercise, kJ</td>
<td>171 ± 22</td>
<td>182 ± 26</td>
<td>173 ± 23</td>
</tr>
<tr>
<td>Recovery WR, W</td>
<td>0 ± 0</td>
<td>13 ± 5</td>
<td>21 ± 4</td>
</tr>
<tr>
<td>Tlim, during CWR recovery exercise, s</td>
<td></td>
<td>600 ± 0</td>
<td>39 ± 31*</td>
</tr>
</tbody>
</table>

Data are means ± SD. Tlim, time to exhaustion; W−CP, work above CP.

*Significantly different from resting and <CP (P < 0.05).

Table 2. Muscle metabolic responses during severe-intensity CWR exercise and subsequent recovery at different intensities

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>CWR Exercise</th>
<th>End-Recovery Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>[PCr], %</td>
<td>100 ± 0</td>
<td>40 ± 8*</td>
<td>96 ± 4*†</td>
</tr>
<tr>
<td>&lt;CP</td>
<td>100 ± 0</td>
<td>40 ± 7*</td>
<td>76 ± 12†‡</td>
</tr>
<tr>
<td>&gt;CP</td>
<td>100 ± 0</td>
<td>38 ± 6*</td>
<td>37 ± 8**§</td>
</tr>
<tr>
<td>pH</td>
<td>7.0 ± 0.1</td>
<td>6.7 ± 0.2*</td>
<td>7.0 ± 0.0†</td>
</tr>
<tr>
<td>&lt;CP</td>
<td>7.0 ± 0.0</td>
<td>6.7 ± 0.2*</td>
<td>7.0 ± 0.2†</td>
</tr>
<tr>
<td>&gt;CP</td>
<td>7.0 ± 0.0</td>
<td>6.7 ± 0.2*</td>
<td>6.6 ± 0.2*‡§</td>
</tr>
<tr>
<td>[Pi], %</td>
<td>100 ± 0</td>
<td>586 ± 105*</td>
<td>68 ± 21†</td>
</tr>
<tr>
<td>&lt;CP</td>
<td>100 ± 0</td>
<td>524 ± 117*</td>
<td>178 ± 105†</td>
</tr>
<tr>
<td>&gt;CP</td>
<td>100 ± 0</td>
<td>534 ± 115*</td>
<td>545 ± 133**§</td>
</tr>
<tr>
<td>[ADP], μM</td>
<td>6 ± 1</td>
<td>51 ± 12*</td>
<td>9 ± 3*†</td>
</tr>
<tr>
<td>&lt;CP</td>
<td>7 ± 2</td>
<td>54 ± 16*</td>
<td>23 ± 12*‡</td>
</tr>
<tr>
<td>&gt;CP</td>
<td>7 ± 1</td>
<td>55 ± 14*</td>
<td>51 ± 10*§</td>
</tr>
<tr>
<td>[ATP], %</td>
<td>100 ± 0</td>
<td>83 ± 5*</td>
<td>93 ± 6*†</td>
</tr>
<tr>
<td>&lt;CP</td>
<td>100 ± 0</td>
<td>82 ± 7*</td>
<td>87 ± 6*</td>
</tr>
<tr>
<td>&gt;CP</td>
<td>100 ± 0</td>
<td>85 ± 4*</td>
<td>79 ± 4*§</td>
</tr>
</tbody>
</table>

Data are means ± SD. [PCr], phosphocreatine concentration; [Pi], inorganic phosphate concentration; [ADP] and [ATP], ADP and ATP concentration, respectively. *Significantly different from Baseline; †significantly different from End exercise; ‡significantly different from Rest; §significantly different from <CP.
CP recovery exercise, muscle [PCr] and [ATP] remained stable with time until exercise was terminated (37% and 79% of the initial baseline, respectively; P < 0.05). There was no further change in [Pi] and [ADP] between the exhaustion point of the initial exhaustive bout and the CP recovery condition, and vertical error bars show mean ± SD [PCr] at end of exhaustive exercise and subsequent “recovery” exercise. *End-recovery muscle [PCr] was significantly lower for CP and <CP recovery compared with resting (P < 0.05). #End-recovery muscle [PCr] was significantly lower for CP recovery compared with resting and <CP recovery (P < 0.05).

In the recovery period, the muscle [PCr], [ATP], [Pi], [ADP], and pH were significantly different among conditions (Table 2). End-recovery muscle [PCr], [ADP], and [Pi] were significantly different for CP recovery compared with resting and <CP recovery and for <CP recovery compared with resting recovery conditions (P < 0.05). In addition, there was a significant difference in end-recovery exercise [ATP] and pH for CP recovery compared with resting and <CP recovery (P < 0.05; Table 2). The group mean muscle metabolic response profiles are depicted in Figs. 1–4.

**DISCUSSION**

The principal novel finding of this investigation was that the muscle metabolic response profile (as assessed by 31P-MRS) and Tlim differed significantly when the exercise intensity used in the recovery from exhaustive exercise was positioned in different intensity domains (CP and <CP). The results of the study were consistent with our hypotheses and indicate that CP recovery exercise can be sustained for an appreciable

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**Fig. 1.** Muscle phosphocreatine concentration ([PCr]) responses to constant work-rate (CWR) severe-intensity exercise and subsequent recovery exercise for resting (black circles), heavy-intensity exercise (<CP; gray circles), and >CP (open circles) conditions. Horizontal error bars show mean ± SD time to exhaustion (Tlim) for the initial exhaustive exercise bout and the >CP recovery condition, and vertical error bars show mean ± SD [PCr] at end of exhaustive exercise and subsequent “recovery” exercise. *End-recovery muscle [PCr] was significantly lower for >CP and <CP recovery compared with resting (P < 0.05). #End-recovery muscle [PCr] was significantly lower for >CP recovery compared with resting and <CP recovery (P < 0.05).

**Fig. 2.** Muscle pH responses to CWR severe-intensity exercise and subsequent recovery exercise for resting (black circles), <CP (gray circles), and >CP (open circles) conditions. Note that the recovery of pH is dependent on the rate of [PCr] recovery (see Fig. 1). Horizontal error bars show mean ± SD Tlim for the initial exhaustive exercise bout and the >CP recovery condition, and vertical error bars show mean ± SD pH at end of exhaustive exercise and subsequent recovery exercise. *End-recovery muscle pH was significantly lower for >CP recovery compared with resting (P < 0.05). #End-recovery muscle pH was significantly lower for >CP recovery compared with resting and <CP recovery (P < 0.05).
duration without significant fatigue development after initial exhaustive exercise, with muscle [PCr] and pH increasing significantly in the recovery period. However, exercise tolerance was severely limited during CP recovery exercise, and there was no recovery in intramuscular high-energy phosphate compounds or metabolites until exercise was terminated. The results indicated that replenishment of the following the limit of tolerance necessitated that work rate be reduced to allow some of the fatigue-related, high-energy phosphate compounds or metabolites to be resynthesized (e.g., ATP and PCr) or cleared from the muscle [e.g., hydrogen ion (H\(^+\))], thereby offsetting the “limiting” intramuscular environment (1, 14, 26).

There was no significant difference in \( W_{>CP} \) across the three conditions during the initial exhaustive exercise bouts (approximately 1.44–1.55 kJ), and these \( W_{>CP} \) were not significantly different from the subjects’ \( W' \) (1.52 kJ). This is consistent with previous studies that have reported that \( W_{>CP} \) equals \( W' \) at the limit of tolerance during severe-intensity exercise and confirms the notion that in this domain, the limit of tolerance is reached when subjects expend the finite work capacity available above CP; i.e., the \( W' \) (8, 9, 21, 30). Historically, the \( W' \) has been considered to represent a finite amount of energy available from oxygen stores (e.g., in blood and tissue), the high-energy phosphates, and a source related to anaerobic glycolysis that may be expended above the CP (17, 18). An
alternative perspective is that the W' is related to the accumulation or depletion of one or more metabolites or substrates that are linked to the process of muscle fatigue until some ‘critical’ concentration is attained, beyond which the same work rate cannot be tolerated (7, 8, 13a, 14, 26). Consistent with this and with the previous study of Vanhatalo et al. (26), the present study shows that $T_{lim}$, during a series of severe CWR exercise bouts, was associated with the attainment of consistently low values of muscle [PCr] and pH and consistently high values of muscle [P] and [ADP]. We have proposed previously that changes in muscle [PCr], [ADP], [P], and H$^+$ concentration ([H$^+$]) are linked to the use of the W' during severe-intensity exercise, with these perturbations in the muscle metabolic milieu also driving the continued development of the VO$_2$ slow component (3, 13, 28). In this way, exercise intolerance during severe-intensity exercise is associated with the complete use of the W', the attainment of some critical combination of muscle high-energy phosphate compound and/or metabolite concentrations (of which [PCr], [ATP], [ADP], [P], and [H$^+$]) may act directly or serve as proxies), and the achievement of V'$_{O2}$max (3, 5, 13, 20, 21, 28).

To our knowledge, the present study is the first to investigate the muscle metabolic responses to recovery exercise performed in different exercise-intensity domains (i.e., below and above CP) following exhaustive exercise. During resting recovery, a steady state in muscle metabolites was achieved relatively rapidly. For example, muscle [PCr] and [ATP] had recovered, on average, to ~96% and ~93% of their respective baseline values, and pH had increased to the resting value within 10 min. Following 10 min of recovery exercise, performed at a work rate that was below CP, muscle [PCr] and [ATP] had recovered, on average, to ~76% and ~87% of their respective baseline values, and pH had increased by 0.2 U from the value recorded at exhaustion. In all cases, the subjects were able to complete 10 min of <CP recovery exercise. The muscle metabolic response to recovery exercise performed above CP after exhaustive CWR exercise was markedly different from the response observed below the CP and in resting conditions. During >CP recovery exercise, despite a 19% reduction in work rate, [PCr] and [ATP] remained stable at ~37% and ~79% of their respective baseline values at the end of the recovery work rate (Fig. 1). Moreover, there was no further change in the muscle pH, [P], and [ADP] between the exhaustion point and the end of recovery exercise. These results support the notion that reconstitution of intramuscular metabolites following $T_{lim}$ necessitates that the work rate be reduced below CP (6, 7, 8, 19).

Our results are consistent with the study of Coats et al. (7). These authors asked subjects to perform severe CWR cycle exercise to $T_{lim}$ (attained in ~6 min) and then reduced the work rate to 80% GET, 90% CP, or 110% CP. The results suggested that replenishment of the W' following $T_{lim}$ necessitated that work rate be reduced <CP (7). The results of the present study are also consistent with our previous study (6), in which we reported that recovery intervals between severe-intensity work bouts enabled the finite W' to be restored, with the magnitude of this reconstitution being related to the intensity of the recovery interval. That the W' is expended during work bouts >CP and reconstituted during recovery intervals <CP may be understood with reference to the study of Jones et al. (14). With the use of $^{31}$P-MRS, these authors showed that when CWR exercise was performed slightly above CP, [PCr] and pH decreased and [P] increased until $T_{lim}$ was reached. During exercise performed just below the CP, however, stable values for [PCr], pH, and [P] were attained within 3 min of the start of exercise, suggesting significant metabolic reserve (14). The results of the present investigation confirm that <CP recovery after exhaustive severe-intensity exercise allows some of the fatigue-related, high-energy phosphate compounds and metabolites to be recovered (e.g., ATP and PCr) or cleared from the muscle (e.g., H$^+$), thereby delaying the attainment of a limiting intramuscular environment (11, 14, 26).

In the present study, the tolerable duration for >CP recovery exercise after the initial exhaustion was ~39 s, which would have required the expenditure of a further ~0.17 kJ of W'$_{>CP}$. Theoretically, there was no opportunity for W' to be reconstituted between the work bouts, given that the work rate remained above the CP (8). The ability of the subjects to sustain exercise for any duration is therefore surprising. It should be considered that this additional W'$_{>CP}$ might be related to variability in the estimation of W' or to a less than complete use of the W' in the initial exhaustive exercise bout. However, the additional W'$_{>CP}$ was significantly greater than the standard error associated with the estimation of W', significantly greater than the ~0.04 kJ of the W', theoretically left “unexpended” after the initial exhaustive exercise bout (due to the $T_{lim}$ of ~173 s being slightly shorter than the predicted $T_{130}$), and not significantly correlated with the variability in the $T_{lim}$ for the initial exhaustive exercise bout. That all the subjects in the present study were able to continue for some period of time during >CP recovery following initial exhaustion suggests that this is a “real” phenomenon that cannot be explained by experimental error. It is of interest that this finding is consistent with the study of Coats et al. (7), in which cycle exercise at a lower severe-intensity work rate could be continued for 30 ± 12 s following initial exhaustion.

The explanation for this ability of subjects to continue to exercise for some (albeit limited) duration following a reduction of work rate within the severe domain is unclear. It is possible that although the subjects were unable to maintain the required work rate at the point of exhaustion in the initial CWR exercise bout, the small reduction in the target work rate allowed a small, further reserve of W' to be used, thus extending the net tolerable duration of >CP exercise. In other words, fatigue impacted the maximal rate at which the W' could be expended during the initial CWR exercise bout, and the decrease of the work rate at the point of exhaustion reduced the rate of W' expenditure required and enabled a further small reserve of W' to be expended. Whereas a constant W' is an implicit assumption in the conventional two-parameter hyperbolic CP model that is derived from several CWR exercise bouts performed to the limit of tolerance, it is possible that different rules apply in other types of exercise tests and/or when work rate is manipulated close to the limit of tolerance (when W' tends toward zero). Consistent with this, repeated maximal voluntary contractions result in a progressive reduction in maximum torque until the “critical torque” is attained (2, 4), indicating that fatigue impacts on maximal force generating capacity. Also, during a 3-min, all-out cycling sprint, the P (and therefore, the maximum rate of W' expenditure) declines with time as fatigue accumulates until the CP is attained (27, 28). These studies indicate that the maximal rate
of W’ expenditure falls as fatigue develops but that W’ continues to be used (albeit at a progressively slower rate) until it is exhausted completely, at which point, the sustainable P = CP. In this model, prior fatiguing sprint exercise simultaneously reduces both the W’ and the peak P that can be achieved during a subsequent 5-min, all-out test (27), further suggesting that there may be a link between the absolute W’ and the peak rate at which it can be expended.

In light of these findings, the classical definition of W’ as a work capacity that is not rate-limited (17–19, 21) might require reconsideration. W’ may instead be more accurately described as a finite work capacity above CP, whose maximum rate of expenditure is reduced progressively as the size of W’ remaining decreases with continued exercise >CP. Theoretically, W’ might be, at least to some extent, protocol dependent, with the W’ estimated from a series of CWR tests being slightly less than the W’, which may be available if work rate is reduced (within the severe domain) as exhaustion approaches. Another factor that may contribute to the subjects’ ability to continue to exercise for a short time >CP following initial exhaustion is that the reduction in work rate altered muscle tension and may have reduced afferent signaling from mechanoreceptors and perhaps the perception of effort, at least temporarily, thereby facilitating an extension of exercise (1). However, it is noteworthy that intramuscular high-energy phosphate and metabolite concentrations were not altered significantly during recovery exercise >CP, such that afferent traffic from metaboreceptors may not have been reduced.

In conclusion, the results of this study indicate that the muscle metabolic responses and exercise tolerance during recovery from exhaustive exercise can be understood with reference to the CP concept. The dynamics of the muscle metabolic response to recovery exercise following exhaustive severe-intensity exercise can be differentiated according to whether the recovery exercise is performed below or above the CP. Specifically, the <CP recovery exercise can be sustained for an appreciable duration without significant fatigue development after the exhaustive exercise, with, for example, muscle [PCr] and pH increasing significantly and rapidly after the initial point of exhaustion. However, exercise tolerance is limited during >CP recovery exercise, wherein intramuscular, high-energy phosphate compound and metabolite concentrations remained stable with time until exercise was terminated. This provides further evidence for the importance of the CP in determining the ability to maintain intramuscular homeostasis and the capacity to tolerate high-intensity exercise.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS


REFERENCES


