Effect of recovery duration from prior exhaustive exercise on the parameters of the power-duration relationship

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Ferguson C, Rossiter HB, Whipp BJ, Cathcart AJ, Murgatroyd SR, Ward SA. Effect of recovery duration from prior exhaustive exercise on the parameters of the power-duration relationship. J Appl Physiol 108: 866–874, 2010. First published January 21, 2010; doi:10.1152/japplphysiol.91425.2008.—The physiological equivalents of the curvature constant (W′) of the high-intensity power-duration (P-tLIM) relationship are poorly understood, although they are presumed to reach maxima/minima at exhaustion. In an attempt to improve our understanding of the determinants of W′, we therefore aimed to determine its recovery kinetics following exhaustive exercise (which depletes W′) concomitantly with those of O2 uptake (VO2,a = which depletes W′) recovery, respectively. The W′ = t1/2, H11006 hyperbola, with CP unchanged. However, W′ = tLIM: a ramp and four constant-load tests, at different work rates, for estimation of lactate threshold, W′, critical power (CP), and maximum VO2. Three further exhausting tests were performed at different work rates, with each preceded by an “conditioning” bout, with intervening recoveries of 2, 6, and 15 min. Neither prior exhaustion nor recovery duration altered VO2 or [L−] at tLIM. Post-conditioning, the P-tLIM relationship remained well characterized by a hyperbola, with CP unchanged. However, W′ = tLIM = 37 ± 5, 65 ± 6, and 86 ± 4% of control following 2, 6, and 15 min of intervening recovery, respectively. The W′ recovery was curvilinear [interpolated half time (t1/2) = 234 ± 32 s] and appreciably slower than VO2 recovery (t1/2 = 74 ± 2 s) but faster than [L−] recovery (t1/2 = 1,366 ± 799 s). This suggests that W′ determines supra-CP exercise tolerance, its restitution kinetics are not a unique function of phosphocreatine concentration or arterial [L−], and it is unlikely to simply reflect a finite energy store that becomes depleted at tLIM. Critical power; curvature constant; lactate clearance; oxygen uptake (59). However, the physiological basis of W′ remains controversial.

W′ is defined through the relationship between P and tLIM; in this sense it is itself a “simple” mathematical value. However, its value presumably reflects some physiological variable (or variables) that (with CP) determines tLIM. It is commonly suggested that the physiological equivalents of W′ are represented by a finite energy store comprising intramuscular high-energy phosphates [ATP and phosphocreatine (PCr)], glycogen, and stored O2 (14, 38–40, 42, 45, 46). This coheres with the suggestion by some investigators that W′ is synonymous with the maximum O2 deficit (24) or anaerobic work capacity (23, 42; although see Ref. 61) and that it is depleted at a rate that bears some proportionality to the magnitude of the power requirement above CP, with exhaustion occurring when this apparent store is depleted (42, 45). More recently, however, W′ has been proposed to reflect the build-up of fatigue-inducing metabolites (e.g., the intramuscular accumulation of inorganic phosphate and H+ and the interstitial/extracellular accumulation of K+) to some critical tolerable limit (7, 13, 28, 45). This, in turn, would require the rate of metabolite build-up to be proportionally coupled to the rate of W′ “utilization” (7).

Establishing the physiological correlate(s) of W′ is complicated by the difficulty in resolving whether their profiles of change during exercise (be it depletion or accumulation) are reflected in the rate of utilization of W′. It seems likely that the putative physiological equivalents would reach some minimum (or maximum) value at the point at which exercise intolerance is reached, with W′ then being mathematically defined. However, whether a predictable relationship [either linear or some more complex function (e.g., 46)] between W′ at its equivalents during exercise can be made requires knowledge of the physiological underpinnings of the W′ construct. That is, the relationship between W′ and its putative physiological determinants might be established by independent experimental manipulation of W′, with CP remaining constant. Here we propose a strategy to achieve this based on our recent demonstration that W′ was reduced following a conditioning bout of supra-CP exercise with an intervening 2-min recovery, whereas key parameters of aerobic function (e.g., the fundamental VO2 time constant, VO2 max, and CP) were unaffected (10). Consequently, redefining the P-tLIM relationship after exhaustive supra-CP exercise with a range of intervening recovery durations may provide a means of establishing the recovery kinetics of W′ in concert with those of its putative physiological determinants. It is suggested that, by establishing the kinetics of W′ and its physiological equivalents (rather than simply a single W′ value at the point of intolerance, as has been the case to date), insight would be provided into the determinants of exercise tolerance.
Consequently, the goal of this study is to determine the kinetics of W’ recovery from exhausting supra-CP constant-load cycle ergometry and to compare them with those of \( \text{VO}_2 \) [a reasonable proxy for intramuscular PCR kinetics (e.g., 48)] and arterialized-capillary blood [L-] (a broad index of recovery arterial lactate clearance). We hypothesized that the degree to which these quantities were kinetically correlated with W’ would allow judgments to be made regarding the involvement of putative energy-store and fatigue-metabolite changes in the restitution and, therefore, composition of W’.

METHODS

Subjects

Six recreationally active, healthy men (24 ± 4.2 yr old, 179.6 ± 7.5 cm height, 86.5 ± 15.3 kg body wt) provided written informed consent to participate in the study. The procedures and protocols were approved by the School of Biomedical Sciences/School of Sport and Exercise Sciences (University of Leeds) Ethical Review Committee and were conducted in accordance with the Declaration of Helsinki.

Equipment

The equipment is described in detail in an earlier report (10). Briefly, subjects exercised on a computer-controlled, electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands). Ventilatory and pulmonary gas exchange variables were determined breath-by-breath using mass spectrometry (MSX, NSpire, Kent, UK) and turbinometry (Interface Associates, Laguna Niguel, CA). The volume and gas concentration signals were sampled and digitized every 20 ms and time aligned for on-line breath-by-breath measurement of ventilatory and pulmonary gas exchange variables (4). Heart rate was calculated beat-by-beat from the R-R interval of a 12-lead electrocardiogram (Quest, Burdick, WA), and arterial \( \text{O}_2 \) saturation was monitored noninvasively using finger pulse oximetry (Biologic 3745, Ohmeda, Louisville, KY).

Protocols

Subjects were initially familiarized with all protocols and procedures. For each subject, only one exercise test was conducted on a given day (therefore requiring a total of ~14 visits/subject), with each individual participating in no more than three experimental sessions in any given week. Prior to each session, subjects were asked to refrain from participation in strenuous physical activity (preceding 24 h), alcohol ingestion (preceding 48 h), and caffeine ingestion (preceding 3 h) and to arrive at least 2 h postprandial. All testing was commenced from a 20-W baseline (2 min) and concluded with a final 20-W recovery phase (2 min).

Protocol 1: maximal incremental ramp test. A 25 W/min incremental ramp test was performed to the tolerable limit, defined as the point at which subjects were unable to maintain a cycling cadence of 60 rpm despite encouragement (Fig. 1A). Peak \( \text{VO}_2 \) (\( \text{VO}_2\text{peak} \)) was determined as the average \( \text{VO}_2 \) for an integral number of breaths over the final ~20 s of the incremental phase, and the lactate threshold (\( \text{LT} \)) was estimated (\( \text{LT}_1 \)) using standard pulmonary gas exchange criteria (62).

Protocol 2: determination of the control P-\( t_\text{LIM} \) relationship. Each subject then completed a randomized series of four constant-load tests to the limit of tolerance, with each test implemented at a different WR chosen to span a \( t_\text{LIM} \) range of ~3–12 min (Fig. 1B). CP and W’ were estimated as the power intercept and slope, respectively, of the least-squares linear regression of P vs. \( t_\text{LIM} \), i.e., \( P = (W/t_\text{LIM}) + \text{CP} \) (23, 45, 60). The standard error (SE) of each individual CP estimation was less than ±3 W and that for W’ was less than ±1.25 kJ. The WR predicted to induce exhaustion at 6 min (WR\(_6\)) was derived by interpolation and, subsequently, used as the WR of the exhaustive “conditioning” exercise bout in protocol 3.

Protocol 3: effects of recovery duration on the postconditioning P-\( t_\text{LIM} \) relationship. The purpose of this protocol was to estimate the recovery kinetics of W’ concomitantly with those of \( \text{VO}_2 \) and arterialized blood [L-]. The P-\( t_\text{LIM} \) relationship was therefore redefined following a conditioning bout of constant-load exercise performed to the limit of tolerance at WR\(_c\), for each of the three intervening 20-W recovery durations (i.e., 2, 6, and 15 min; Fig. 1C). For each recovery condition, subjects completed three supra-CP constant-load exercise tests to the limit of tolerance (cf. Protocol 2), where possible at WRs that corresponded to those previously utilized in Protocol 2. As a result of postconditioning reductions in W’, on occasion some of the higher WRs performed during the control P-\( t_\text{LIM} \) characterization would have been of too short a duration to be meaningfully used for determination of the postconditioning P-\( t_\text{LIM} \) relationships (Fig. 2). To constrain attainment of \( t_\text{LIM} \) to within the recommended range of ~3–12 min (e.g., 14, 45, 46), in these instances the WRs in question therefore had to be decreased slightly. Tests were randomly assigned.

Blood Sampling

At designated points in all protocols (Fig. 1, arrows), capillary blood samples (~25 \( \mu \)l) were taken from the fingertip of the heated hand and analyzed immediately after the test for [L-] using an automated analyzer (GM-7, Analox Instruments, London, UK). Before analysis of each set of blood samples, the analyzer was calibrated.
using an 8 mM standard solution, the concentration of which was also checked postanalysis.

**Analysis**

Each breath-by-breath data set was edited to eliminate any occasional extraneous breaths (more than ±4 SD of the local mean) (34). For control tests (Protocol 2), baseline VO2 was calculated as the mean VO2 over the final 60 s of the 20-W warm-up phase. Following the exhaustive conditioning bout at WR6, the “baseline” VO2 was calculated as the mean VO2 recorded in the last 20 s of recovery prior to the imposition of the subsequent supra-CP constant-load test. VO2 peak was calculated as the average VO2 for an integral number of breaths over the last ~20 s prior to attaining the limit of tolerance. As VO2 peak was found not to vary over the range of different work rates performed for any subject (see RESULTS), the mean VO2 peak value was taken as VO2 max (8). VO2 max and the duration of the conditioning bout (conducted at WR6) were each monitored throughout the study to check for the presence of any training effect. These were each found to vary randomly within the day-to-day limits of 10% reported by Day et al. (8) for similarly intense fatiguing constant-load exercise, from which we concluded that our results were not influenced by a training effect.

We did not attempt to formally partition individual VO2 responses into their kinetic components (e.g., 28, 44), as the low signal-to-noise ratio for single transitions (because of breath-to-breath variability) (34) and the absence of a discernible steady state or asymptote in VO2 precluded an acceptable level of statistical confidence for the parameter estimations. Even single repeats of each of the tests would have required an unacceptably large number of studies (i.e., given the initial ~14 tests typically required of each subject). Not only would this have been overly demanding for the subjects, but it would also be quite likely to introduce a training effect.

**Statistical Analysis**

The significance of the effect of recovery duration following exhausting exercise at WR6 on the parameters of the P-tLIM relationship (i.e., CP and W′), VO2, and [L−] were compared using repeated-measures ANOVA and post hoc analysis (Tukey’s honestly significant difference) where necessary. Values are means ± SD unless otherwise stated.

**RESULTS**

**Protocol 1: Maximal Incremental Ramp Test**

VO2 peak averaged 3.82 ± 0.55 l/min, while b1 averaged 1.87 ± 0.35 l/min (equivalent to 49 ± 5% VO2 peak).

**Protocol 2: Control P-tLIM Relationship**

The P-tLIM relationship for each subject conformed well to a hyperbola, as demonstrated by the goodness-of-fit of the P-tLIM-1 relationship to a linear function (R2 > 0.996 in all cases). The SE of each CP estimation was, on average, 1.7 W (range 1.2–2.4 W) and that of W′ was, on average, 0.48 kJ (range 0.34–0.69 kJ; Fig. 2) (cf. Refs. 7, 10). CP and W′ averaged 212 ± 34 W and 21.6 ± 5.2 kJ, respectively (Table 1), and were unaltered when power was set as the independent variable [CP = 212 ± 34 W (P = 0.999), W′ = 21.73 ± 6.26 kJ (P = 0.875)] (cf. Refs. 7, 10). WR6 interpolated from the P-tLIM-1 relationship averaged 269 ± 34 W. VO2 peak attained during these constant-load tests was not influenced by WR (P = 0.426), thus meeting the criterion for VO2 max, which averaged 3.78 ± 0.56 l/min for the group as a whole (Table 1). [L−] at the limit of tolerance ([L−]lim) was also not influenced by WR (P = 0.203) and averaged 10.14 ± 0.97 mM (Table 1), representing an increase (Δ[L−]) from the preexercise (20-W) baseline of 9.09 ± 1.16 mM.

**Table 1. Group mean values of maximum O2 uptake, CP, W′, and [L−] at [L−]lim in protocols 2 and 3**

<table>
<thead>
<tr>
<th>Protocol 2</th>
<th>2 min Rec</th>
<th>6 min Rec</th>
<th>15 min Rec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum O2 uptake, l/min</td>
<td>3.78 ± 0.56</td>
<td>3.64 ± 0.50</td>
<td>3.76 ± 0.50</td>
</tr>
<tr>
<td>CP, W</td>
<td>212 ± 34</td>
<td>213 ± 36</td>
<td>213 ± 36</td>
</tr>
<tr>
<td>SE of CP estimate, W</td>
<td>1.7 (1.2–2.4)</td>
<td>1.1 (0.5–2.1)</td>
<td>1.1 (0.1–2.3)</td>
</tr>
<tr>
<td>W′, kJ</td>
<td>21.6 ± 5.16</td>
<td>7.8 ± 1.40</td>
<td>14.1 ± 3.70</td>
</tr>
<tr>
<td>SE of W′ estimate, kJ</td>
<td>0.48 (0.34–0.69)</td>
<td>0.20 (0.01–0.33)</td>
<td>0.26 (0.01–0.54)</td>
</tr>
<tr>
<td>[L−]lim, mM</td>
<td>10.14 ± 0.97</td>
<td>10.38 ± 1.34</td>
<td>10.77 ± 1.15</td>
</tr>
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</table>

Values are means ± SD, except critical power (CP) and curvature constant (W′) estimates, which are shown as mean SE and range. [L−]lim, lactate concentration measured at the limit of tolerance. Protocol 2, control values; Protocol 3, values obtained after exhaustive conditioning exercise at work rate predicted to induce exhaustion at 6 min (WR6) with intervening 20-W recoveries of 2, 6, and 15 min (2 min Rec, 6 min Rec, and 15 min Rec). *Significantly different from Protocol 2 (P < 0.05). †Significantly different from 2 min Rec (P < 0.05). ‡Significantly different from 6 min Rec (P < 0.05).
Protocol 3: Effects of Recovery Duration on the Postconditioning P-tLIM Relationship

The tolerable duration of the WR₆₆₆ conditioning bout averaged 366 ± 21 s, which was not significantly different between subjects (P = 0.537) or between each of the nine bouts within subjects (P = 0.635). VO₂peak (3.81 ± 0.52 l/min) in the conditioning bout was not significantly different from VO₂max (P = 0.969). [L⁻][tLIM] was also not significantly different between each of the nine conditioning bouts (P = 0.884) or from the control value obtained during Protocol 2 (i.e., 10.14 ± 0.97 mM, P = 0.288), averaging 10.11 ± 1.02 mM (Table 2), which was equivalent to Δ[L⁻] of 9.06 ± 1.16 mM.

Following the supra-CP conditioning bout, tLIM at any particular WR was systematically reduced compared with control (Protocol 2) and to an extent that was more marked the shorter the intervening 20-W recovery duration, averaging 38 ± 8, 65 ± 5, and 84 ± 5% of the control duration following intervening 20-W recoveries of 2, 6, and 15 min, respectively. The P-tLIM relationship remained hyperbolic, with the R² of the P⁻tLIM⁻¹ linear transform being >0.994 and the SE of CP and W' estimates remaining within ±3 W and 1.25 kJ, respectively (see Table 1 for mean and range of values). CP was unchanged from control values (Protocol 2, 212 ± 34 W), regardless of the intervening recovery duration (P = 0.922), averaging 213 ± 36, 213 ± 34, and 213 ± 36 W following 20-W recoveries of 2, 6, and 15 min, respectively (Figs. 2 and 3, Table 1).

In contrast, compared with control values, W' was significantly reduced (P = 0.001) in each of the postconditioning cases by an amount that was highly dependent on the intervening recovery duration (Figs. 2 and 3). Following the 2-min 20-W recovery, W' averaged 7.8 ± 1.4 kJ, equivalent to a 37 ± 5% recovery, which was significantly lower than the control estimate (Protocol 2, P = 0.001). The 6-min recovery resulted in a greater depletion of W', to 14.1 ± 3.7 kJ (corresponding to 65 ± 6% recovery). However, although this value attained at 6 min was significantly greater than that obtained following the 2-min recovery (P = 0.002), W' remained significantly lower than control (Protocol 2, P = 0.001). Following the 15-min recovery, W' averaged 18.5 ± 4.6 kJ, equivalent to 86 ± 4% depletion. However, although this is significantly greater than the level to which W' recovered following both the 2-min (P = 0.001) and 6-min (P = 0.001) recoveries, it was still significantly lower than the control estimate (Protocol 2, P = 0.001; Table 1).

The baseline value to which VO₂ recovered following the conditioning bout at WR₆₆₆ (i.e., prior to the onset of the postconditioning supra-CP bouts) was significantly dependent on the intervening recovery duration (P = 0.001; Fig. 4). That is, following the 2-min 20-W recovery, VO₂ averaged 1.37 ± 0.20 l/min, which was significantly higher than the control 20-W baseline obtained prior to the onset of WR₆₆₆ (0.74 ± 0.08 l/min, P = 0.001). There was a further significant, although still incomplete, recovery of VO₂ after the 6-min 20-W recovery period (i.e., to 1.06 ± 0.12 l/min) compared with the 2-min 20-W recovery (P = 0.001). VO₂ also continued to recover throughout the 15-min 20-W recovery phase to values significantly lower than those obtained during both the 2-min (P = 0.001) and 6-min (P = 0.001) recovery phases (i.e., to 0.87 ±

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Table 2. Blood [L⁻] at the limit of tolerance of the exhaustive conditioning bout (WR₆₆₆) and after 2, 6, and 15 min of 20-W recovery

<table>
<thead>
<tr>
<th>Subj No.</th>
<th>[L⁻], mM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Postconditioning recovery</td>
</tr>
<tr>
<td></td>
<td>WR₆₆₆</td>
</tr>
<tr>
<td>1</td>
<td>11.25</td>
</tr>
<tr>
<td>2</td>
<td>10.09</td>
</tr>
<tr>
<td>3</td>
<td>10.61</td>
</tr>
<tr>
<td>4</td>
<td>9.11</td>
</tr>
<tr>
<td>5</td>
<td>8.69</td>
</tr>
<tr>
<td>6</td>
<td>10.93</td>
</tr>
</tbody>
</table>

Mean ± SD 10.11 ± 1.02 10.00 ± 0.91 9.09 ± 1.28 6.43 ± 1.58†

WR₆₆₆, limit of tolerance of exhaustive conditioning bout; [L⁻], blood lactate concentration. *Significantly different from WR₆₆₆ (P < 0.05). †Significantly different from 6 min Rec (P < 0.05).

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Fig. 3. CP (A) and W' (B) under control conditions (Protocol 2) compared with estimates obtained following exhaustive conditioning exercise and intervening 20-W recoveries of 2, 6, and 15 min (Protocol 3). Individual values for each subject are compared against line of identity. Note that CP was unchanged following supra-CP conditioning exercise, conducted to the tolerable limit, regardless of the intervening recovery duration, while W' was consistently reduced and highly dependent on the intervening recovery duration.
end-exercise WR6 value, although this was not statistically significant ($P = 0.061$). The 15-min recovery period allowed a further decrease in [L⁻] compared with the end-exercise WR6 value ($P = 0.001$), averaging $6.43 \pm 1.58$ mM prior to the onset of the second phase of the protocol (Table 2). However, even following the 15-min 20-W recovery, [L⁻] remained significantly elevated compared with the control 20-W baseline value ($P = 0.001$; Figs. 6B and 7, Table 2). Furthermore, in contrast to VO₂, recovery of [L⁻] was slower than that of W' (Fig. 5), with an interpolated $t_{1/2} = 1366 \pm 799$ s (because of the slow [L⁻] recovery relative to the recovery time frame, for simplicity, the $t_{1/2}$ estimation assumed that the immediately subsequent [L⁻] recovery followed the same profile that was observed between 6 and 15 min). This temporal discrepancy is evident in the proportional recoveries of W' and [L⁻] (Fig. 6B).

VO₂ peak at the end of Protocol 3 (i.e., at the limit of tolerance in the 2nd bout of Protocol 3) was not influenced by WR following either the 2-min ($P = 0.053$), 6-min ($P = 0.436$), or 15-min ($P = 0.071$) recovery (again meeting the VO₂ max criterion; see Table 1 for VO₂ max values). Furthermore, post-conditioning VO₂ max was invariant across each of the recovery durations and corresponded to VO₂ max attained from control supra-CP constant-load tests (Protocol 2, $P > 0.05$; Fig. 4). VO₂ attained at the limit of tolerance following 2 min of recovery was $0.17 \pm 0.16$ l/min lower than that attained during the WR6 conditioning bout ($P = 0.015$) but remained well within the expected test-retest variability of ~10% (8). Similarly, [L⁻] max, attained following intolerance in either the conditioning bout or subsequent bouts, was similar between all protocols (Table 1, Fig. 7).

**DISCUSSION**

This, we believe, is the first study to attempt characterization of W’ recovery kinetics and its putative physiological equivalents for supra-CP exercise. The data demonstrate that W’ reconstitution following exercise is curvilinear (cf. Ref. 46), as determined by supra-CP exercise to tLIM after an exhausting conditioning bout and subsequent variable recovery periods,
being progressively reduced the shorter the intervening recovery (20-W) period, while CP remained unchanged. The constancy of CP means that the reduction in tLIM following the exhaustive conditioning exercise with variable recovery durations (2–15 min) is solely dependent on \( W = \) . This extends our previous work (10), which used a single 2-min recovery period, and suggests that \( W = \) reconstitution is not linear (as inferred from its mathematical calculation) but follows a trajectory that lies somewhere between the physiological surrogates for the “depletion” and “accumulation” hypotheses measured here: neither \( \dot{V}_{O_2} \) nor \([L^-]/H_11002 \) recovery well represented the \( W = \) profile. However, given that \( W = \) appears only to be replenished during exercise if recovery WRs are below CP (allowing predominantly aerobic energy transfer (7, 14)), these observations together demonstrate that \( W = \) depletion alone shapes the tolerance of supra-CP exercise, thus justifying preliminary attempts to gain insight into its physiological determinants.

That exhaustive supra-CP conditioning exercise did not affect CP or, indeed, \( \dot{V}_{O_2\max} \), is consistent with our earlier findings (10) and those from others using heavy-intensity (28) and sprint (22, 58) conditioning. Thus \( W = \) depletion during the conditioning bout does not appear to influence CP, in contrast to previous suggestions (7). Although this supports the “anaerobic” constitution of \( W = \), it does not rule out \( W = \) being related to aerobic energetic parameters via its influence on the energetic equivalency of CP; that is the rate of ATP turnover to sustain CP may not be constant. For example, \( W = \) depletion by prior supra-CP exercise has been reported to reduce work efficiency (52) and, as such, may play a role in increasing the aerobic metabolic equivalent of CP and, thus, limiting the “role” of \( W = \) in inducing intolerance in a subsequent bout. Therefore, \( W = \) might better reflect influences related to fatigue induction, rather than those solely of anaerobic energy-transfer origin.

We suggested that insight into the physiological determinants of \( W = \) might be derived from its temporal recovery profile relative to those of \( \dot{V}_{O_2} \) [a reasonable proxy for intramuscular PCr kinetics (48)] and \([L^-]/H_11002 \) (a broad, albeit coarse, index of blood lactate clearance under these conditions) following exhausting supra-CP conditioning exercise. While these profiles were each clearly curvilinear (Fig. 5), we did not attempt formal kinetic characterization (e.g., exponential, hyperbolic, or some more complex function), given the small number of data points in each subject data set (\( n = 3 \)). Rather, we confined ourselves to simply estimating the interpolated \( t_{1/2} \) and inferring what these correlations may suggest regarding the constitution of \( W = \) with respect to depletion of putative substrate pools and/or accumulation of key fatigue-inducing metabolites.

Phosphocreatine

Intramuscular PCr stores have been proposed to be one of the major determinants of \( W = \) (14, 38, 40, 42, 45, 46, 55). The
classical characterization of intramuscular PCr concentration ([PCr]) kinetics following fatiguing exercise in terms of parallel “fast” and “slow” components (18) coheres with the more recent description of VO2 recovery kinetics from very high-intensity exercise in terms of “fundamental” and “slow” component elements (44). Furthermore, [PCr] kinetics both during and following high-intensity exercise have been shown to be closely correlated with those of VO2 (e.g., 48), the later providing a close estimate of muscle O2 consumption kinetics (2, 17). It might therefore be reasonably supposed that the VO2 recovery profile following supra-CP conditioning was reasonably reflective of the corresponding intramuscular [PCr] profile. However, it should be noted that it is only the fraction of the entire intramuscular PCr pool actually expended above CP that would be involved in W′ replenishment in the subsequent recovery. Indeed, while the fractional recoveries of W′ and VO2 were linearly related following fatiguing exercise, W′ clearly recovered more slowly than VO2 (t1/2 = 234 ± 32 s; Figs. 5 and 6A).

W′ recovery relative to that of VO2 recovery was relatively linear over the range investigated (Fig. 6A). However, its linear extrapolation to the VO2 axis suggests that, following the attainment of VO2max at tLIM, no W′ recovery could occur until VO2 had recovered by some 60%. We consider this to be an unlikely scenario, as W′ is known to recover whenever WR falls below CP (7, 14). Also, this extrapolated value is approximately equal to the group mean (1.87 ± 0.35 l/min). A more curvilinear W′ recovery profile (Fig. 6A, dashed-dotted line) thus seems more likely, although the precise relationship is an issue requiring resolution. Regardless, the discrepancy between the W′ and VO2 recovery kinetics seems to suggest that if PCr is indeed a determinant of W′, then other mechanisms with slower recovery kinetics also contribute.

Muscle Glycogen

As one such source has been proposed to be the intramuscular glycogen store (14, 40, 42, 45, 46), it would be of interest to establish whether glycogen loading can increase W′. Glycogen depletion has been shown to decrease W′ (39), although it an ~50–70% decrease in muscle glycogen (20) has been demonstrated to result in only an average 20% decrease in W′ (39) and 2) the global muscle glycogen content is unlikely to be limiting over the exercise durations we utilized (53). However, the heterogeneous properties and recruitment patterns of muscle raise the possibility that a degree of fiber-type dependent regional glycogen depletion (16, 57) may account for (or contribute to) the ~15% shortfall in W′ reconstitution that was evident by the end of the 15-min recovery (Fig. 5). The exact mechanism(s) through which such glycogen depletion might be expressed is uncertain but may include 1) a reduced pyruvate availability that constrains mitochondrial respiration rate, independently of any effect on pyruvate dehydrogenase (37) and of anaplerotic reactions leading to falls in key citric acid cycle intermediates (e.g., 51), and 2) impaired excitation-contraction coupling consequent to factors such as reduced sarcoplasmic reticulum Ca2+ release (1). Regardless, the large dissociation between the decrease in muscle glycogen (20) and the decrease in W′ (39), coupled with evidence suggesting that intramuscular ATP concentration is unchanged (e.g., 48) and the total intramuscular PCr store is not completely depleted at tLIM (e.g., 29, 31, 49) (although PCr depletion in individual fibers, reflecting intramuscular heterogeneities, is of course possible), suggests that it is unlikely that W′ is simply reflecting a source of stored energy that is depleted during supra-CP exercise.

Lactic Acidosis

The role of the fatigue mediators L− (e.g., 9, 25) and H+ (e.g., 11) in W′ repletion kinetics is unclear, as we were not able to monitor intramuscular levels and regional distributions of L− and H+. Studies from the literature suggest that muscle [L−] is likely still to be falling at the 15-min recovery point (e.g., 21, 50), although the constraints on biopsy sampling limit descriptions to only a few points dispersed over the entire recovery. The modeling analysis of Freund and Zoulimou (12) indicates a slightly more rapid recovery of muscle [L−] relative to blood [L−], although the assumptions of lactate release rate from the recovering muscles bearing a simple proportionality to muscle [L−] (suggestive of a unitary process) and of the muscle itself operating as an homogenous structure are known to be oversimplifications. We are confined to noting that recovery of blood [L−] (interpolated t1/2 = 1,366 ± 799 s) was substantially slower than recovery of W′ and that there was no clear proportionality in the magnitude of [L−] recovery relative to W′ recovery (Fig. 6B). This suggests that lactate recovery (and possibly glycogen repletion) is ongoing when W′ is fully recovered, consistent with earlier reports of slow blood [L−] recovery kinetics (e.g., 12, 15, 35).

We can only speculate on the extent to which the recovery profile of intramuscular [L−] might resemble that of blood [L−] under the highly non-steady-state conditions of the present study. However, any involvement of intramuscular L− clearance in W′ recovery kinetics is likely to be complex, including, for example, 1) clearance to the interstitium via sarcolemmal monocarboxylate transporter (MCT) 1 and MCT4 action (e.g., 5, 6, 30), free diffusion, and nonspecific anion exchange (reviewed in Refs. 15, 35), 2) subsequent L− uptake and oxidation into adjacent oxidative fibers via MCT1-mediated transport (e.g., 19; cf. Ref. 63) and diffusion (reviewed in Refs. 6, 15, 35). Even so, simply on kinetic grounds, it seems unlikely that intramuscular L− clearance is an exclusive mediator of W′ restoration.

Establishing the profile of intramuscular [H+] is even more problematic. A considerable proportion (~70%) of H+ clearance takes place in an MCT1- and MCT4-mediated 1:1 coupling with L− (e.g., 5, 6, 30). Other routes include 1) the influence of intramuscular PCO2, for which an appreciable transient increase has been reported early in recovery (e.g., 32), 2) intramuscular H+ production related to the rapid rates of PCr resynthesis early in recovery following the exhaustive conditioning bout (33), 3) sarcolemmal Na+/H+ -mediated H+ efflux (reviewed in Ref. 30), and 4) bicarbonate-mediated H+ buffering in muscle and blood (with carbonic anhydrase exerting an important facilitating action on reaction rate) (3).

Substrate Depletion and Metabolite Accumulation

As discussed above, W′ is traditionally suggested to represent the depletion of putative substrate pools (e.g., 23, 42). We hypothesized that determining the recovery kinetics of such pools, such as might be reflected in the temporal profiles of VO2 as an index of intramuscular PCr kinetics (e.g., 48), for
example, might provide pointers elucidating the constitution of \( W^* \). Our data suggest, however, that a model of \( W^* \) as a “depletable” energy pool is likely to be too simple. Rather, the complex recovery kinetics of \( W^* \) suggest that it might be better reflected in the integrated action of variables that contribute to fatigue via the accumulation of key fatigue-inducing metabolites, such as 1) inorganic phosphate through its effects on sarcoplasmic reticulum \( \mathrm{Ca}^{2+} \) handling (e.g., 1), 2) increased extracellular \( K^+ \) concentration, which predisposes to sarcosomal depolarization and compromised membrane excitability (54), and 3) the possibility of intramuscular oxidative stress (reviewed in Ref. 56), or 4) an interaction of these effects. Our results suggest that subsequent studies might therefore be focused on identifying specific fatigue-inducing metabolites that have been postulated to represent \( W^* \), via procedures such as the serial sampling of muscle tissue by biopsy and of muscle-venous blood, as well as magnetic resonance spectroscopy of muscle (e.g., 17, 18, 48, 52).

**Assumptions**

While \( W^* \) is a mathematical construct implicit in the hyperbolic \( P_{\text{TLM}} \) relationship (14, 42, 45), it is one that compels physiological elucidation. A fundamental assumption in the present study was that the supra-CP conditioning bout fully depleted \( W^* \) at the tolerable limit or that its mediator(s) is depleted to a low, but limiting, value. Furthermore, as we are aware of no technique that is available for direct assessment of the time course of \( W^* \) recovery following fatiguing exercise, it was also assumed that the \( W^* \) estimate obtained from each of the postconditioning \( P_{\text{TLM}} \) relationships reflected the value to which \( W^* \) would have recovered by the end of the intervening recovery period; i.e., it was assumed that there was no further recovery during the postconditioning bout itself. This assumption is supported by the demonstration that no significant amount of work can be performed at a lower WR within the very heavy-intensity domain (i.e., supra-CP) immediately following attainment of the tolerable limit, suggesting that the “flux” of \( W^* \) is unidirectional above CP (7, 43).

We also cannot rule out the possibility that the recovery profile of \( W^* \) following exhaustive exercise may have been modified as a result of an altered work efficiency effect (52) on the \( W^* \) depletion rate. However, currently available techniques do not allow for a more direct estimation of the rate of \( W^* \) utilization during volitional exercise.

**Conclusions**

The retained hyperbolic \( P_{\text{TLM}} \) relationship, with unchanged CP, following fatiguing exercise supports the assertion that \( W^* \) depletion shapes high-intensity exercise tolerance. We suggest that the complexity of its recovery kinetics may be more consistent with \( W^* \) reflecting the accumulation of fatigue-related metabolites, rather than expenditure of a simple source of stored energy. However, it is also possible that an integrated function of these processes is involved in determining \( W^* \). Resolution of these issues will require more invasive targeting of particular mechanisms.

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