W' Reconstitution Accelerates More with Decreasing Intensity in the Heavy versus the Moderate Intensity Domain

Maarten Lievens¹², Kevin Caen¹², Jan G. Bourgois¹², Kobe Vermeire¹, Jan Boone¹²

¹Department of Movement and Sports Sciences, Ghent University, Ghent, Belgium; ²Department of Physical Rehabilitation and Medicine, Center of Sports Medicine, Ghent University Hospital, Ghent, Belgium

Accepted for Publication: 17 November 2020
W' Reconstitution Accelerates More with Decreasing Intensity in the Heavy versus the Moderate Intensity Domain

Maarten Lievens\textsuperscript{1,2}, Kevin Caen\textsuperscript{1,2}, Jan G. Bourgois\textsuperscript{1,2}, Kobe Vermeire\textsuperscript{1}, Jan Boone\textsuperscript{1,2}

\textsuperscript{1}Department of Movement and Sports Sciences, Ghent University, Ghent, Belgium;
\textsuperscript{2}Department of Physical Rehabilitation and Medicine, Center of Sports Medicine, Ghent University Hospital, Ghent, Belgium

CORRESPONDING AUTHOR:
Jan Boone
Watersportlaan 2, 9000 Ghent, Belgium
+32 (0)9 264 6302
Jan.Boone@ugent.be

Maarten Lievens and Kevin Caen share first authorship.

The authors received no financial support for the research, authorship, and/or publication of this article. \textbf{CONFLICT OF INTEREST:} Results of the present study do not constitute endorsement by the American College of Sports Medicine and are presented clearly, honestly,
and without fabrication, falsification, or inappropriate data manipulation. No conflicts of interest, financial or otherwise, are declared by the authors.
ABSTRACT

Introduction. The purpose was to investigate the effect the recovery intensity domain on $W'$ reconstitution. We used the $W'_{\text{BAL}}$ model as a framework and tested its predictive capabilities ($W'_{\text{PRED}}$) across the different intensity domains. Methods. Twelve young men (51.7 ± 5.9 mL·kg$^{-1}$·min$^{-1}$) completed a ramp incremental test, three to five constant power output (PO) tests to determine critical power (CP) and $W'$ and minimally two trials to verify the maximal lactate (La$'$) steady state. During four experimental trials, subjects performed two work bouts (WB1 and WB2) at P6 (i.e., PO that predicts exhaustion within 6 min) separated by a recovery interval at CP – 10 W, Δgas exchange threshold (GET)-CP, GET and 50% GET, respectively. WB1 was designed to deplete 75% $W'$ and the recovery time varied in order to replenish 50% $W'$. WB2 was performed to exhaustion ($W'_{\text{ACT}}$). $W'_{\text{PRED}}$ was compared with $W'_{\text{ACT}}$ to evaluate the accuracy of the $W'_{\text{BAL}}$ model. Excess post-exercise oxygen consumption (EPOC) was calculated as the difference between the measured and predicted oxygen uptake during recovery. Results. $W'_{\text{ACT}}$ averaged 49%±24%, 69%±24%, 81%±28% and 93%±21% for respectively CP – 10 W, ΔGET-CP, GET and 50% GET ($P=0.002$). $W'_{\text{PRED}}$ overestimated $W'_{\text{ACT}}$ in CP-10W (34%±32%, $P=0.004$) and underestimated $W'_{\text{ACT}}$ in 50% GET (24%±28%, $P=0.013$). EPOC was lowest in CP – 10 W ($P<0.01$) and higher in GET compared to ΔGET-CP ($P=0.01$). Conclusion. We demonstrated that $W'_{\text{PRED}}$ overestimated and underestimated $W'_{\text{ACT}}$ in the heavy and moderate intensity domain, respectively. Therefore, the practical applicability of a single recovery time constant, which only relies on the difference between the recovery PO and CP, is questionable.

Key Words: Critical Power, $W'$, Recovery, Intensity domain
INTRODUCTION

The power-time relationship provides a framework to explore and better understand the physiological responses to exercise (1). This relationship can be mathematically described by a hyperbolic function in which the asymptote, termed the critical power (CP), sets the demarcation between the heavy and severe intensity domain, and the curvature constant $W'$ (J) represents a fixed amount of work that can be performed above CP (2).Interestingly, the moment of task failure in the severe intensity domain seems to be accompanied by a consistent level of metabolic perturbation (i.e., accumulation of metabolites and depletion of substrates), that is independent of the exercise intensity, suggesting that fatigue occurs when a ‘critical threshold’ at the level of the working muscles is reached (3–5). For many years, the CP concept could only be applied to continuous exercise until the kinetics of $W'$ recovery were incorporated in a model for intermittent exercise (6). The ability to track the energetic reserve capacity of the body during exercise is promising with regard to the optimization of training and even racing strategies (7). In 2012, Skiba et al. proposed a refined model in which the $W'$ reconstitution is assumed to occur in a curvilinear manner (8):

\[ W'_{\text{BAL}} = W' - \int_{0}^{t} (W'_{\text{EXP}})(e^{-(t-u)/\tau_{W'}}) \, du \]

In this model, the dynamic balance of $W'$ ($W'_{\text{BAL}}$) at any time ($t$) is calculated by reducing the subjects known $W'$ with the amount of $W'$ that has already been expended ($W'_{\text{EXP}}$) during efforts above CP. The recharge of $W'$ becomes possible when the power output (PO) drops below CP and is believed to follow a mono-exponential course of which the speed is determined by the recovery time constant ($\tau_{W'}$). The calculation of $\tau_{W'}$ takes into account the amplitude between the recovery PO and CP (i.e., $D_{\text{CP}}$):
\[ \tau_{W'} = 546e^{-0.01D_{CP}} + 316 \]

As expected from the \( W'_{BAL} \) model, the \( W' \) reconstitution and/or the ability to continue after exhaustive exercise is enhanced by either decreasing the recovery PO (9–11) or increasing the recovery duration (12,13). Although the concept of \( W'_{BAL} \) is innovative, several studies suggested that its current modelling tends to underestimate actual \( W' \) reconstitution (\( W'_{ACT} \)), especially after short recovery and/or short interval durations (5,14,15). From a practical point of view, inaccurate estimations of the available amount of \( W' \) during exercise may lead to suboptimal training stimuli (i.e., too hard or too low) or race performance (i.e., early exhaustion or not using your full potential).

One explanation for the inaccuracy of the current \( W'_{BAL} \) model might be that \( D_{CP} \), the parameter that determines \( \tau_{W'} \), does not take into account the intensity domain in which the recovery takes place. For example, a recovery interval with a \( D_{CP} \) of 50 W might be situated in the heavy intensity domain for one athlete but in the moderate intensity domain for another. Neglecting the impact of the recovery intensity domain seems incorrect given the markedly different contribution of energy systems and different mechanisms for fatigue development during exercise within these domains (3,16). In this context, the oxygen uptake (\( \bar{VO}_2 \)) is amongst the most studied physiological responses to characterize the onset and offset of exercise (16,17).

During the recovery of intense exercise, \( \bar{VO}_2 \) does not instantly return to baseline levels, a phenomenon well-known as excess post-exercise oxygen consumption (EPOC) (18). EPOC is linked to the restoration of metabolic processes such as the refilling of the tissue \( O_2 \) stores, removal of lactate (\( La^- \)) and \( H^+ \), and resynthesis of glycogen (19). As such, EPOC can be
considered as a measure for the oxidative metabolic reserve capacity and therefore, it is likely that differences in EPOC during recovery exercise across different intensity domains may attribute to the inaccuracy of $W'_{\text{BAL}}$.

The purpose of this study was to investigate the effect of the intensity domain in which the recovery takes place on $W'$ reconstitution. We used the $W'_{\text{BAL}}$ model as a predictive framework and hypothesized that $W'$ reconstitution is not only dependent on $D_{\text{CP}}$ per se but also depends on the relative intensity (i.e., the intensity domain) of the recovery interval. More specifically, we hypothesized that $W'_{\text{ACT}}$ would be underestimated by $W'_{\text{PRED}}$ in the moderate intensity domain (gas exchange threshold (GET) and 50% GET) (5,14,15). Additionally, we hypothesized that $W'_{\text{ACT}}$ would be overestimated by $W'_{\text{PRED}}$ in the heavy intensity domain (CP - 10 W and $\Delta \text{GET-CP}$) since we expected a lack of metabolic oxidative reserve (measured as EPOC) to efficiently recover $W'$.

**METHODS**

**Subjects**

Twelve male physical education students (26.1 ± 2.6 yr, 1.82 ± 0.06 m, 75.6 ± 9.9 kg) were recruited to voluntarily take part in this investigation. All subjects were active in recreational sports and trained cyclists were not included in the study. All participants declared to be in good health and were accustomed to maximal exercise efforts. Before the onset of the study, each subject provided written informed consent. This study was approved by the ethical committee of the Ghent University Hospital (Belgium).
Experimental protocol

All tests were executed on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) in a controlled laboratory environment. Room air temperature was set at 19°C with a relative humidity of 50%. A fan was provided during tests with a duration of > 20 min. For each subject, testing was conducted at the same time of the day (± 2 h) and separated with at least 48 h. During all tests, heart rate (HR) was continuously monitored (H7 sensor, Polar, Kempele, Finland) and pulmonary gas exchange was registered on a breath-by-breath basis (Metalyzer 3B, Cortex, Leipzig, Germany). Subjects were encouraged to maintain their regular activity pattern but were instructed to refrain from strenuous exercise 24 h prior to testing. All subjects completed the study protocol within a period of 7 ± 1 weeks.

Ramp incremental test. During the first visit, a ramp incremental test was performed to exhaustion, which was defined as the inability to maintain the preferred cadence (between 70 and 90 rpm) for five consecutive seconds despite strong verbal encouragement. The test started with 6 min of cycling at 100 W in order to calculate the VO₂ mean response time (MRT) (see data analysis) (20). After 2 min of rest, subjects cycled an additional 4 min at 50 W before the PO increased at a rate of 30 W•min⁻¹ until exhaustion. Subjects were instructed to remain seated during pedaling and to keep their self-selected cadence constant during this and all following tests.

CP tests. CP and W’ were determined from a randomized series of three to five exhaustive constant PO tests. The intensities were chosen to provoke exhaustion within 2 to 20 min and were derived from specific percentages of the individual peak power output (PO_peak) of the ramp
incremental test (i.e., 70%-100% $\text{PO}_{\text{peak}}$) (21,22). Each trial started with a 4-min warm-up at 50 W followed by an abrupt increase to the target PO. Time to exhaustion (TTE) was manually recorded.

**MLSS tests.** A minimum of two 30-min constant PO tests were performed to determine MLSS with the aim of physiologically verifying CP as the upper limit of the heavy intensity domain (23,24). Each trial started with a 4-min warm-up at 50 W followed by an abrupt increase to the target PO (i.e., CP for the first test). Capillary blood samples were taken from the fingertip at minute 0, 5, 10, 15, 20, 25, and 30 to analyze blood $[\text{La}^-]$ (Biosen C-line, EKF-diagnostic GmbH, Barleben, Germany). If $[\text{La}^-]$ increased $> 1.0 \text{ mmol} \cdot \text{L}^{-1}$ between the 10th and 30th min of exercise, the PO of the following test was decreased by 10 W. In case $[\text{La}^-]$ increased $\leq 1.0 \text{ mmol} \cdot \text{L}^{-1}$, the PO of the subsequent test was increased by 10 W. This procedure was repeated until MLSS could be determined (25).

**Experimental trials** (Fig. 1). During four randomized experimental trials, subjects performed two work bouts (i.e., WB1 and WB2) separated with a varying recovery interval (REC). After a 4-min warm-up at 50 W, the PO was abruptly increased to an intensity corresponding to P6 (i.e., the PO that causes exhaustion after 6 min according to the CP model). At this intensity, the subjects had to cycle for 270 s in order to deplete 75% of $W'$ (i.e., $W'_{\text{PRED}}= 25\%$) (WB1). Previous research of our group has shown that the model-predicted TTE of P4 and P8 exercise did not differ from the actual TTE in a similar population (5). Immediately after WB1, the PO was reduced to one of four randomly chosen recovery intervals (REC) including CP - 10 W, ΔGET-CP, GET and 50% GET. The recovery time at this PO was individually calculated by
means of the $W'_{\text{BAL}}$ model in order to replenish 50% of $W'$ (i.e., $W'_{\text{PRED}} = 75\%$) (8). Immediately following REC, PO abruptly increased to P6 for a second WB (WB2), but this time until exhaustion. Capillary blood samples were taken immediately after WB1 and WB2 and during the last 10 s of recovery to determine [La−].

**Data analysis**

**Ramp incremental test.** PO$_{\text{peak}}$ and HR$_{\text{peak}}$ were defined as the highest values and $\dot{V}O_{2\text{peak}}$ as the highest 30-s average obtained during the test. GET was identified as the point at which [1] carbon dioxide output ($\dot{V}CO_2$) increased disproportionate to oxygen uptake ($\dot{V}O_2$) (i.e., V-slope method), [2] the first departure from the linear increase in minute ventilation ($\dot{V}E$), and [3] an increase in $\dot{V}E/\dot{V}O_2$ without a concomitant increase in $\dot{V}E/\dot{V}CO_2$ (26). GET was determined from visual inspection by three independent researchers. In case of conflicting results, data were reexamined until a consensus was reached. The $\dot{V}O_2$/PO relationship was corrected for the individual MRT which takes into account the time delay between energy demand at the level of the working muscles and the expression of elevated $\dot{V}O_2$ at the lungs. To calculate the MRT, [1] a linear regression was fitted from the point of the first systemic rise in $\dot{V}O_2$ up to GET and [2] the steady state $\dot{V}O_2$ at 100W was superimposed on the regression to calculate the corresponding PO. As such, the MRT was retrieved as the difference in PO between the ramp-identified PO and 100 W (20,27):

$$\text{MRT (W)} = (\text{[\dot{V}O}_2 \text{ at 100 W - intercept]} / \text{slope}) - 100 \text{ W}$$

**CP tests.** CP and $W'$ were determined using four regression models: the linear work-time model 

$[W_{\text{lim}} (J) = W' (J) + CP (W) \times t (s)]$, the linear inverse-of-time model 

$[PO (W) = W' (J) \times (1/t (s)) + CP (W)]$, the hyperbolic two-parameter power–time model 

$[t (s) = W' (J) / (PO (W) - CP (W))]$
and the hyperbolic three-parameter power–time model \[ t (s) = \left( \frac{W' (J)}{PO (W) - CP (W)} - \left( \frac{W' (J)}{P_{max} (W) - CP (W)} \right) \right) \] (22). Only trials that lasted between 2 and 20 min and with a minimum difference of 5 min between the shortest and longest trial were included in the analysis (22,28).

An end-exercise \( \dot{V}O_2 > 95\% \dot{V}O_{2peak} \) was set as an additional criterion to include trials in the models (29). For all models, the \( CV\% \) associated with CP (criterion < 5\%) and \( W' \) (criterion < 10\%) were calculated and expressed as the total error of the PO-t relationship (30). All possible combinations of trials, with a minimum of three trials per model, were calculated (22). The model that resulted in the smallest total error was chosen for each individual (i.e., “best individual fit approach”) (30,31).

**MLSS.** MLSS was defined as the highest PO that could be maintained for 30 min without an increase in [La\textsuperscript{-}] of more than 1.0 mmol\cdot L\textsuperscript{-1} between the 10\textsuperscript{th} and 30\textsuperscript{th} min of exercise (32). In subjects in which the PO at CP differed from the PO at MLSS, the original CP was substituted by the PO at MLSS (i.e., CP-corrected) and the size of \( W' \) was recalculated (\( W' \)-corrected) using the following steps:

\[
P_6 (W) = W' (J) \times \left( \frac{1}{360 \text{ (s)}} \right) + CP (W)
\]

\[
W' \text{-corrected (J)} = (P_6 (W) - CP \text{-corrected (W)}) \times 360 \text{ (s)}
\]

This procedure assured that each recovery condition, especially CP - 10 W, for each subject was situated below the highest PO at which a metabolic steady state could be achieved.

**Experimental trials.** The protocol was designed to deplete 75\% of \( W' \) during WB1 (CP model), and recover 50\% of \( W' \) during REC (\( W'_{\text{BAL}} \) model). The predicted TTE of WB2 was therefore
270 s, the equivalent of expanding 75% $W'$ at P6. $W'_{ACT}$ was calculated by dividing the TTE of WB2 by the theoretically expected TTE of WB1 (i.e., 360 s). For each subject, the average $W'_{ACT}$ was calculated across the four conditions. To investigate the effect of the recovery intensities across the different recovery durations, $W'_{ACT}$ and $W'_{PRED}$ were transformed into a mean actual ($W'_{ACT}$-RATE) and mean predicted ($W'_{PRED}$-RATE) recovery rate, respectively. To do so, the true $W'$ reconstitution (i.e., $W'_{ACT}$ - 25% and $W'_{PRED}$ - 25%, respectively) was expressed per unit of time ($J\cdot s^{-1}$). $\dot{V}O_2$ and HR values of WB1, REC and WB2 were expressed relatively to the peak values obtained during the ramp incremental test. Additionally, the temporal profile of $\dot{V}O_2$ and [La$^-$] during REC was quantified. EPOC during REC was calculated as the difference between the predicted steady-state $\dot{V}O_2$ and the measured $\dot{V}O_2$ integrated over time (33). The predicted steady-state $\dot{V}O_2$ was derived from the individual $\dot{V}O_2$-PO relationship. For intensities below or equal to the PO at GET, a linear regression was fitted based on the ramp incremental test. This linear regression started from the point of the first systemic rise in $\dot{V}O_2$ up to GET and the PO was left-shifted to correct for the MRT (20). For intensities above GET, a second regression was established between the $\dot{V}O_2$ response associated with the MRT-corrected PO at GET and the $\dot{V}O_2$ and PO at MLSS (derived from the 30-min test) in order to account for the loss of mechanical efficiency (i.e., $\dot{V}O_2$ slow component) (27). Lactate kinetics were quantified as net [La$^-$] clearance (mmol•L$^{-1}$) (i.e., [La$^-$]$_{WB1}$ - [La$^-$]$_{REC}$) and net [La$^-$] clearance rate (mmol•L$^{-1}$•min$^{-1}$) (i.e. ([La$^-$]$_{WB1}$ - La$^-$]$_{REC}$) • t$_{REC}^{-1}$).

**Statistical analysis**

Descriptive data are presented as mean ± SD for n = 12 participants. Normal distribution of all variables was demonstrated by the Shapiro–Wilk test. A paired sample $t$-test was conducted to
compare the model-predicted CP and MLSS. One-way (4 recovery conditions) repeated-measures (RM) ANOVA’s with pairwise comparison were performed to investigate differences in end-physiological measures (End-\(\bar{V}O_2\), End-HR, [La’]) [WB1 and WB2] and performance parameters (\(W'_\text{ACT}, W'_\text{ACT-RATE}\)) [WB2]. Paired samples \(t\)-tests were used to check for differences with physiological peak values (\(\bar{V}O_2\text{peak}, \text{HR}_{\text{peak}}\)) [WB1 and WB2] and model-predicted performance (\(W'_{\text{PRED}}, W'_{\text{PRED-RATE}}\)) [WB2]. To identify differences in REC characteristics (\(D_{CP}, \text{duration}\)) and physiological characteristics (End-\(\bar{V}O_2\), EPOC, End-HR and [La’]) one-way RM ANOVA’s (4 recovery conditions) with pairwise comparison were performed. Statistical analyses were executed using SPSS 25 (IBM Corp., Armonk, NY). \(P\) values < 0.05 were considered statistically significant.

**RESULTS**

\(\bar{V}O_2\text{peak}\) of the ramp incremental test averaged 3875 ± 372 mL\(\cdot\)min\(^{-1}\) (51.7 ± 5.9 mL\(\cdot\)kg\(^{-1}\)\(\cdot\)min\(^{-1}\)). \(PO\text{peak}\) and the MRT-corrected GET were 388 ± 33 W and 181 ± 28 W, respectively. The best individual fit for CP and \(W'\) was derived from the hyperbolic two-parameter power-time model in nine subjects and from the linear inverse-of-time model in three subjects. Mean model-predicted CP and \(W'\) were 250 ± 29 W and 20.6 ± 3.4 kJ, respectively. The MLSS (i.e., CP-corrected) occurred at a significantly lower PO (241 ± 31 W) than the model-predicted CP (\(P = 0.014\)). Consequently, the size of \(W'\)-corrected (23.9 ± 5.4 kJ) was higher than the initially determined \(W'\) (\(P = 0.015\)). For the purpose of clarification, from this point onwards CP and \(W'\) will refer to the corrected CP and \(W'\) unless stated otherwise. Table 1 provides an overview of individual CP and MLSS estimates.
**WB1** (Table 2). All subjects were able to complete WB1 (307 ± 29 W, 270 s) during the four trials and no differences were observed in the end values for $\dot{V}O_2$ (97% ± 4% $\dot{V}O_2\text{peak}$, $P = 0.836$), HR (90% ± 2% $HR_{\text{peak}}$, $P = 0.283$) or [La$^-$] (9.1 ± 1.0 mmol•L$^{-1}$, $P = 0.927$) among conditions. End-$\dot{V}O_2$ and end-HR were lower compared to $\dot{V}O_2\text{peak}$ ($P = 0.015$) and $HR_{\text{peak}}$ ($P < 0.001$).

**WB2** (Table 2). $W'_\text{ACT}$ was lowest in CP - 10 W (49% ± 24%, $P < 0.01$) and highest in 50% GET (93% ± 21%, $P < 0.01$), but did not differ between ∆GET-CP and GET ($P = 0.100$) (Fig. 2A). The average $W'_\text{ACT}$ ($P = 0.731$) and $W'_\text{ACT}$ in ∆GET-CP ($P = 0.384$) and GET ($P = 0.494$) did not differ from $W'_\text{PRED}$. However, $W'_\text{ACT}$ was overestimated in CP - 10 W (-34% ± 32%, $P = 0.004$) and underestimated in 50% GET (+24% ± 28%, $P = 0.013$). Similarly, mean $W'_\text{ACT-RATE}$ was overestimated in CP - 10 W (-51% ± 48%, $P = 0.005$), underestimated in 50% GET (+35% ± 42%, $P = 0.006$) and did not differ from the mean $W'_{\text{PRED-RATE}}$ in ∆GET-CP ($P = 0.486$) and GET ($P = 0.551$). Both $W'_\text{ACT-RATE}$ ($P < 0.05$) and $W'_{\text{PRED-RATE}}$ ($P < 0.001$) were highest in 50% GET followed by GET, ∆GET-CP and CP - 10 W (Fig. 2B). $\dot{V}O_2$ was higher at the end of WB2 in the 50% GET condition compared to CP - 10 W and ∆GET-CP ($P < 0.05$). $\dot{V}O_2$ did not reach $\dot{V}O_2\text{peak}$ during CP - 10 W ($P = 0.014$) and ∆GET-CP ($P = 0.028$). End-HR did not differ among conditions ($P > 0.05$) and did not reach $HR_{\text{peak}}$ ($P < 0.001$). [La$^-$] at exhaustion was lower in CP - 10 W ($P < 0.001$) and higher in 50% GET ($P < 0.05$) compared to the other conditions.

**REC** (Table 3). $\dot{V}O_2$ and HR at the end of REC was significantly different between all conditions ($P < 0.001$): CP - 10 W > ∆GET-CP > GET > 50% GET. EPOC was lowest in CP - 10 W ($P < 0.01$) and higher in GET compared to ∆GET-CP ($P = 0.01$) (Fig. 3 and 4A). The
amount of $[\text{La}^-]$ clearance was higher during $\Delta\text{GET-CP}$ and GET compared to CP - 10 W ($P < 0.05$) and higher in GET in comparison to 50% GET ($P = 0.011$) (Fig. 4B). The clearance rate of $[\text{La}^-]$ was slowest in CP - 10 W ($P < 0.05$) and was faster in GET compared to $\Delta\text{GET-CP}$ ($P = 0.022$).

**DISCUSSION**

The results of this study confirmed our hypothesis that the $\dot{W}'$ reconstitution is influenced by the intensity domain of the recovery bout. $W'_{\text{PRED}}$ underestimated the recovery of $\dot{W}'$ in the moderate intensity domain (50% GET) and overestimated recovery in the heavy intensity domain (CP - 10 W). These differences between the recovery conditions emphasize the importance of the demarcation of the intensity domains for both the expenditure and reconstitution of $\dot{W}'$.

It is beyond question that the recovery from severe intensity exercise, and thus the speed of $\dot{W}'$ reconstitution, depends on the PO of the recovery bout. Skiba et al. (2012) quantified the reconstitution of $\dot{W}'$ using a mono-exponential model with a time constant (i.e., indicative of the speed of $\dot{W}'$ reconstitution) that varies according to the absolute difference between the recovery PO and CP (i.e., $D_{\text{CP}}$) (8). This means that, provided that $D_{\text{CP}}$ is equal, the speed of $\dot{W}'$ reconstitution would be the same in individuals with a strongly differing CP, irrespective of the relative intensity (or the intensity domain) of the recovery bout. However, this study indicates that the $W'_{\text{BAL}}$ model overestimates $W'_{\text{ACT}}$ when the recovery intensity is situated at a high PO in the heavy intensity domain (i.e., CP - 10 W) and underestimates $\dot{W}'$ reconstitution at a low PO in the moderate intensity domain (i.e., 50% GET). To highlight this, three subjects with the lowest $D_{\text{CP}}$ (36 ± 2 W) in the GET recovery condition (i.e., moderate intensity) were matched with three
subjects with the highest $D_{CP}$ (44 ± 4 W) in the ∆GET-CP condition (i.e., heavy intensity). Since $D_{CP}$ was similar between groups, the imposed recovery duration (i.e., time to reach $W'_{PRED} = 75\%$) was similar as well and no differences in $W'_{ACT}$ should be expected. However, the subjects that recovered in the moderate intensity domain restored 76% ± 24% of $W'$ compared to 53% ± 2% for the subjects recovering in the heavy intensity domain. Although $D_{CP}$ is the primary factor driving the exponential recovery of $W'$, it seems that the intensity domain also has a substantial impact on the recovery speed. Future research should address whether the implementation of relative exercise intensities (i.e., percent CP instead of absolute $D_{CP}$) can improve the accuracy of $W'$ reconstitution modelling. Furthermore, exploring the possibility of using a domain-specific relationship between $D_{CP}$ and $\tau_{W}$ (i.e. for recovery in the heavy intensity domain) and $D_{GET}$ and $\tau_{W}$ (i.e. for recovery in the moderate intensity domain) could enhance the models’ predictive capabilities.

CP represents the highest sustainable oxidative metabolic rate and exercise below CP allows a certain oxidative metabolic reserve to restore the metabolic milieu (7). For this study, we physiologically validated the model-derived CP by MLSS testing and found that the CP was situated at a higher power output than the MLSS (∆9 ± 11 W) (23,24). This finding is consistent with previous research (25,34,35). It has to be pointed out that some debate exists which estimate represents the true boundary of the heavy-severe domain (23,24,29). However, in our opinion, both CP and MLSS can be regarded as the critical intensity of exercise with their determination being subject to a certain degree of measurement error. On the one hand, the determination of CP is influenced by the duration of the predictive trials and some additional measurement error might arise from its dependency on the exhaustive efforts of the participants (22). On the other
hand, the definition of MLSS implies the possibility of a small underestimation of the upper limit of the heavy intensity domain, although the small differences in PO between the trials (i.e., 10 W) enhances its precision (29). Given the interdependency of CP and $W'$ as parameters of the power-time relationship, it was necessary to adjust the model-predicted $W'$ in accordance with the “true CP” (see methods). However, this would have no direct impact on our conclusions since the objective was to compare the different recovery intensities rather than to justify the exact determination of the critical intensity of exercise and $W'$.

In the present study, the $W'_{\text{BAL}}$ model was used to calculate the theoretical recovery duration in order to reconstitute 50% of $W'$ (i.e., from 25 to 75%). Consequently, the imposed recovery duration increased with increasing intensity (i.e., from ~8 min at 50% GET to ~15 min at CP - 10 W). In an attempt to isolate the effect of the different recovery intensities, $W'_{\text{ACT}}$ was divided by the recovery duration and expressed as a mean actual recovery rate (i.e., $W'_{\text{ACT-RATE}}$). In contrast to the expected linear increase of the $W'$ recovery rate (i.e., $W'_{\text{PRED-RATE}}$) with decreasing recovery intensities, it was shown that $W'_{\text{ACT-RATE}}$ was slower at high intensities and increased in a nonlinear manner as the PO decreased from the heavy into the moderate intensity domain (Fig. 2B). The nonlinear relationship between $W'_{\text{ACT-RATE}}$ and $D_{CP}$ shows that a reduction of the PO in the heavy intensity domain has a larger impact on the reconstitution of $W'$ compared to the same reduction in the moderate domain, supporting the idea of a domain specific relationship. It should be kept in mind that when different recovery durations are applied both the $W'_{\text{PRED-RATE}}$ (given the exponential nature of the $W'_{\text{BAL}}$ model) and the $W'_{\text{ACT-RATE}}$ would change. Moreover, the duration of a recovery bout in the heavy intensity domain might at some point start to counteract further $W'$ reconstitution, thereby slowly inducing complete exhaustion (36). It has
already been shown that immediately after exhaustive exercise, subjects were able to complete a 20-min target WB in the moderate intensity domain (i.e., 80% GET) but could only sustain exercise in the heavy intensity domain (i.e., 90% CP) for another 13 min, which indicates that no further reconstitution of \( W' \) occurred (9).

Although the importance of the exercise intensity domain for the recovery of fatigue related metabolites has previously been demonstrated (10,36), the exact physiological mechanisms for the differences in \( W' \) reconstitution after a short and low intensity recovery vs. a longer and harder recovery has yet to be clarified. Chidnock et al. (2013) demonstrated that muscle phosphocreatine (PCr) concentration following exhaustive exercise recovered to 96 and 76% of its baseline values after a 10 min passive recovery and a 10 min below CP recovery, respectively. The pH values also increased in both conditions from 6.7 to 7.0. When PO during recovery remained >CP, exercise could only be sustained for 39 s and no recovery of [PCr] nor pH occurred (10). The shorter TTE after recovery in the heavy intensity domain in this study might be explained by a reduced possibility to restore the intramuscular milieu.

For a higher recovery PO, a greater amount of oxygen is required in terms of acute energy supply which ultimately results in lower oxygen availability to restore the intramuscular metabolic milieu (37). In an attempt to quantify the amount of oxygen available to restore the homeostasis after WB1, the difference between the measured \( \dot{V}O_2 \) and the predicted \( \dot{V}O_2 \) for a given recovery PO was calculated and quantified as EPOC. The size of EPOC during CP - 10 W was found to be significantly smaller than during the other REC conditions. In concert with the size of EPOC, the net amount of [La\(^-\)] that was removed from the bloodstream was lower during REC CP - 10 W
compared to ΔGET-CP and GET but did not differ from 50% GET. These differences can be linked to the increased production of lactate during the recovery bouts above GET compared to the recovery below GET but might also indicate that the amount of oxygen available to restore the oxygen stores of the tissues, to remove [La\(^{-}\)] and to neutralize pH was insufficient to recover from WB1 (19,38). It appears that the larger oxidative reserve in the moderate intensity domain allows a better restoration of the intramuscular metabolic milieu compared to recovery in the heavy intensity domain which in turn increases the range to reach critical limits in the depletion/accumulation of intramuscular substrates/metabolites during subsequent severe intensity exercise. However, in this context, it should be noted that the size of EPOC did not differ between the recovery conditions in the moderate intensity domain (i.e., GET and 50% GET), despite a higher $W'_\text{ACT}$ in 50% GET compared to GET. This can indicate that beyond a certain threshold of oxidative reserve, other factors have a more pronounced impact on the recovery kinetics. We believe that this is the first study that investigates the potential link between EPOC and $W'$ recovery.

Current technologies allow us to track the energetic reserve capacity of the body during exercise by means of a $W'$ recovery model. However, the accuracy of the $W'_\text{BAL}$ model for recovery intensities situated in the heavy intensity domain is lacking experimental evidence. Overall, this study showed that the average $W'_\text{ACT}$ did not differ from $W'_\text{PRED}$ but this is not representative for the accuracy of the $W'_\text{BAL}$ model across the intensity domains. $W'_\text{ACT}$ was underestimated after recovery in the moderate intensity domain (50% GET) and overestimated after recovery in the heavy intensity domain (CP - 10 W). This means that the current applicability of the model is questionable for, for example, a competitive cyclists in the final of a road race or during a team
pursuit where the PO rarely drops below GET (39). To increase the accuracy and as such the practical applicability of future $W'$ reconstitution models, research should explore the possibilities of an intensity specific relationship between the recovery PO (i.e. $D_{CP}$ and $D_{GET}$, respectively) and $\tau_{W'}$ and as such incorporate the boundary between the moderate and the heavy intensity domain (i.e., GET) in the model.
ACKNOWLEDGMENTS

The authors thank the subjects for their commitment to the study. The authors received no financial support for the research, authorship, and/or publication of this article.

CONFLICT OF INTEREST

Results of the present study do not constitute endorsement by the American College of Sports Medicine and are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. No conflicts of interest, financial or otherwise, are declared by the authors.
REFERENCES


24. Keir DA, Maturana FM, Murias JM. Reply to "Discussion of 'Can measures of critical power precisely estimate the maximal metabolic steady-state?' - Is it still necessary to compare critical power to maximal lactate steady state?" - When is it appropriate to compare critical power to maximal lactate steady-state?. Appl Physiol Nutr Metab. 2018;43(1):96–7.


FIGURES

Figure 1: Design of the experimental trials in which the recovery durations were adjusted to the recovery intensity in order to match the predicted $W'$ reconstitution across conditions. To calculate the actual $W'$ reconstitution (i.e., $W'_{ACT}$), TTE of WB2 was divided by 360 s (i.e., the predicted time to exhaustion in rested conditions) and expressed relatively (%). Capillary blood samples were taken immediately after WB1, recovery and WB2.

Figure 2A: $W'_{ACT}$ compared to $W'_{PRED}$ as a function of $D_{CP}$ across intensity domains and figure 2B: $W'_{ACT}$-RATE compared to $W'_{PRED}$-RATE as a function of $D_{CP}$ across intensity domains.

$W'_{ACT}$ (black dots), $W'_{PRED}$ (white dots).

*Different from predicted ($P < 0.05$)

†Different from each other ($P < 0.05$)

Figure 3: Temporal course of $\dot{V}O_2$ across WB1, REC (A= CP - 10 W, B= ΔGET-CP, C= GET and D= 50% GET) and WB2 to exhaustion.

Since the recovery times between subjects differed for B, C and D, data were time-aligned to the subject with the shortest recovery bout. The first part of the curve (//) shows the $\dot{V}O_2$ for WB1 and the REC, which was cut-off 60 s before the shortest end-recovery time. The second part of the curve illustrates the final 60 s of recovery and the onset of WB2, which was cut-off at the shortest TTE, and also the average maximum TTE and $\dot{V}O_2$ is shown.

Figure 4A: Excess post-exercise oxygen consumption (L) during REC following WB1 and figure 4B: Lactate clearance (mmol•L$^{-1}$) during REC following WB1.

*Different from each other ($P < 0.05$)
Figure 1
Figure 2

A

$W^* (%)$

Heavy

Moderate

$D_{CP} (W)$

B

$W_{\text{RATE}} (J/s^{-1})$

$D_{CP} (W)$
Figure 4
Table 1
Model-predicted CP and W' estimates for each subject according to the best individual fit model, MLSS verification (CP-corrected) and W'-corrected.

<table>
<thead>
<tr>
<th>Subject</th>
<th>CP (W)</th>
<th>CV (%)</th>
<th>W' (kJ)</th>
<th>CV (%)</th>
<th>Model</th>
<th>MLSS (W)</th>
<th>W’-corrected (kJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>209</td>
<td>1.1</td>
<td>17.8</td>
<td>8.4</td>
<td>Hyp-2</td>
<td>199</td>
<td>21.2</td>
</tr>
<tr>
<td>2</td>
<td>252</td>
<td>0.9</td>
<td>18.7</td>
<td>9.4</td>
<td>Hyp-2</td>
<td>252</td>
<td>18.7</td>
</tr>
<tr>
<td>3</td>
<td>250</td>
<td>1.0</td>
<td>21.4</td>
<td>9.8</td>
<td>Hyp-2</td>
<td>240</td>
<td>25.2</td>
</tr>
<tr>
<td>4</td>
<td>206</td>
<td>1.2</td>
<td>27.0</td>
<td>8.7</td>
<td>Hyp-2</td>
<td>196</td>
<td>30.6</td>
</tr>
<tr>
<td>5</td>
<td>264</td>
<td>0.7</td>
<td>20.0</td>
<td>6.9</td>
<td>Hyp-2</td>
<td>284</td>
<td>12.6</td>
</tr>
<tr>
<td>6</td>
<td>243</td>
<td>1.1</td>
<td>26.3</td>
<td>7.3</td>
<td>Hyp-2</td>
<td>233</td>
<td>29.9</td>
</tr>
<tr>
<td>7</td>
<td>252</td>
<td>1.9</td>
<td>18.9</td>
<td>5.4</td>
<td>Lin-P</td>
<td>242</td>
<td>22.3</td>
</tr>
<tr>
<td>8</td>
<td>231</td>
<td>0.3</td>
<td>17.4</td>
<td>4.5</td>
<td>Hyp-2</td>
<td>211</td>
<td>24.5</td>
</tr>
<tr>
<td>9</td>
<td>287</td>
<td>1.8</td>
<td>15.9</td>
<td>9.3</td>
<td>Lin-P</td>
<td>277</td>
<td>19.4</td>
</tr>
<tr>
<td>10</td>
<td>311</td>
<td>1.9</td>
<td>22.3</td>
<td>9.8</td>
<td>Lin-P</td>
<td>291</td>
<td>29.5</td>
</tr>
<tr>
<td>11</td>
<td>250</td>
<td>0.4</td>
<td>20.2</td>
<td>5.4</td>
<td>Hyp-2</td>
<td>240</td>
<td>23.8</td>
</tr>
<tr>
<td>12</td>
<td>244</td>
<td>0.2</td>
<td>21.8</td>
<td>1.9</td>
<td>Hyp-2</td>
<td>224</td>
<td>29.2</td>
</tr>
<tr>
<td>Mean</td>
<td>250 ± 29</td>
<td>1.0 ± 0.6</td>
<td>20.6 ± 3.4</td>
<td>7.2 ± 2.5</td>
<td>241 ± 31*</td>
<td>23.9 ± 5.4*</td>
<td></td>
</tr>
</tbody>
</table>

Hyp-2= hyperbolic 2-parameter power–time model; Lin-P= linear inverse-of-time model

*Different from model-predicted CP and W’ (P < 0.05)
Table 2
Characteristics (PO, TTE) and physiological responses (\(\dot{V}O_2\), HR, [La\(^-\)]) of WB1 (average four recovery conditions) and WB2 (all-out) for the four experimental conditions.

<table>
<thead>
<tr>
<th></th>
<th>WB1</th>
<th>WB2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CP - 10 W</td>
<td>ΔGET-CP</td>
</tr>
<tr>
<td>PO (W)</td>
<td>307 ± 29</td>
<td>307 ± 29</td>
</tr>
<tr>
<td>TTE (s)</td>
<td>270 ± 0</td>
<td>177 ± 87*‡</td>
</tr>
<tr>
<td>W(^*)_ACT (%)</td>
<td>49 ± 24*‡$</td>
<td>69 ± 24†#</td>
</tr>
<tr>
<td>W(^*)_ACT-RATE (J(\cdot)min(^{-1}))</td>
<td>6.9 ± 7.1*‡$</td>
<td>13.5 ± 8.2*‡#</td>
</tr>
<tr>
<td>W(^*)_PRED-RATE (J(\cdot)min(^{-1}))</td>
<td>13.4 ± 3.1*‡#</td>
<td>15.0 ± 3.2*‡#</td>
</tr>
<tr>
<td>End-(\dot{V}O_2) (mL(\cdot)min(^{-1}))</td>
<td>3753 ± 266</td>
<td>3644 ± 290#</td>
</tr>
<tr>
<td>End-(\dot{V}O_2) (% (\dot{V}O_2)_peak)</td>
<td>97 ± 4</td>
<td>94 ± 7#</td>
</tr>
<tr>
<td>End-HR (% HR_peak)</td>
<td>90 ± 2</td>
<td>96 ± 2*</td>
</tr>
<tr>
<td>[La(^-)] (mmol(\cdot)L(^{-1}))</td>
<td>9.1 ± 1</td>
<td>10.2 ± 2.2*‡#</td>
</tr>
</tbody>
</table>

TTE: time to exhaustion; W\(^*\)\_ACT: actual \(W^*\) reconstitution; W\(^*\)\_ACT-RATE: actual \(W^*\) reconstitution per second recovery; W\(^*\)\_PRED-RATE: predicted \(W^*\) reconstitution per second recovery; End-: end exercise
*Different from WB1 / predicted (\(P < 0.05\))
†Different from CP - 10 W (\(P < 0.05\))
‡Different from ΔGET-CP (\(P < 0.05\))
$Different from GET (\(P < 0.05\))
#Different from 50% GET (\(P < 0.05\))
### Table 3
Characteristics (PO, T) and physiological response (\(\dot{V}O_2\), HR, [La\(^{-}\)]) of REC separating WB1 and WB2.

<table>
<thead>
<tr>
<th></th>
<th>CP - 10 W</th>
<th>∆GET-CP</th>
<th>GET</th>
<th>50% GET</th>
</tr>
</thead>
<tbody>
<tr>
<td>(D_{CP}) (W)</td>
<td>10 ± 0(^{+#})</td>
<td>30 ± 6(^{+#})</td>
<td>60 ± 12(^{+#})</td>
<td>150 ± 19(^{+#})</td>
</tr>
<tr>
<td>Time (s)</td>
<td>890 ± 0(^{+#})</td>
<td>795 ± 26(^{+#})</td>
<td>680 ± 39(^{+#})</td>
<td>483 ± 26(^{+#})</td>
</tr>
<tr>
<td>End-(\dot{V}O_2) (% (\dot{V}O_2)peak)</td>
<td>83 ± 6(^{+#})</td>
<td>77 ± 5(^{+#})</td>
<td>70 ± 7(^{+#})</td>
<td>45 ± 5(^{+#})</td>
</tr>
<tr>
<td>EPOC (L)</td>
<td>1.63 ± 1.19(^{+#})</td>
<td>2.57 ± 1.14(^{+#})</td>
<td>3.75 ± 2.01(^{+#})</td>
<td>3.26 ± 1.23(^{+#})</td>
</tr>
<tr>
<td>End-HR (% HR(_{peak}))</td>
<td>87 ± 4(^{+#})</td>
<td>83 ± 4(^{+#})</td>
<td>75 ± 5(^{+#})</td>
<td>64 ± 3(^{+#})</td>
</tr>
<tr>
<td>([La^{-}]) clearance (mmol•L(^{-1}))</td>
<td>1.9 ± 1.6(^{+#})</td>
<td>2.9 ± 1.5(^{+#})</td>
<td>3.6 ± 1.8(^{+#})</td>
<td>2.4 ± 1.2(^{+#})</td>
</tr>
<tr>
<td>([La^{-}]) clearance rate (mmol•L(^{-1})•min(^{-1}))</td>
<td>0.13 ± 0.11(^{+#})</td>
<td>0.22 ± 0.11(^{+#})</td>
<td>0.32 ± 0.16(^{+#})</td>
<td>0.30 ± 0.15(^{+#})</td>
</tr>
</tbody>
</table>

EPOC: Excess post-exercise oxygen consumption; \([La^{-}]\) clearance: net lactate clearance during recovery (= [La\(^{-}\) end-WB1 - [La\(^{-}\) end-REC]); \([La^{-}]\) clearance rate: net lactate clearance per minute during recovery

\(^{*}\)Different from CP - 10 W \((P < 0.05)\)
\(^{†}\)Different from ∆GET-CP \((P < 0.05)\)
\(^{‡}\)Different from GET \((P < 0.05)\)
\(^{§}\)Different from 50% GET \((P < 0.05)\)
\(^{#}\)Different from \(W_{PRED-RATE}\) \((P < 0.05)\)