The Reconstitution of $W'$ Depends on Both Work and Recovery Characteristics

KEVIN CAEN$^{1,2}$, JAN G. BOURGOIS$^{1,2}$, GIL BOURGOIS$^1$, THIBAUX VAN DER STEDE$^1$, KOBE VERMEIRE$^1$, and JAN BOONE$^{1,2}$

$^1$Department of Movement and Sport Sciences, Ghent University, Ghent, BELGIUM; and $^2$Center of Sports Medicine, Ghent University Hospital, Ghent, BELGIUM

ABSTRACT

CAEN, K., J. G. BOURGOIS, G. BOURGOIS, T. VAN DER STEDE, K. VERMEIRE, and J. BOONE. The Reconstitution of $W'$ Depends on Both Work and Recovery Characteristics. Med. Sci. Sports Exerc., Vol. 51, No. 8, pp. 1745–1751, 2019. Purpose: This study aimed to investigate the effects of different work and recovery characteristics on the $W'$ reconstitution and to test the predictive capabilities of the $W'$ $^*$ $^*$ $^*$ model. Methods: Eleven male participants ($22\pm3$ yr, $55\pm4$ mL kg$^{-1}$ min$^{-1}$) completed three to five constant work rate tests to determine CP and $W'$. Subsequently, subjects performed 12 experimental trials, each comprising two exhaustive constant work rate bouts (i.e., WB1 and WB2), interspersed by an active recovery interval. In each trial, work bout characteristics (P4 or P8, i.e., the work rate predicted to result in exhaustion in 4 and 8 min, respectively), recovery work rate (33% CP or 66% CP), and recovery duration (2, 4, or 6 min) were varied. Actual ($W'_{ACT}$) and model-predicted ($W'_{PRED}$) reconstitution values of $W'$ were calculated. Results: After 2, 4, and 6 min recovery, $W'_{ACT}$ averaged $46\%\pm2.7\%$, $51.2\%\pm3.3\%$, and $59.4\%\pm4.1\%$, respectively ($P=0.003$). $W'_{ACT}$ was 9.4% higher after recovery at 33% CP than at 66% CP ($56.9\%\pm3.9\%$ vs $47.5\%\pm3.2\%$) ($P=0.019$). $W'_{ACT}$ yielded a 11.3% higher $W'_{ACT}$ than P8 exercise ($57.8\%\pm3.9\%$ vs $46.5\%\pm2.7\%$) ($P=0.001$). $W'_{ACT}$ was higher than $W'_{PRED}$ in the conditions P4-2 min ($+29.7\%$), P4-4 min ($+18.4\%$), and P8-2 min ($+18\%$) ($P<0.01$). A strong correlation ($R=0.68$) between the rate of $W'$ depletion and $W'$ recovery was found ($P=0.001$). Conclusion: This study demonstrated that both the work and recovery characteristics of a prior exhaustive exercise bout can affect the $W'$ reconstitution. Results revealed a slower $W'$ reconstitution when the rate of $W'$ depletion was slower as well. Furthermore, it was shown that the current $W'_{BAL}$ model underestimates actual $W'$ reconstitution, especially after shorter recovery. Key Words: CRITICAL POWER, $W'$, RECONSTITUTION, RECOVERY, MODELING

The critical power (CP) concept provides a mathematical and physiological framework for exploring and understanding performance capacity and fatigue mechanisms in athletes and healthy individuals, but also in patient populations (1). Its parameters, CP (i.e., the lower boundary of the severe-intensity domain) and $W'$ (i.e., the amount of work available above CP), are traditionally used for the assessment of physical fitness, estimation of athletic performance, and evaluation of training and intervention programs (2). Although originally the CP model was only applied to continuous exercise, more recent developments extended its application to intermittent exercise as well (3–6). This translation of the model seems highly relevant because many sports are characterized by frequent changes between high-intensity efforts (i.e., >CP) and low-intensity recovery periods (i.e., <CP). These typical variations in exercise intensity cause alternate consumption and replenishment of $W'$, respectively.

Morton and Billat (3) were the first to introduce an adapted CP model for intermittent exercise. Their model assumed linear kinetics for both $W'$ utilization and reconstitution. However, Ferguson et al. (7) found that the refilling of $W'$ occurred in a curvilinear way. On the basis of these observations, Skiba et al. (5) developed a new model for tracking the dynamic balance of $W'$ ($W'_{BAL}$) at any given time during intermittent exercise:

$$W'_{bal} = W' - \int_0^t \left( W'_{exp} \left( e^{-\left(t-u\right)/\tau_{W'}} \right) \right) du$$

$W'_{BAL}$ is calculated by subtracting an athlete’s known $W'$ by the amount of $W'$ that has already been expended ($W'_{EXP}$) during efforts above CP. According to the model, a monoeponential recharge of $W'_{EXP}$ becomes possible when the athlete drops below CP $t-u$. The speed of this recharge is influenced by the recovery power, which is incorporated in the calculation of the recovery time constant ($\tau_{W'}$) as the difference between CP and recovery power output ($D_{CP}$):

$$\tau_{W'} = 546 e^{(-0.01D_{CP})} + 316$$

The $W'_{BAL}$ model provides a new way of calculating residual performance capacity in athletes and therefore, it carries a lot of potential for use within the field. Real-time monitoring of athletic performance during both training and racing may
be a promising application of the model (5), especially with the advent of portable GPS devices and power meters in cycling. Accurate knowledge of $W'_{BAL}$ could support athletes in optimizing pacing strategies or making important decisions during races. By extension, it could assist coaches in developing more individualized training protocols.

Although the $W'_{BAL}$ model has been validated for cycling, using field data in a group of well-trained triathletes (8), it has recently been reported that the model-predicted $W'$ recovery ($W'_{PRED}$) can underestimate actual $W'$ recovery ($W'_{ACT}$) (9). One possible explanation for this underestimation is that in the current $W'_{BAL}$ model, recovery kinetics are only determined by the recovery modalities during exercise, whereas the characteristics of the preceding exercise (i.e., work bout intensity and duration) are not accounted for. Thus, the $W'_{BAL}$ model implies that $W'$ recovery kinetics remain unchanged, regardless of the intensity and duration of previous exercise efforts. However, as recognized by Morton and Billat (3), exercise tolerance during intermittent work is determined by four independent parameters: work intensity, work duration, recovery intensity, and recovery duration. Chorley et al. (10) recently demonstrated that, without manipulating work or recovery characteristics, repeated maximal exercise bouts slowed down the $W'$ reconstitution kinetics. Yet the question remains whether the rate of $W'$ depletion, which can be manipulated through changes in the intensity and/or duration of the severe-intensity work bout, could affect the rate of subsequent $W'$ replenishment.

Therefore, the goals of the present study were twofold. First, we aimed to investigate the effects of different work and recovery characteristics on the reconstitution of $W'$. We hypothesized that, besides the duration and the work rate of the recovery, the characteristics of a prior exhaustive constant work rate bout would also affect the rate of the $W'$ reconstitution. In accordance, we expected to find a relationship between the rate of $W'$ depletion and the rate of $W'$ reconstitution, showing faster $W'$ kinetics as the rate of $W'$ depletion is higher. In addition, to investigate the underpinning mechanisms of $W'$, we also explored the response of several physiological parameters (i.e., HR, VO$_2$, lactate concentration ([La]$_t$), pH level, and bicarbonate concentration ([HCO$_3$]))]. Second, we examined whether $W'_{ACT}$ differed from $W'_{PRED}$ as calculated by means of the $W'_{BAL}$ model. We hypothesized that the $W'_{BAL}$ model would underestimate $W'_{ACT}$.

**METHODS**

**Subjects**

Eleven male PE students (21.7 ± 2.7 yr, 1.78 ± 0.06 m, 74.5 ± 7.3 kg) were recruited to voluntarily participate in the study. All subjects were physically active and participated in a variety of recreational sports on a regular basis. None of them had a history of structured cycling training. Participants were familiar with laboratory exercise procedures and habituated to maximal exercise efforts. On the basis of a medical examination that took place before the onset of the study, each subject was declared to be in good health. Written informed consent was received, and the study was approved by the ethical committee of the Ghent University Hospital (Ghent, Belgium).

**Experimental Protocol**

All tests were conducted in the laboratory (Sport Science Laboratory–Jacques Rogge, Ghent University) on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Room air temperature was set at 19°C with a relative humidity of 50%. Throughout the study, participants were asked to avoid strenuous exercise in the last 24 h before each laboratory visit, and single tests were always separated by at least 48 h recovery. All tests were completed within a period of 7 wk.

**Ramp incremental exercise test.** Before the experimental trials, subjects performed a maximal ramp incremental exercise test to assess the individual cardiorespiratory response. After a 3-min warm-up at 50 W, work rate increased continuously with 30 W·min$^{-1}$. Subjects cycled with a self-selected cadence between 70 and 90 rpm and were instructed to repeat this cadence during all subsequent test sessions. The test was terminated at voluntary exhaustion, which was defined as the inability to maintain the preferred cadence for more than five consecutive seconds.

**CP tests.** To determine CP and $W'$, three to five exhaustive constant work rate tests were completed in a randomized order. Each test started with a 3-min warm-up at 50 W, immediately followed by an abrupt increase to the appropriate work rate (70%, 75%, 85%, 90%, and 100% $P_{peak}$). Work rates were chosen to provoke exhaustion between 2 and 15 min (2). Subjects cycled at their previously chosen cadence, and each test was terminated at voluntary exhaustion.

**Experimental trials.** All participants performed a total of 12 experimental trials in a randomized order. After a 3-min warm-up at 50 W, each trial consisted of two identical constant work rate bouts to exhaustion (i.e., WB1 and WB2), interspersed by an active recovery interval (Fig. 1). In each trial, work bout characteristics (P4 or P8, i.e., the work rate predicted to result in exhaustion in 4 and 8 min, respectively), recovery work rate (CP$_{33}$ or CP$_{66}$, i.e., 33% or 66% CP), and recovery duration (2, 4, or 6 min) were combined, resulting in a total of 12 conditions (2 × 2 × 3). In accordance with all previous test sessions, subjects were asked to maintain their self-selected cadence and cycled until volitional fatigue.

**Measurements**

During all test sessions, HR was monitored continuously (H7 sensor; Polar, Kempele, Finland). During the experimental trials, pulmonary gas exchange was measured breath by breath using an automatic metabolic instrument (Jaeger Oxycon Pro; Viasys Healthcare GmbH, Höchberg, Germany), time to exhaustion (TTE) for WB1 and WB2 was manually recorded, and capillary blood samples (65 μL) from the fingertip were taken immediately after each work bout (Fig. 1). These blood
samples were analyzed (Radiometer ABL90 FLEX; Radium- 
er Medical ApS, Bronshøj, Denmark) to measure [La−], pH, and
[HCO3−].

Data Analysis

Ramp incremental exercise test. Ppeak and HRpeak were defined as the highest values obtained during the test, and VO2peak was defined as the highest 30-s average achieved throughout the protocol.

CP tests. CP and W′ were determined using three mathematical models: the hyperbolic power–time (P–Tlim) model, the linear work–time (W–Tlim) model, and the linear inverse-of-time (1/Tlim) model. A “best individual fit” approach was used, meaning that for each participant separately, the model with the smallest total error (i.e., sum of the CV% associated with CP and W′) was chosen (11,12).

Experimental trials. For each trial, the TTE of WB2 was expressed relative to the TTE of WB1. Because it can be theoretically assumed that W′ = 0 at exhaustion, this calculation yielded the actual W′ reconstitution (W′ACT) after the recovery interval. For comparison, the model-predicted W′ reconstitution (W′PRED) was calculated using equations 1 and 2 (5) and was expressed relative to the total amount of work performed in WB1. Given that in theory W′ is fully depleted during WB1, the TTE of WB1 was used as a measure to express the rate of W′ depletion (i.e., shorter TTE indicates a faster rate of W′ depletion and vice versa). To quantify the rate of the W′ reconstitution, actual time constants (τW′) of the W′ recovery were calculated. For each trial, the model-predicted τW′ was varied by an iterative process until it matched W′ACT. Subsequently, mean τW′ values for P4 and P8 conditions were calculated across all recovery conditions, yielding two data points per subject.

Statistical Analysis

Data are presented as mean ± SD for n = 11 participants. Normal distribution of all variables was demonstrated by the Shapiro–Wilk test. One sample t-tests were conducted to compare the TTE of WB1 with the theoretical estimation (i.e., 240 s for P4 trials and 483 ± 105 s for P8 trials). Paired sample t-tests were performed to compare VO2 between the ramp incremental exercise test and the experimental trials and to check for possible training effects. A three-way (work bout characteristics [P4 vs P8] × recovery work rate [CP33 vs CP66] × recovery duration [2 vs 4 vs 6 min]) repeated-measures ANOVA with pairwise comparison was chosen to identify differences in W′ reconstitution. An additional within-subjects factor (W′ recovery [actual vs predicted]) was added to the analysis to compare W′ACT and W′PRED. The Pearson correlation coefficient (R) was selected to investigate the relationship between the rate of W′ depletion and the rate of W′ reconstitution. To identify differences in the physiological parameters (i.e., HR, VO2, [La−], pH, [HCO3−]) between P4 and P8 exercise and between WB1 and WB2, a two-way (work bout [1 vs 2] × work bout characteristics [P4 vs P8]) repeated-measures ANOVA with post hoc comparisons was conducted. All statistical analyses were performed with SPSS Statistics 24 (IBM Corp., Armonk, NY). Statistical significance was accepted at P < 0.05.

RESULTS

During the ramp incremental exercise test, Ppeak and VO2peak averaged 382 ± 28 W and 4071 ± 463 mL·min−1 (54.7 ± 3.6 mL·kg−1·min−1), respectively. Mean HRpeak was 187 ± 8 bpm. For the calculation of CP and W′, the best individual fit was derived from the P–Tlim model in 10 subjects and from the W–Tlim model in one subject. Mean CP was 248 ± 30 W, and mean W′ was 18.2 ± 5.2 kJ. Individual model estimates and their corresponding CV% are presented in Table 1.

On average, the TTE values of WB1 were 239 ± 48 s for P4 trials and 483 ± 105 s for P8 trials (Table 2). These values did not differ from the theoretically predicted the TTE of 240 s (P = 0.967) and 480 s (P = 0.927), respectively. The mean VO2 at the end of WB1 was 3938 ± 365 mL·min−1. This value was not different from the VO2peak attained during the ramp incremental exercise test (P = 0.086). Analysis of the experimental tests in a chronological order revealed no training effects for the TTE of WB1 or for VO2 at the end of WB1 (P > 0.05).

Effects of work and recovery characteristics. No interaction effect was found between work bout characteristics,
recovery work rate, and recovery duration (P > 0.05), but all three parameters showed a significant main effect on W'\text{ACT}.

W'\text{ACT} differed significantly between trials with 2 min recovery (46% ± 2.7%), 4 min recovery (51.2% ± 3.3%), and 6 min recovery (59.4% ± 4.1%) (P < 0.003, η\text{p}^2 = 0.727). Furthermore, W'\text{ACT} was 9.4% higher after recovery at CP33 than at CP66 (56.9% ± 3.9% vs 47.5% ± 3.2%) (P = 0.019, η\text{p}^2 = 0.44). Finally, exhaustive P4 exercise provoked a 11.3% higher W'\text{ACT} than P8 exercise (57.8% ± 3.9% vs 46.5% ± 2.7%) (P = 0.001, η\text{p}^2 = 0.664). A strong association (R = 0.68) was found between the rate of W' depletion and the rate of W' reconstitution (P = 0.001) (Fig. 2).

Physiological parameters (i.e., HR, VO₂, \([\text{La}^-]\), pH, and [HCO₃⁻]) at the end of WB1 were not different between P4 and P8 conditions, except for HR (P = 0.025). On the contrary, several differences were detected in WB2. After P4 exercise, \(\text{VO}_₂\) and \([\text{La}^-]\) showed higher values compared with P8 exercise (P = 0.033 and P = 0.003), whereas lower values were found for pH and [HCO₃⁻] (P < 0.001 and P = 0.007). Differences in physiological parameters were also detected between WB1 and WB2. After WB2 in P4 trials, HR and \([\text{La}^-]\) were higher (P = 0.009 and P < 0.001), and pH and [HCO₃⁻] were lower (P = 0.013 and P < 0.001) compared with WB1. In P8 trials, only a difference in \(\text{VO}_₂\) was found between both work bouts (P < 0.001). A detailed overview of TTE and the values for HR, \(\text{VO}_₂\), \([\text{La}^-]\), pH, and [HCO₃⁻] at the end of WB1 and WB2 for both P4 and P8 trials is provided in Table 2.

**Actual versus predicted reconstitution of W'**. Comparison of W'\text{ACT} and W'\text{PRED} yielded significant differences (P = 0.001, η\text{p}^2 = 0.693) that were dependent on work bout characteristics (P = 0.001) and recovery duration (P < 0.001). However, these differences were not affected by the recovery work rate (P = 0.621), and therefore, further results make no distinction between CP33 and CP66 conditions.

In the P4 trials, W'\text{ACT} was 29.7% higher than W'\text{PRED} after 2 min recovery (51.8% ± 2.8% vs 22.2% ± 0.3%; P < 0.001) and 18.4% higher after 4 min recovery (57.7% ± 4.3% vs 39.3% ± 0.5%; P = 0.001), but no significant differences were observed after 6 min recovery (64% ± 5.8% vs 52.7% ± 0.6%; P = 0.07). In the P8 trials, W'\text{PRED} underestimated W'\text{ACT} by 18% after 2 min recovery (40.1% ± 3.9% vs 22.2% ± 0.3%; P = 0.001). On the contrary, there were no differences between W'\text{ACT} and W'\text{PRED} after 4 min (44.8% ± 3% vs 39.3% ± 0.5%; P = 0.092) or 6 min (54.8% ± 3.8% vs 52.7% ± 0.6%; P = 0.576) recovery. Figure 3 presents an overview of the differences between W'\text{ACT} and W'\text{PRED} for all conditions and for the combined data of CP33 and CP66 trials.

**DISCUSSION**

The present study aimed to investigate the effects of different work and recovery characteristics on the W' reconstitution and to test the predictive capabilities of the W'\text{BAL} model. It was demonstrated that, besides the recovery modalities, the characteristics of a prior exercise work bout can also affect the W' reconstitution. More specifically, exhaustive P8 exercise led to a slower reconstitution of W' in comparison with P4 exercise, and this discrepancy was irrespective of the intensity and the duration of the recovery interval. The slower W' reconstitution in the P8 conditions was accompanied by overall lower \([\text{La}^-]\) and higher pH and [HCO₃⁻] in WB2. In support of these findings, a strong correlation (R = 0.68) between the rate of W' depletion and the rate of W' reconstitution was found. Furthermore, comparison of the model-predicted W' reconstitution (W'\text{PRED}) and the actual W' reconstitution (W'\text{ACT}) revealed that the current W'\text{BAL} model underestimates W'\text{ACT} especially during shorter recovery.

As could be expected from the W'\text{BAL} model of Skiba et al. (5), increasing the recovery duration (i.e., 2 vs 4 vs 6 min) and lowering the recovery work rate (i.e., CP66 vs CP33) resulted in a higher W'\text{ACT} in the present study. These findings are in

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**TABLE 1.** CP and W' estimates for each subject with the best individual fit model.

<table>
<thead>
<tr>
<th></th>
<th>CP (W)</th>
<th>SEE (%)</th>
<th>W' (kJ)</th>
<th>SEE (%)</th>
<th>Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>265</td>
<td>0.7</td>
<td>20.2</td>
<td>5.0</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>2</td>
<td>268</td>
<td>0.2</td>
<td>16.0</td>
<td>2.9</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>3</td>
<td>267</td>
<td>0.8</td>
<td>16.4</td>
<td>5.8</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>4</td>
<td>263</td>
<td>0.5</td>
<td>15.0</td>
<td>5.0</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>5</td>
<td>202</td>
<td>2.8</td>
<td>21.9</td>
<td>7.8</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>6</td>
<td>229</td>
<td>0.9</td>
<td>17.8</td>
<td>5.1</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>7</td>
<td>290</td>
<td>1.9</td>
<td>19.6</td>
<td>9.7</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>8</td>
<td>203</td>
<td>2.4</td>
<td>29.0</td>
<td>7.7</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>9</td>
<td>261</td>
<td>1.5</td>
<td>23.2</td>
<td>6.7</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>10</td>
<td>268</td>
<td>0.5</td>
<td>9.6</td>
<td>7.6</td>
<td>(\mu\text{T}_{182})</td>
</tr>
<tr>
<td>11</td>
<td>214</td>
<td>1.1</td>
<td>17.1</td>
<td>6.2</td>
<td>(\mu\text{T}_{182})</td>
</tr>
</tbody>
</table>

\(\mu\text{T}_{182}\), hyperbolic power–time model; \(\mu\text{T}_{182}\), linear power–time model.

**TABLE 2.** Mean TTE and HR, \(\text{VO}_₂\), \([\text{La}^-]\), pH, and [HCO₃⁻] at the end of each work bout in P4 and P8 trials.

<table>
<thead>
<tr>
<th>Work bout 1</th>
<th>P4</th>
<th>P8</th>
<th>Work bout 2</th>
<th>P4</th>
<th>P8</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTE (s)</td>
<td>239 ± 48</td>
<td>483 ± 105*</td>
<td>133 ± 23†</td>
<td>207 ± 29†*</td>
<td>181 ± 10*</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>178 ± 11</td>
<td>181 ± 10*</td>
<td>181 ± 11*</td>
<td>182 ± 10</td>
<td>181 ± 10</td>
</tr>
<tr>
<td>(\text{VO}_₂) (mL·min⁻¹)</td>
<td>3959 ± 435</td>
<td>3963 ± 365</td>
<td>3954 ± 359</td>
<td>3839 ± 364*</td>
<td>181 ± 10</td>
</tr>
<tr>
<td>([\text{La}^-]) (mmol·L⁻¹)</td>
<td>14.8 ± 2.8</td>
<td>15.0 ± 3.0</td>
<td>17.5 ± 3.2†</td>
<td>15.3 ± 2.9</td>
<td>181 ± 11</td>
</tr>
<tr>
<td>pH</td>
<td>7.22 ± 0.04</td>
<td>7.24 ± 0.03</td>
<td>7.20 ± 0.05</td>
<td>7.25 ± 0.04*</td>
<td>181 ± 10</td>
</tr>
<tr>
<td>[HCO₃⁻] (mmol·L⁻¹)</td>
<td>16.3 ± 1.3</td>
<td>15.7 ± 1.1</td>
<td>14.2 ± 1.3†</td>
<td>15.2 ± 1.1*</td>
<td>181 ± 10</td>
</tr>
</tbody>
</table>

*Different from P4 trials within WB1 or WB2 (P < 0.05).
†Different from WB1 within P4 or P8 trials (P < 0.05)
accordance with the work of Chidnok et al. (4), which already demonstrated that the magnitude of the \( W^\prime \) reconstitution is related to the duration and intensity of the recovery interval. According to the \( W^\prime_{\text{BAL}} \) model, the rate of \( W^\prime \) depletion does not play a role in the \( W^\prime \) recovery kinetics because work bout characteristics are currently not incorporated into its formula. The rationale for this is based on the assumption that exhaustion occurs when a critical accumulation of metabolites (e.g., \( H^+ \), Pi, ADP, and \( H_2PO_4^- \)) and/or depletion of substrates (e.g., PCr) is reached and that this “critical threshold” remains unchanged, irrespective of the exercise intensity within the severe-intensity domain (13–16). In this context, Vanhatalo et al. (15) demonstrated that intramuscular [PCr] and pH showed consistently low values at the end of four different constant work rate bouts performed above CP in both hyperoxic and normoxic conditions. More recently, Black et al. (17) analyzed muscle biopsies that were taken at the end of severe-intensity exercise bouts and found an identical muscle metabolic milieu (i.e., [ATP], [PCr], [La^-] and pH), irrespective of work rate. In accordance, [La^-], pH, and [HCO_3^-] measured after WB1 did not differ between P4 and P8 conditions in the present study, which supports the idea of a “critical threshold” in metabolites and/or substrates concentrations that could limit (intermittent) exercise tolerance. However, a shorter relative TTE of WB2 in the P8 trials was found compared with the P4 trials. Because we assumed that \( W^\prime = 0 \) at exhaustion in WB1 and, thus, the relative duration of WB2 reflects the amount of \( W^\prime \) that has been reconstituted (i.e., \( W^\prime_{\text{ACT}} \)), the present study results suggest that the reconstitution of \( W^\prime \) might be slower as the speed at which \( W^\prime \) was depleted is slower as well (i.e., P8 vs P4). This is supported by the relationship that we found between the TTE of WB1 (i.e., used to reflect the \( W^\prime \) depletion rate) and the actual \( \tau_{W^\prime} \) of the \( W^\prime \) recovery (i.e., used to reflect the \( W^\prime \) recovery rate) (Fig. 2). Recently, Chorley et al. (10) observed a fatiguing effect, slowing down the \( W^\prime \) reconstitution after repeated exhaustive bouts of severe-intensity exercise. In the present study, however, it can be questioned whether the lower \( W^\prime \) reconstitution after P8 exercise can be attributed to a similar fatiguing effect as in the work of Chorley et al. (10), given that \( W^\prime \) was depleted only once.

A second important finding of the present study is that the \( W^\prime_{\text{BAL}} \) model seems to underestimate the reconstitution of \( W^\prime \). In particular, after shorter recovery durations and after work bouts with the highest intensity/duration ratio (i.e., P4), \( W^\prime_{\text{ACT}} \) was significantly higher than \( W^\prime_{\text{PRED}} \). Skiba et al. (8) already reported that the \( W^\prime_{\text{BAL}} \) model underestimates the remaining \( W^\prime \) during short intermittent work. Furthermore, the present results are also in line with a recent study of Bartram et al. (9) in which high-performance cyclists showed substantially faster \( W^\prime \) recovery kinetics than predicted by the \( W^\prime_{\text{BAL}} \) model. In addition, the calculation of the model-predicted \( \tau_{W^\prime} \) revealed a group mean of \( 422 \pm 24 \text{ s} \) for recovery at CP_{33} and \( 554 \pm 33 \text{ s} \) for recovery at CP_{66}. These values were clearly higher than the actual \( \tau_{W^\prime} \) exhibited by our subjects, which ranged from \( 261 \pm 118 \text{ s} \) (CP_{33} recovery) to \( 350 \pm 169 \text{ s} \) (CP_{66} recovery) in P4 trials and from \( 360 \pm 112 \text{ s} \) (CP_{33} recovery) to \( 486 \pm 135 \text{ s} \) (CP_{66} recovery) in P8 trials. On average, these actual recovery rates seem to be close to results that were previously reported by Ferguson et al. (7) (\( \pm 360 \text{ s} \)) and Skiba et al. (5) (377 s). However, recovery in these studies occurred at a considerably lower work rate (i.e., 20 W). The large CV% values that are associated with the calculated \( \tau_{W^\prime} \) in the present study point at a substantial degree of variation, not only resulting from the different conditions that were used in the study but also because of a large inter- and intrasubject variability.

One of the premises inherent to the \( W^\prime_{\text{BAL}} \) model is the assumption that CP and \( W^\prime \) remain constant throughout exercise. As a consequence, the model is not sensitive to possible changes in CP and/or \( W^\prime \), although it has previously been documented that both parameters can exhibit a certain plasticity. Priming heavy-intensity exercise has been found to increase CP and/or \( W^\prime \) (18–20). Skiba et al. (21) observed faster \( W^\prime \)
recovery kinetics compared with their model predictions after intermittent exercise and suggested that one of these priming mechanisms (i.e., an increased CP or \( W^* \)) could offer an explanation. It can be suggested that also in the present study, the initial P4 or P8 work bout might have exerted a similar priming effect and, as such, could explain the underestimation of \( W^{\text{ACT}} \) by the \( W^{\text{BAL}} \) model. However, although the aforementioned studies have used heavy-intensity exercise as a priming stimulus, work bouts in this study were situated above CP (i.e., in the severe-intensity domain). Studies that have investigated the priming effects of severe-intensity exercise concluded that CP remains unaffected and that \( W^* \) is unchanged or even reduced (7,19,22). Therefore, it is rather unlikely that WB1 would have increased CP and/or \( W^* \) in the current study.

Instead of a positive priming effect on CP and/or \( W^* \), it could be questioned whether the duration of exercise, and especially the part within the severe-intensity domain, could exert a negative (i.e., fatiguing) effect on the power–duration relationship. Regarding the duration of the P8 trials compared with the P4 trials (15.5 ± 3.1 vs 10.2 ± 2 min), CP and/or \( W^* \) could be reduced because of the overall duration of the experimental bouts. This would mean that WB2 in the P8 conditions is performed at a higher relative intensity (i.e., assuming a reduction in CP) and/or with a lower work capacity above CP (i.e., assuming a reduction in \( W^* \)). If the TTE of WB2 is then related to the TTE of WB1, this would result in a lower \( W^* \) reconstitution in the P8 conditions. Clark et al. (23) showed that CP and \( W^* \), estimated using end-test power and work done above end-test power from a 3-min all-out test, had decreased 8% and 20%, respectively, after 2 h of heavy-intensity exercise. The authors associated this decrease in CP and \( W^* \) with a reduced glycogen content in Type I and Type II muscle fibers. In the present study, it seems unlikely that a reduction in glycogen content would have affected CP and/or \( W^* \), given the short duration of the exercise bouts (P4 or P8). Hence, this could not explain the differences in \( W^* \) reconstitution between P4 and P8 conditions. However, the duration of exercise within the severe-intensity domain could yield a different fatiguing effect on the subjects’ exercise tolerance during WB2, especially in the P8 trials. Although the afferent feedback from peripheral fatigue in the working muscles (i.e., accumulation of metabolites and/or depletion of substrates and thus the depletion of \( W^* \)) strongly determines the termination of exercise in the severe-intensity domain, also sensory information from other sources in the body (e.g., respiratory muscles, remote muscles not involved in the exercise, etc.) is integrated within the central nervous system. It has been proposed that exercise intensity is decreased or task failure occurs when a hypothetical “threshold,” the so-called sensory tolerance limit is exceeded (24). It is possible that the longer duration of the P8 trials has enhanced the afferent signaling originating from other sources than the locomotor muscles and that as a result, the sensory tolerance limit was reached slightly earlier in WB2 of the P8 trials. Consequently, the same level of peripheral fatigue and, thus, accumulation of metabolic and depletion of substrates might not be reached in WB2 of the P8 trials, and this implies that \( W^* \) might not be truly depleted when exercise is stopped. Although this is supported by the observation that [La\(^-\)] was lower and pH and [HCO\(_3\)] were higher at the end of WB2 in the P8 conditions, this interpretation is rather speculative because we do not have direct proof of increased afferent feedback compared with the P4 conditions.

Surprisingly however, instead of reaching consistent concentrations in metabolite accumulation and/or substrate depletion, the “critical threshold” in the P4 trials seemed to have shifted to a higher level in WB2 ([La\(^-\)] was higher and pH and [HCO\(_3\)] were lower in WB2 vs WB1), whereas this shift was not apparent in the P8 conditions. The short recovery durations that were used in this study (i.e., 2–6 min) may have been not sufficient to achieve complete lactate removal from the blood stream (25). Although the same critical threshold could have been reached within the muscle, the incomplete removal of [La\(^-\)] in the blood after recovery might have resulted in an accumulated measurement of [La\(^-\)] at the end of WB2 compared with WB1. In the P8 trials, this higher [La\(^-\)] might then not be visible because the sensory tolerance limit did not allow that critical levels in intramuscular metabolites and substrates were reached. It should be noted that these interpretations are based solely on capillary blood measurements and that we do not have information about the release of La\(^-\) from the working muscles and the clearance of La\(^-\) from the bloodstream.

On the basis of the present study results, it is clear that it is necessary to account for work bout characteristics in the modeling of \( W^{\text{BAL}} \). However, work bout characteristics encompass both work bout intensity and work bout duration, and it is less clear to what extent each of these two variables contribute to the total recovery. The observation that work bout characteristics of severe-intensity exercise may affect the rate of \( W^* \) recovery has important practical implications with regard to performance during intermittent exercise. More specifically, in intermittent sports that are characterized by prolonged duration and several surges above CP, it can be assumed that the \( W^* \) recovery will be limited and that as a consequence exercise intensity will have to be lowered earlier than expected. Modifications to the \( W^{\text{BAL}} \) model that would account for the characteristics of prior exercise efforts may improve its predictive capabilities. Particularly in sports disciplines with an intermittent character, an accurate insight into the \( W^* \) recovery kinetics would facilitate prescription and evaluation of training, as well as tactical decisions during a race (e.g., when to make a surge above CP). However, besides thorough knowledge of the general pattern of the \( W^* \) recovery kinetics, this would also require adjustments to individual characteristics of the athletes (e.g., training status, aerobic fitness, muscle fiber type, etc.). Altogether, it may be clear that caution is advised when applying the current \( W^{\text{BAL}} \) model for training or race purposes in the field. Future studies should focus on improving the predictive capabilities of the \( W^{\text{BAL}} \) by making both general and individualized adjustments to the model.
In conclusion, the present study demonstrated that the characteristics of a prior exhaustive constant work rate bout alter the $W'$ recovery kinetics. In particular, exhaustive constant work rate exercise with a higher intensity/duration ratio induced a faster reconstitution of $W'$, and moreover, the rate of $W'$ depletion and the rate of $W'$ reconstitution were correlated. These results suggest that the rate of $W'$ reconstitution may be slower when the rate of $W'$ depletion is slower as well.

Furthermore, the present study results showed that the current $W'_{BAL}$ underestimates $W'_{ACT}$, especially after shorter recovery.

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