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COMPARISON OF INTER-TRIAL RECOVERY TIMES FOR THE DETERMINATION OF CRITICAL POWER AND W' IN CYCLING

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25 Abstract

Critical Power (CP) and W are often determined using multi-day testing protocols. To investigate this cumbersome testing method, the purpose of this study was to compare the differences between the conventional use of a 24 h inter-trial recovery time with those of 3 h and 30 min for the determination of CP and W. Methods: Nine moderately trained cyclists 31 performed an incremental test to exhaustion to establish the power output associated with the 32 maximum oxygen uptake (p VO_{2}_{max}), and three protocols requiring time-to-exhaustion trials 33 at a constant work-rate performed at 80%, 100% and 105% of p $^{\dot{VO}_{2}}_{max}$ Design: Protocol A 34 utilised 24 h inter-trial recovery (CP_{24}/W_{24}), protocol B utilised 3 h inter-trial recovery 35 (CP₃/ W'_3), and protocol C used 30 min inter-trial recovery period (CP_{0.5}/ $W'_{0.5}$). CP and W'_3 were calculated using the inverse time (1/t) versus power (P) relation (P = W'(1/t) + CP). Results: 95% Limits of Agreement between protocol A and B were -9 to 15 W; -7.4 to 7.8 kJ (CP/W) and between protocol A and protocol C they were -27 to 22 W; -7.2 to 15.1 kJ 39 (CP/W). Compared to criterion protocol A, the average prediction error of protocol B was 40 2.5% (CP) and 25.6% (W), whilst for protocol C it was 3.7% (CP) and 32.9% (W). 41 Conclusion: 3 h and 30 min inter-trial recovery time protocols provide valid methods of 42 determining CP but not W in cycling.

49 INTRODUCTION

Critical Power (CP), the maximum power that can be sustained without a progressive loss of homeostasis, demarcates the heavy and the severe exercise domains (Jones, Vanhatalo, Burnley, Morton, & Poole, 2010). CP is sensitive to changes in aerobic metabolism and is therefore predictive of future performance (Jenkins & Quigley, 1990; Stickland, Petersen, & Dressendorfer, 2000). Exceeding CP results in the utilisation of its related finite anaerobic energy source, W, with the depletion rate of W being proportional to the degree to which power output (PO) exceeds CP. The determination of CP and W has traditionally required an incremental maximal exercise test to determine the power output associated with the maximum oxygen uptake ($p^{VO_{2}}_{max}$), followed by fixed intensity time to exhaustion (TTE) trials at three or more predetermined work-rates. These trials generally require one or more 61 24 h inter-trial recovery period. Critical Power testing is therefore a multi-day process.

The time consuming and resource intensive process of CP testing would be more easily incorporated into an athlete's training schedule if testing could be completed within one day. A number of authors have therefore examined alternatives to multi-day methods by employing inter-trial recovery periods from 30 min to 4 h (Barker, Bond, Toman, Williams, & Armstrong, 2012; Brickley et al., 2007; Carter et al., 2005; Dekerle et al., 2009; Housh & Terry, 1989). However, most of these investigations have failed to report direct comparisons of their findings with the traditional 24 h recovery protocol. In running, Galbraith, Hopker, & Lelliott (2014) recently demonstrated that a recovery period of 30 min in between exhaustive trials is sufficient to determine Critical Velocity (analogous of CP) but not the Anaerobic Running Distance (analogue of W') when compared against the conventional 24 h recovery 73 testing method. In cycling only, Bishop and Jenkins (1995) also directly compared protocols

vitilising 24 h and 3 h recovery periods in untrained participants, and reported no significant
differences between estimates of CP and *W* derived from each.

It is questionable whether a reduced recovery time allows for full W restoration (Ferguson et al., 2010), whilst shorter intra-trial times can result in 'primed' \dot{VO}_2 kinetics and performance enhancements (Bailey et al., 2009). Providing a 20-min intra-trial recovery, Bailey et al. (2009) demonstrated a significant increase in exercise tolerance during a subsequent 2^{nd} bout of severe exercise due to a priming of the kinetic response. However, this shorter recovery between repeated bouts of heavy intensity exercise to exhaustion are associated with elevated fatigue-related muscle metabolites, such as inorganic phosphate molecules, hydrogen and potassium ions (Westerblad, Allen, & Lännergren, 2002). Therefore, deciding on a shortest possible inter-trial recovery period, which allows a full recovery of W whilst avoiding either a detrimental or a performance enhancing effect, is challenging.

Whilst previous work has made some progress in investigating brief CP testing, the variety of modes of exercise, level of participant and recovery periods leave several questions unanswered. Arguably therefore, further research is warranted. The purpose of the present study was to directly compare estimates of CP and *W*' derived using inter-trial recovery periods of 24 h, 3 h, and 30 min using trained cyclists. We hypothesised an acceptable level of agreement (i.e. mean difference \pm 1.96 SD) between CP derived from the three different protocols (with 24 h serving as the criterion measurement). In relation to *W*' we hypothesised an acceptable level of agreement with the criterion measure in the 3 h recovery method only.

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98 DESIGN

99 Participants were nine moderately trained recreational road cyclists [age 33 ± 8 yr, body mass 100 78 ± 10 kg, maximal oxygen consumption (${}^{\dot{V}O_2}_{max}$) 3.9 ± 0.4 L·min⁻¹, power associated with 101 ${}^{\dot{V}O_2}_{max}$ (p ${}^{\dot{V}O_2}_{max}$) 358 ± 35 W]. The study was approved by the institutional Ethics 102 Committee in accordance with the Declaration of Helsinki. Prior to providing written 103 informed consent, cyclists were fully informed of the nature and risks of the study.

Protocol. In visit 1, values for $\dot{VO}_{2_{\text{max}}}$ and $\dot{PVO}_{2_{\text{max}}}$ were established. In randomised order, each cyclist then completed three CP protocols. Protocol A used a traditional 24 h inter-trial recovery (3 visits to laboratory), protocol B a 3 h inter-trial recovery (1 visit to laboratory), and protocol C a 30 min inter-trial recovery (1 visit to laboratory). For each protocol fluid intake was permitted ad libitum. During all tests, participants were blinded to power and elapsed time. Participants were required to refrain from heavy exercise and from food and caffeine intake for 24 h and 3 h prior to testing, respectively. To minimise training effects, all visits were separated by a minimum of 24 h and were completed within a maximum period of 14 days. Each cyclist completed each of their six visits at the same time of day.

115 A road bicycle equipped with a PowerTap Elite wheel (CycleOps, Madison, USA) and a 116 magnet for direct cadence measurement was used in this study. The road bike was attached to 117 a Computrainer system (RacerMate, Seattle, USA). The saddle and handlebar were adjusted 118 to suit each participant and settings were replicated exactly during subsequent tests. The 119 PowerTap device was zero offset prior to each test according to the manufacturer's 120 instructions.

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Maximal oxygen uptake test protocol. Following a standardised warm-up, cyclists completed a progressive, incremental exercise test to exhaustion. The maximal test commenced at a work rate of 150 W. Thereafter, intensity increased at a step rate of 20 W min⁻¹. Cyclists were allowed to self-select cadence and were instructed to maintain this cadence throughout all tests. The test was terminated when cadence dropped by more than 10 rev-min⁻¹ for more than 10 seconds. Expired gases were collected breath-by-breath throughout the test using a Cortex MetaLyzer 3B gas analyser (Cortex Biophysik, Leipzig, Germany). Fingertip blood lactate was analysed using the Biosen C line analyser (EFK Diagnostics, Barleben, Germany), and heart rate (HR) was continuously observed using the monitor built in the Cortex gas analyser. $p^{VO_{2}}_{max}$ was calculated as the highest 30-s mean PO value (W). VO_{2 max} was calculated as the highest mean oxygen consumption over the same period.

Critical Power tests. Each protocol required cyclists to complete three TTE trials at work-rates equivalent to 80%, 100% and 105% p $VO_{2_{max}}$. Protocol A used a randomised TTE trial order, with protocol B and C requiring participants to perform trials in the order lowest work rate (i.e., 80% $p^{\dot{VO}_{2}}_{max}$) to highest work rate (i.e., 105% $p^{\dot{VO}_{2}}_{max}$). After a 5-min standardised warm-up, the test resistance was set and participants were instructed to maintain their preferred cadence for as long as possible. At TTE termination participants continued with a 5 minute unloaded cycling phase before dismounting the bike. HR (bmin⁻¹), PO (W) and cadence (rev min⁻¹) were recorded continuously via the PowerTap, and expired gases were continuously sampled. Tests were terminated when cadence dropped by 10 rev min⁻¹ below preferred cadence for more than 10 seconds. Fingertip capillary blood samples were 145 collected prior to and post TTE trials. All cyclists reached their individual VO_2 max value (±

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146 0.08 L^{min⁻¹}), a post-test blood [lactate] of ≥ 8 mM and a HR within ± 5 beats of their 147 maximal HR values established during the \dot{VO}_2 max test.

Calculation of Critical Power and W. Linear regression was used to calculate CP and W150 using the power-1/time (P = W'(1/t) + CP) model. Results using protocol A were termed 151 CP_{24}/W'_{24} and for protocol B and protocol C they were termed CP_3/W'_3 and $CP_{0.5}/W'_{0.5}$ 152 respectively.

Statistical Methods. Data were examined using the Shapiro-Wilk normality test. Pearson product moment correlation analysis was used to provide an indication of the strength of relationship between the different inter-trial protocols for CP or W. Agreement between different testing protocols for CP_{24}/W'_{24} , CP_3/W'_3 and $CP_{0.5}/W'_{0.5}$ was assessed using a repeated measures ANOVA and Limits of Agreement (LOA; Atkinson & Nevill, 1998; Bland & Altman, 1986). A repeated measures ANOVA was also used to assess differences between the protocol specific durations of TTE trials and resting and in-exercise blood [lactate] between and within different protocols. Linear regression was used to calculate the Standard Error of Estimates (SEE) to determine the error associated with predicting experimental CP and W' values. Partial eta squared (η_{p}^{2}) values are reported to provide an estimate of standardised effect size (small $\eta_p^2 = 0.01$; moderate $\eta_p^2 = 0.06$; and large $\eta_p^2 = 0.14$). Greenhouse-Geisser correction was used to correct the violation of sphericity. Statistical significance was accepted at P < 0.05. Results are reported as mean and standard deviation (SD) unless otherwise stated.

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171 **RESULTS**

- 172 CP and *W* were normally distributed. Repeated measures ANOVA demonstrated no
- 173 significant differences for CP, F(1.4,11.17) = 1.22, p = 0.31 and W'(F[1.6,12.89] = 4.03, p =
- 174 0.07) between protocols. Where the assumption of sphericity was not met, the Greenhouse-
- 175 Geisser correction was used. There was a large effect of the protocol for CP and for $W'(\eta_{\mathbb{P}}^2 \ge$
- 176 0.14). Applying the power-1/time CP model, mean r for protocol A was 0.94 ± 0.12 (SEE
- 177 10.0 ± 9.0 W) for protocol B it as was 0.97 ± 0.04 (SEE 8 ± 6 W) and for protocol C it was

178 0.99 ± 0.01 (SEE 5 ± 3 W). Table 1 illustrates mean difference and 95% LoA for all results

179 with Table 2 illustrating mean CP/W' values and average prediction errors for the

180 experimental protocols. Figure 1 depicts a graphical presentation of the Bland-Altman

181 analysis, including SEE and r values.

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---Fig 1 about here---

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Significant differences (P < 0.05) were observed for mean resting [lactate] in protocol C between 80% TTE trials and both 100% and 105% TTE trials but also between protocol C 186 100% and 105% TTE trials and their protocol B and C counterparts. For post [lactate], 187 significant differences were observed between protocol A 80% TTE trials and 105% TTE 188 trials in protocols B and C (Table 3). No significant differences in TTE durations between 189 respective protocol trials were observed (Table 4).

- 190 ----Table 1 a and 1 b about here---
- 191 ----Table 2 a and 2 b about here---
- 192 ----Table 3 about here---

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195 DISCUSSION

The present study investigated whether recovery times of 3 h or 30 min are sufficient to 196 derive values of CP and W equivalent to those derived using the 'standard' 24 h inter-trial 197 recovery method. Mean differences between protocol A (24 h recovery) and protocol B (3 h 198 recovery) and between protocol A and protocol C (30 min recovery) were 3 ± 6 W and $-2 \pm$ 199 12 W respectively. This suggests that CP can be determined using either a 3 h or a 30 min 200 inter-trial recovery period. LoA for standard and experimental CP_3 and $CP_{0.5}$ values (Table 201 1a; Fig 1) also suggest an acceptable level of agreement between the 24 h and the shorter 202 recovery duration protocols. Table 2a demonstrates average prediction errors for all 203 experimental CP. Our levels of error are considerably lower than those reported by 204 Nimmerichter, Williams, Bachl, & Eston (2010), who suggested that a field test with a 205 random error of 5% and levels of agreement between -0.4 W and 49 W was valid. CP 206 findings were also consistent with those of Bishop and Jenkins (1995), and of Galbraith et al. 207 (2014) which further suggests that recovery periods as short as 30 min provide good 208 estimates of CP/CV. 209

Although not reaching statistical significance, data for *W*['] indicated an unacceptably low level of agreement (Table 1b), as well as high average prediction errors for both 3 h and 30 min testing protocols (Table 2b). These data allow us to reject our hypothesis that an acceptable level of agreement with the criterion measure would be observed in the 3 h recovery method. Previous research suggests that prior exercise such as a TTE trial can be detrimental to subsequent exercise when it is too intense (Wilkerson, Koppo, Barstow, & Jones, 2004) or when recovery periods are too short (Ferguson et al., 2007). Arguably only minimal 217 detrimental effects are evident in the current study, as indicated by shorter exhaustive trial 218 durations in Table 4. However, these might explain the lack of agreement for W across 219 protocols. Our results, furthermore, support findings by Galbraith et al. (2014) who also 220 identified differences in values of anaerobic running distances using a 30 min and 60 min 221 inter-trial recovery method. However our results do not explain findings for W under 222 protocol B, where a 3 h inter-trial recovery period should have been sufficiently long or a full 223 reconstitution of this parameter.

Nielsen, de Paoli & Overgaard (2001) suggested that acidosis caused by elevated blood [lactate] actually protects the muscle from fatigue. Moreover, an optimal [lactate] of $\sim 2-3$ mM has been suggested by Jones et al. (2003) as a level at which, through the preservation of muscle K^+ , performance can be enhanced. Resting blood [lactate] was significantly elevated for both the 100% and 105% TTE trials in protocol C (Table 3) without indicating such performance enhancement. Protocol C 105% TTE trial durations on average were ~36 s shorter when compared with their protocol A counterparts. Ferguson et al. (2010) suggested that lactate recovery kinetics are slower than those of W, which implies that full recovery was not evident in protocol C, since W' was considerably smaller when compared to protocol A.

There appears to be a lack of consensus as to the true nature and role of W'. W' defined as a finite amount of energy was believed to result in exhaustion when depleted (Moritani, Nagata, Devries, & Muro, 1981). More recently W' has been suggested to represent the accumulation of fatigue-related metabolites to some critical tolerable limit (Coats et al., 2003; Jones, Wilkerson, DiMenna, Fulford, & Poole, 2008). According to Coats et al. (2003), et al. (2003), depletion of W' resulting from a prior bout of severe exercise negatively influences

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subsequent performances around CP intensity. This was seen in the present study in that 100% and 105% test durations under protocol C conditions were shorter than those of their protocol A counterparts. Challenging a finite capacity-based explanation for tasks to failure, Ferguson et al. (2010) explored the effects of an exhaustive conditioning bout on CP and W'. The study demonstrated that W' reflects an ability to exercise under increasing levels of fatigue caused by its own utilisation. Consequently, Ferguson et al. (2010) found no differences in CP, but a reduction in W when employing different recovery durations (2 to 15 min) after a W depleting exercise bout followed by TTE trials. In agreement with Ferguson et al. (2010), the results of the present study suggest the reductions in time to exhaustion after prior exhaustive exercise seem to be primarily dependent on the variability of W'. In this regard, Ferguson et al (2010) demonstrated an exponential repletion of W' and not, as is assumed by the 2-parameter CP model, a linear one, and therefore this might suggest that W' is not fully reconstituted by the end of the 3 h and 30 minute recovery period used in Protocol B and C.

Investigating the influence of moderate hypoxia on high intensity exercise tolerance, Dekerle, Mucci & Carter (2012) found that the ranges of TTE did not differ between normoxic and hypoxic conditions. However CP was significantly affected (mean 13%) under hypoxic conditions with W not demonstrating a significant difference but exhibiting large intra-individual responses (-36 to +66%). Dekerle et al. (2012) consequently questioned whether the two parameter model allows a valid estimation of W. Indeed some recent research attempts have been made to account for some shortcomings in the two-parameter CP model (Chatagnon, Pouilly, Thomas, & Busso, 2005; Heubert et al., 2005) with Gaesser, Carnevale, Garfinkel, Walter, & Womack (1995) highlighting an inherent difficulty in accurately and

reliably determining W. With an apparent disagreement in the literature about the true constitution of this parameter, we can only suggest that an additional TTE trial would have resulted in an increased accuracy of CP and W predictions whilst arguably adding to a time consuming and cumbersome testing protocol. It is however unlikely that W' derived through 4 TTE trials would have provided acceptable results. The present study was limited in that 3 h and 30 min protocols were not repeated on more than one occasion. Therefore, it is difficult to ascertain the level of reliability within these methods to determine CP. Nevertheless, using a similar protocol, Karsten et al. (2015) investigated the reliability of CP in the field with 30 min recovery periods in a group of recreational athletes. Over three repeated trials, Karsten et al. (2015) reported mean coefficient of variation values of 2.35%, with intraclass correlation coefficient values of value of 0.99 (CI 0.98–0.99). Therefore, it is not unreasonable to expect that similar levels of reliability could be expected in the current study.

279 CONCLUSION

280 CP has traditionally been determined using 24 h inter-trial recovery periods. Results of the 281 present study suggest a high agreement and a low prediction error in CP using 3 h and 30 min 282 inter-trial recovery periods. With *W*' requiring further investigations to fully understand its 283 mechanistic underpinnings, CP appears to be robust to the manipulation of TTE recovery 284 times.

286 PRACTICAL APPLICATIONS

287 CP can be determined in a single session of 1.5 h. A substantially reduced inter-trial recovery
288 period – as low as 30 min – increases the possibility for CP testing to be incorporated into an
289 athlete's training regimen.

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414 Figure captions

. in represent the mean difference. . and the dashed line represents 95% Look Figure 1. Bland-Altman plots of the relationship (panel A and B) and the limits of agreement (panel C and D) between CP₂₄ and CP₃ and between CP₂₄ and CP_{0.5} respectively. In panel C and D the horizontal line represent the mean difference between CP24 and CP3 and between CP_{24} and $CP_{0.5}$, and the dashed line represents 95% LoA.

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Figure 1. Bland-Altman plots of the relationship (panel A and B) and the limits of agreement (panel C and D) between CP24 and CP3 and between CP24 and CP0.5 respectively. In panel C and D the horizontal line represent the mean difference between CP24 and CP3 and between CP24 and CP3 and between CP24, and CP0.5, and the dashed line represents 95% LoA.

254x190mm (96 x 96 DPI)

	Table 1a	. Mean	Difference	(±SD),	95%	Limits of A	Agreement	between	CP results
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	Mean Difference (W)	95% LoA (W)
Prot. A vs. B/CP ₂₄ vs. CP ₃	3 ± 6	-9 to 15
Prot. A vs. C/CP ₂₄ vs. CP _{0.5}	-2 ± 12	-27 to 22

Table 1b. Mean Difference (±SD), 95% Limits of Agreement between W'results

	Mean Difference (kJ)	95% LoA (kJ)
Prot. A vs. B/W' ₂₄ vs. W' ₃	0.2 ± 3.9	-7.4 to 7.8
Prot. A vs. C/W' ₂₄ vs. W' _{0.5}	3.9 ± 5.7	-7.2 to 15.1

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Table 2a. Mean CP (±SD), Standard error of estimates (lower and upper confidence limits) and average prediction errors (%)

	Mean (W)	SEE (W)	Lower CL	Upper CL	Average pred. error (%)
Protocol A vs. B:	277 ± 26 vs.	7	4.7	12.0	2.5
(CP ₂₄ vs. CP ₃)	274 ± 25				
Protocol A vs. C:	277 ± 26 vs.	10	7.1	18.1	3.7
$(CP_{24} \text{ vs. } CP_{0.5})$	279 ± 33				

Table 2b. Mean W' (±SD), Standard error of estimates (lower and upper confidence limits) and average prediction errors (%)

	Mean (kJ)	SEE (kJ)	Lower CL	Upper CL	Average pred. error (%)
Protocol A vs. B:	15.2 ± 4.7 vs.	3.9	2.7	7.0	25.6
$(W'_{24} \text{ vs. } W'_{3})$	15.0 ± 4.2				
Protocol A vs. C:	15.2 ± 4.7 vs.	5.0	3.5	9.0	32.9
$(W'_{24} \text{ vs. } W'_{0.5})$	11.3 ± 3.5				

Applying the power-1/time CP model, mean r for protocol A was 0.94 ± 0.12 (SEE 10 ± 9 W) for protocol B it as was 0.97 ± 0.04 (SEE 8 ± 6 W) and for protocol C it was 0.99 ± 0.01 (SEE 5 ± 3 W).

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Prior TTE trial	Lactate (mM) 80% TTE trial	Lactate (mM) 100% TTE trial	Lactate (mM) 105% TTE trial
Protocol A	1.5 ± 0.6	1.5 ± 0.7	1.4 ± 0.6
Protocol B	1.5 ± 0.5	1.8 ± 0.8	1.5 ± 0.5
Protocol C	1.2 ± 0.3	$3.5 \pm 0.8^{*}$	$4.1 \pm 1.3^{**}$
Post TTE trial	Lactate (mM)	Lactate (mM)	Lactate (mM)
	80% I I E trial	100% ITE trial	105% ITE trial
Protocol A	12.5 ± 1.5	11.8 ± 3.0	10.5 ± 2.8
Protocol B	13.2 ± 2.7	11.0 ± 2.6	10.1 ± 2.3
Protocol C	11.5 ± 3.1	10.4 ± 2.2	9.2 ± 2.0

Table 3. Group	mean (±SD) resting and	post-TTE	trials blood	[La]	(mM) results
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A and L Significantly different to protocol A and B TTE trial resting value (p = 0.000)

** Significantly different to protocol A and B TTE trial resting values (p = 0.000)

	00/0111 (8)	100 /0 1 1 L (S)	103 /0 1 1 E (S
Α	650 ± 237	251 ± 81	179 ± 59
B	623 ± 213	222 ± 81	169 ± 49
C	578 ± 170	$\frac{210 \pm 79}{210 \pm 79}$	143 ± 23
TTE trial	Protocol A vs. B	Protocol A vs. C	Protocol B vs
80% TTF	p-value	<u>p-value</u> 0.75	<u>p-value</u> 0.87
00 % 11E 100% TTE	0.83	0.10	0.37
105% TTEC	0.84	0.10	0.08

n durations (±SD) of set TTE trials for each protocol and p-values rotocol comparisons

Protocol B vs. C