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#### LETTER TO THE EDITOR

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# Functional threshold power and the (manufactured) critical power controversy

#### Stephen Wong (D<sup>a</sup>, Mark Burnley (D<sup>b</sup>, Alexis R Mauger (D<sup>a</sup>, Fenghua Sun (D<sup>c</sup> and James Hopker (D<sup>a</sup>)

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#### ABSTRACT

We read with concern yet another communication from Dotan regarding the critical power (CP) concept which contains a litany of factual errors, false statements, and dated physiological interpretations. Space does not permit us to rebut every incorrect point made about our work (Wong et al., 2022) and the wider field in which it sits, but we will address what we consider to be some of the more egregious errors in his letter. We would first note, however, that our paper was not actually focused on the critical power concept.

#### **ARTICLE HISTORY**

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#### **KEYWORDS**

VO2 kinetics; functional threshold power; maximal metabolic steady state; VO2 slow component; VO2 steady state

### To the editor,

We read with concern yet another communication from Dotan regarding the critical power (CP) concept which contains a litany of factual errors, false statements, and dated physiological interpretations. Space does not permit us to rebut every point made about our work (Wong et al., 2022) and the wider field in which it sits, but we will address what we consider to be some of the more contentious points in his letter. We would first note, however, that our paper was not actually focused on the critical power concept.

In his letter, Dotan states that "no single test can precisely predict maximal performance at durations other than its own". In contrast, the 3 min all-out test developed by Vanhatalo et al. (2007) has been shown to accurately predict performance in constant load exercise (Vanhatalo et al., 2007, 2011). He goes on to confidently state that " ... shorter maximal performances, such as the FTP<sub>20</sub> test, depend more on glycolytic power and less on aerobic capacity than do longer maximal efforts, such as the FTP<sub>60</sub> test". Neither "glycolytic power" nor "aerobic capacity" are defined, but it has been well known since the synopsis of Gastin (2001) that maximal paced efforts beyond 2-3 min are heavily dependent on oxidative energy transfer. As a result, the difference in the net glycolytic energy contribution between a 20 min effort and a 60 min effort is likely to be small and does not justify statements alluding to "vastly different capacities" between such efforts.

In several places, the letter imposes upon us interpretations we either do not hold, or states interpretations that are too simplistic to be credible. For example, he states "Although  $FTP_{20}$  is very close to the 2–15-min bout durations, which Wong et al. consider a pre-requisite for proper CP determination ... ". At no point in our paper do we make such a statement, and the statement itself does not accurately represent the methods commonly used to determine CP (*cf.* Burnley, 2023). In the paragraphs following this statement, a number of erroneous physiological interpretations are presented. First, it is

implied that blood lactate concentration is a measure of glycolysis. Blood lactate represents the dynamic balance between lactate appearance and disappearance in the blood, not the rate of glycolytic metabolism. It is not an appropriate variable with which to determine whether *muscle* metabolism is in a steady state. Furthermore, our figure 2 is interpreted to suggest an ~150 mL/min rise in pulmonary oxygen uptake (\_V O<sub>2</sub>) occurred over the last 16 min of the FTP<sub>20</sub> +15W test. This number was not reported in the paper and seems to be something estimated "by eye". It is further suggested that the attainment of a steady state\_VO<sub>2</sub> is "patently false". We invite readers to examine the final two data points of this condition: \_VO<sub>2</sub> does not change. What is being interpreted is a \_VO<sub>2</sub> slow component, which stabilises after 10–15 min during heavy exercise, exactly as shown in the plot.

Dotan claims that "Creeping \_VO<sub>2</sub> is commonly seen in intense prolonged exercise, due to rising core temperature and the associated metabolic and thermoregulatory cost". However, the notion that the rise in VO<sub>2</sub> during intense exercise is mediated by core temperature was refuted more than 30 years ago, because muscle, blood and core temperatures also rise significantly during moderate intensity exercise, in the absence of a progressive increase in \_VO<sub>2</sub> (Poole et al., 1991). Furthermore, passively heating the muscle by 2-3°C prior to performing heavy exercise does not increase \_VO<sub>2</sub> (Burnley et al., 2002; Koppo et al., 2002). Lastly, Koga et al. (1997) showed that the VO<sub>2</sub> slow component was actually reduced when muscle temperature was passively increased. In short, the increase in \_VO<sub>2</sub> beyond the initial primary phase during heavy- or severe-intensity exercise is not caused by an increase in muscle or core temperature.

Finally, in response to the question: "if CP's predictive powers are limited only to the 2–15 min tested range, what is the practical or conceptual value of the construct?" we would answer, first, that its predictive powers are not so limited (Burnley, 2023), and, second, that the range of performances

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to which the CP concept does apply encompasses the majority of competitive endurance events in running, swimming, cycling and rowing. This is why the CP concept is widely used in applied practice (e.g., Pugh et al., 2022).

We consider the critical power "controversy" to be manufactured because it is based upon the misunderstanding, misinterpretation, and misrepresentation of the concept and its underlying physiology.

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