When is it appropriate to compare critical power to maximal lactate steady-state?

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When is it appropriate to compare critical power to maximal lactate steady-state?

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In his editorial, de Lucas indicates that the coherence between the intensities corresponding to maximal lactate steady-state (MLSS) and critical power (CP) is dependent on the methods and model used to determine CP. While there is no question that different mathematical models applied to power-time series datasets of varying time limits can produce different CP values, we completely disagree that this refutes an equivalence between these two constructs. CP is not a variable that is dependent on power output and time to exhaustion; rather, it is a parameter that contributes to determining how long one can sustain a given power output!

The CP concept is predicated on the hyperbolic relationship that exists between exercise intensity and tolerable duration at that constant intensity (Monod and Scherrer 1965). From a mathematical standpoint, such a relationship implies that exercise performed constantly at the asymptotic intensity should be tolerated indefinitely. From a physiological perspective, we know that this intensity cannot be sustained ‘indefinitely’ because other factors (e.g. substrate depletion, muscle fatigue, hyperthermia, motivation, etc.) inevitably intervene to determine tolerable duration. The discord between the mathematical and physiological constructs that predict respectively ‘infinite’ versus ‘finite’ exercise tolerance at CP has contributed to its notoriously ambiguous definition as “the highest intensity that can be sustained for a prolonged time”. However, in our view, acceptance as true of the CP output from a mathematical model applied to a power-time series dataset (without verification) is the main obfuscator of what CP truly represents.

In his letter, de Lucas, like many others, neglects to acknowledge the appropriate precedence that links the mathematical CP model to distinct underlying physiology. Poole et al., (1988) were among the first to identify CP as the highest intensity above which respiratory and metabolic measures (which included blood lactate) may no longer achieve steady-state. Since this seminal
study, CP has been verified to demarcate an intensity above which oxygen uptake, blood and muscle lactate and hydrogen ion concentration, intramuscular phosphocreatine, and inorganic phosphate may no longer be stabilized (e.g. Jones et al., 2008; Black et al., 2017). Collectively, these studies expanded the colloquial definition of CP to: “the highest intensity that can be sustained for a prolonged time solely by oxidative energy provision”. An important caveat of this expanded definition is that exercise at CP does not draw upon anaerobic metabolism and therefore progressive depletions in phosphocreatine and progressive accumulations of lactate in muscle and blood are not evident with time. Presumably, this minimizes metabolic and acid-base disturbance and reduces (or delays) the initiation of fatigue processes – prolonging exercise tolerance. Therefore, the physiological response expected at CP encompasses the concept of MLSS; any differences observed between these two interdependent indices are related to inaccuracies inherent with the methods and protocols designed for their determination.

So why bother comparing CP to MLSS? de Lucas points out that – in the majority of studies – CP is associated with time limit ranges of “20 to 60 minutes” and that “lactate steady state responses” are not observed during continuous exercise at CP. In our view, these findings are precisely why such comparisons are warranted. Time limit ranges aside, how can it truly be CP if the physiological responses do not exhibit what is known to occur at this intensity? For starters, we would argue that CP was not actually “determined” in these studies. Traditionally, CP is determined by acquiring power-time series data over several visits and fitting a model to those data. The CP parameter is an element of the model that contributes to determine the dependent variables (i.e., time-to-exhaustion at a given PO). Technically speaking, a greater model fit suggests a truer description of the characteristics of the underlying physiological system and greater accuracy of the estimated parameters to predict time-to-exhaustion at any
given constant-power output. However, due to the limited number of data points that are typically available and the difficulty of producing truly exhaustive efforts (particularly for longer exhaustive efforts), it is nearly impossible to ascertain the accuracy of the model (i.e., how close the parameter estimate is to the actual CP) without experimental validation. Still, the model-derived output of CP is often assumed (in our view, incorrectly) to represent the actual CP. The protocol-dependence of CP estimations speaks directly to the illegitimacy of this assumption. For example, different trial ranges (e.g. maximum of 10, 15, or 20 min) and fitting strategies (e.g. 2- and 3-parameter hyperbolic, linear models) applied to construct and fit, respectively, an individual’s power-time data are known to yield drastically different CP values. Are we to assume that the individual’s actual CP is changing? No! Only the estimation of CP is changing! Therefore, “protocol-dependence of CP” cannot be used as evidence in favour of an inequivalence between CP and MLSS. However, it does emphasize that validation of the CP estimate, irrespective of the method and model, is an obligatory step to increase the accuracy of CP determination.

Based on the expanded definition of CP stated above, there are two features that could be used to validate its model-derived estimation: 1) the time limit at CP; and 2) the physiological response profile. Because the former is dependent on a multitude of factors which are likely individual-specific, verifying CP based on a pre-determined time range is not feasible. However, the physiological response profiles of exercise above versus below CP have been shown to be consistent between individuals including healthy young individuals (Poole et al. 1988), older adults (Overend et al. 1992), and patients with chronic conditions (Mezzani et al. 2010). Since blood lactate measurement is relatively low cost and easy to perform and interpret, we have advocated its use to verify estimations of CP by incorporating a 30-minute constant-intensity
trial with a protocol identical to traditional MLSS testing. In Mattioni Maturana et al., (2016) we demonstrated that exercise at CP derived from the two-parameter hyperbolic model applied to 5 exhaustive cycling bouts engendered a non-stable blood lactate response between the 10th and 30th minute of exercise in 12 of 13 individuals. In contrast to the interpretation of de Lucas, the main message from this study was not that CP and MLSS were different rather, MLSS testing was used to show that in most instances, estimates of CP may not accurately predict metabolically stable responses expected at the ‘true’ CP. These data further emphasize the need to experimentally verify model-derived estimates of CP.

de Lucas also correctly points out a discrepancy between the results of Keir et al., (2015) and Mattioni Maturana et al. (2016). That is, the 3-parameter hyperbolically-derived CP was shown to approximate (mean bias = -2 W, p>0.05) and overestimate (mean bias = 19 W, p<0.05) the MLSS in two different sample groups, respectively. Despite the difference in bias, the dispersion of the data was remarkably similar in both studies (the 95% confidence interval was 54 W vs 61 W, respectively) indicating that the error associated with the estimate of CP was consistently high and that model parameter estimates were not accurate in approximating the ‘true’ CP. We also demonstrated that the CP estimated from traditional modelling methods and the 3-min all-out test (Burnley et al. 2006) differed in many participants by more than 30 W (this despite similar group mean power outputs) indicating large intra-individual variability between methods (Mattioni Maturana et al. 2016). Furthermore, using blood lactate-based experimental verification of CP, we recently demonstrated that young healthy individuals were capable of self-selecting their CP with greater accuracy than traditional methods (Mattioni Maturana et al. 2017).
Regardless of the model used to ‘estimate’ CP, we cannot continue to accept the interpretation that the model output represents the ‘true’ CP. We acknowledge that MLSS-type protocols have their own limitations and that experimental verification increases the time commitment of ascertaining this critical intensity; however, experimental rigor should not be abandoned for convenience, especially in a research context. When CP is of interest it is imperative that we remember what we are measuring and why. de Lucas poses the question: “Is it still necessary to compare critical power to maximal lactate steady-state?” In our opinion, theoretical comparisons are not necessary – they are the same – but from a practical perspective it is obligatory to do so!
References


