

## Critical oxygenation: Can muscle oxygenation inform us about critical power?

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### ABSTRACT

The power-duration relationship is well documented for athletic performance and is formulated out mathematically in the critical power (CP) model. The CP model, when applied properly, has great predictive power, e. g. pedaling at a specific power output on an ergometer the model precisely calculates the time over which an athlete can sustain this power. However, CP presents physiological inconsistencies and process-oriented problems. The rapid development of near-infrared spectroscopy (NIRS) to measure muscle oxygenation ( $SmO_2$ ) dynamics provides a physiological exploration of the CP model on a conceptual and empirical level. Conceptually, the CP model provides two components: first CP is defined as the highest metabolic rate that can be achieved through oxidative means. And second, work capacity above CP named  $W'$ .  $SmO_2$  presents a steady-state in oxygen supply and demand and thereby represents CP specifically at a local level of analysis. Empirically, exploratory data quickly illustrates the relationship between performance and  $SmO_2$ , as shown during 3-min all-out cycling tests to assess CP. During these tests, performance and  $SmO_2$  essentially mirror each other, and both CP and  $W'$  generate solid correlation with what would be deemed their  $SmO_2$  counterparts: first, the steady-state of  $SmO_2$  correlates with CP. And second, the tissue oxygen reserve represented in  $SmO_2$ , when calculated as an integral corresponds to  $W'$ . While the empirical data presented is preliminary, the proposition of a concurring physiological model to the current CP model is a plausible inference. Here we propose that  $SmO_2$  steady-state representing CP as critical oxygenation or CO. And the tissue oxygen reserve above CO would then be identified as  $O'$ . This new CO model could fill in the physiological gap between the highly predictive CP model and at times its inability to track human physiology consistently. For simplicity's sake, this would include acute changes in physiology as a result of changing climate or elevation with travel, which can affect performance. These types of acute fluctuations, but not limited to, would be manageable when applying a CO model in conjunction with the CP model. Further, modeling is needed to investigate the true potential of NIRS to model CP, with a focus on repeatability, recovery, and systemic vs local workloads.

### Introduction

In their 2016 publication, Poole and colleagues [42] outline the relevance of the hyperbolic power-duration relationship termed the critical power (CP) model, as accredited to findings by Archibald Hill in 1925 [22] and later Monod and Scherrer [36]. The model identifies the boundary between tolerable and intolerable severe intensity exercise [7,8]. Exercise attempted above this boundary – known as CP – will result in exhaustion when attempting to maintain a constant work rate. This time to exhaustion is in direct relation to the extent to which work is being performed above CP [49,50]. When plotting constant power or speed in relation to time to exhaustion the obvious non-linearity of this

relationship becomes apparent. There is a curvilinear nature in the strong decline of performance in relation to duration, with an ever-developing asymptote of tolerable performance as the time axis extends outwards [7,8].

Perhaps, the extremes of performance best serve as an example of this phenomenon. Consider, at first the top speed of current 100 m world record holder Usain Bolt, at speed exceeding 40 km/h (average greater than 37 km/h). Secondly, consider the average speed of current 1500 m world record holder Hicham El Guerrouj, at 26.2 km/h. Then, finally consider the average speed of current marathon world record holder Eliud Kipchoge, at approximately 21 km/h; Eliud Kipchoge's performance is highly predicted by a CP model [27]. All three speeds represent

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the maximum of human performance, but a very different frame of reference when discussing tolerable performance. All three speeds maximize speed for the allotted distance, but the fall-off in speed between 100 m and 1500 m is greater than 10 km/h for a change in distance of 1400 m. Whereas, the decrease in average speed between 1500 m and a full marathon – a distance difference of more than 40 km – is merely 5 km/h. This simple example should make the relationship between speed or power and time to exhaustion clear, as well as its adaptability and trainability; thereby highlighting the value of the CP model. The model itself has two main components. First and foremost is the asymptote described above, designated CP. In our runner example, Eliud Kipchoge benefits from an extremely high CP or critical speed, 6.04 m/s to be exact [27]. The second component is the finite work capacity that can be achieved above CP, designated  $W'$ ; in other words, the integral of the power-duration curve. In our running example, Usain Bolt benefits from an extremely large  $W'$  (or  $D'$  for distance rather than work), which is associated with this extreme speed above what must be a much lower critical speed.

CP has proven to be a formidable diagnostic tool with predictive power in various endurance disciplines as would be expected; including cycling [1], running [34], and swimming [11]. Attempts to apply the CP model to other, non-classic or non-endurance variable-pace exercise sporting disciplines are also on the rise [30,50]. For example, Giles and colleagues [19] recently showed that critical force, as a counterpart to CP can be used to understand the limits of exercise tolerance in finger flexor performance in rock climbers. Still, how well a CP model represents the excess of athletes and athletic disciplines is difficult to evaluate.

### Physiological inconsistency

Numerous avenues of critique exist and perhaps the most intriguing is the question of physiological inconsistency [6,12,55]. The human system is inherently volatile as it struggles with daily adjustments to function as efficiently as possible. High-performance demands and training load increase this volatility. It is therefore difficult to assume that a CP model – even if individualized for a specific athlete – would represent performance accurately in all situations, under all conditions, at all times. For example, altitude change directly affects CP, reducing CP in trained cyclists [52]. Concretely, in the study by Townsend and colleagues, nine trained cyclists completed time trials at five different altitudes between 250 m and 4250 m to determine CP and  $W'$ . CP decreased with increasing altitude and at maximum altitude,  $W'$  was also decreased. Salam and colleagues [46] showed that mental fatigue can affect the CP model. In this case, trained cyclists completed a 30-min Stroop task to induce mental fatigue before a time to exhaustion test. The mental fatigue had no negative effects on CP, but reduced  $W'$ .

### Process-oriented problems

Alongside these physiological inconsistencies, the CP model is also susceptible to process-oriented problems. For example, Bishop and colleagues demonstrated that the duration selected for the predictive test used to determine CP significantly alters the outcome [4]. In the study, ten students completed five all-out predictive tests at preselected constant power out-puts. Then three variations of the CP model were calculated, 1) using the three highest power outputs tests, 2) the three lowest power output tests, and 3) using tests 1, 3, and 5. The CP prediction was significantly different for the three calculations with the group concluding that duration and intensity selected for the test(s) significantly influence the results. The same was confirmed by Mattioni Maurana and colleagues [35] using a similar design with five trials, which also pointed out that the mathematical model chosen to calculate CP also varied the results significantly. For example, when choosing an exponential model, the CP outcome is significantly higher than when choosing either a two or three-parameter hyperbolic model. The effect of

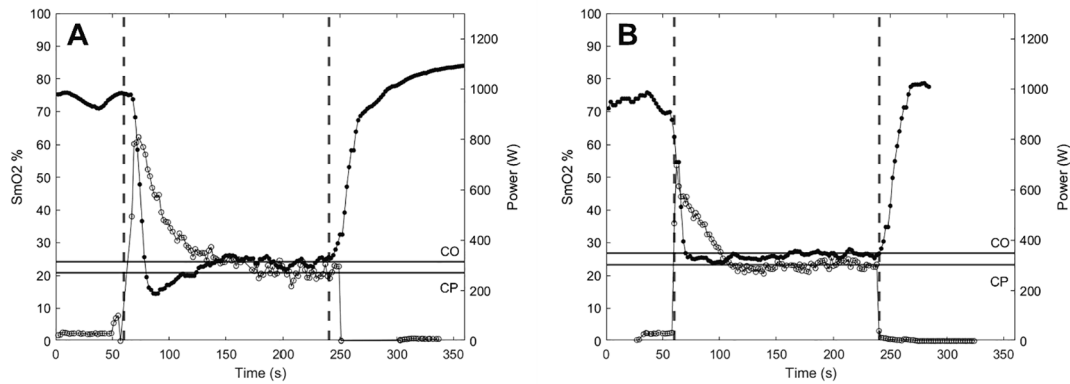
the mathematical model selection to determine CP is seconded by Gaesser and colleagues [18], who concluded that the parameter estimates of five different mathematical models differed significantly. Even when ignoring the aforementioned problems with determining CP, a considerable discrepancy in CP can be found. A study by Smith and Hill [51] showed that a minimum variation in CP parameters of 5% can be assumed – and this is under experimental conditions. Jones and colleagues [25], strong proponents of CP, argue for a minimal “gray area” of “3–5% with careful attention to protocol”. While CP itself may be a powerful predictive tool in various sporting disciplines [1,19,27], the experimental data provided above questions the practicality of CP as a purely output-oriented model. Nonetheless, the relationship between CP and other physiological markers is well documented.

### CP and physiological markers

CP itself is defined as the highest metabolic rate that can be achieved through oxidative means [42]. It has been proposed that CP represents a specific metabolic rate, in the case of Barker and colleagues [1] a critical oxygen consumption ( $VO_2$ ). When assessing CP through the lens of pulmonary gas exchange Poole and colleagues [43] examined the physiological response of steady-state exercise over 24 min of constant load cycling at CP, resulting in a steady-state response in the under-mentioned parameters. In contrast to exercise above CP, which resulted in increases in  $VO_2$ , blood lactate, lactate-pyruvate ratio, and respiratory frequency, as well as decreases in pH and bicarbonate. Furthermore, there appears to be a relationship between CP and the respiratory compensation point (RCP) providing additional physiological insight into the separation of tolerable and intolerable severe intensity exercise [3,10]. Considering the definition of CP provided, it would follow that CP and maximal lactate steady state (MLSS) would reflect one another. However, this is not the case, and the disagreement has been shown in numerous instances [10,44]. The discrepancy has recently been commented on by Jones et al. [25], who acknowledge the difference between CP and MLSS, with CP occurring at a greater power output than MLSS. The authors claim that CP, and not MLSS, represents a maximal metabolic steady state. They argue that maximal metabolic steady-state should be defined by distinct physiological responses rather than arbitrary exercise durations. Accepting this line of argument, CP has a strong foundation of support, beyond the already mentioned relationships to systemic gas exchange data. A study by Chidnok and colleagues [9] shows a clear metabolic threshold between work rates above and below CP. Muscle metabolite levels at work rates above CP result in increases in inorganic phosphate (Pi) and ADP levels accompanied by decreases in creatine phosphate (PCr) and pH, which are all important factors in fatiguing muscles. These findings are seconded by Jones et al. [29]. To further push the point that CP is the highest metabolic rate that can be achieved through oxidative means it was shown that the inspiration of hyperoxic gas resulted in a significant upward shift of CP and increased time to exhaustion, despite maintaining the same overall change in pH and PCr at point of exhaustion [60]. This strongly indicates a relationship between oxygen supply and demand and CP, while maintaining a finite work capacity above CP, or  $W'$ . In the case of  $W'$ , hyperoxic gas resulted in a decrease in  $W'$ ; contradicting the notion of  $W'$  being equivalent to an anaerobic reserve to be discussed later.

### Rectifying the problem: can NIRS help?

The relationship between CP and other physiological threshold markers has been outlined, as have inconsistencies [6,10,44]. These discrepancies are attributable to several factors as established by Jones et al. [25], but some may also be a cause of volatility in a physiological system, as mentioned earlier. Can this dilemma be rectified? As a point of deduction, if performing tasks at CP is reflected by stabilization of varying physiological metrics, it can be assumed that measuring these physiological metrics could give us insight into the attainment of CP,



**Fig. 1.** Two examples are exploratory data collected to assess the relationship between CP and  $SmO_2$ . A) presents data with an  $O_2$  extraction reserve (area below CO) that requires the application of model 2 in order to fit the CO model. B) presents data that does not exhibit this  $O_2$  extraction reserve. Dashed vertical lines represent the start and stop of cycling protocol; 3-min all-out test. Closed circles (●) represent  $SmO_2$ . Open circles (○) represent power output. Horizontal lines represent CO and CP respectively.

and would do this in a much more dynamic fashion than the rigidity of a power meter, for example. However, unlike a power meter the various measurement tools used to assess the physiological data discussed above are either impractical or expensive; or a combination of both. For example, gas exchange data collection requires tedious laboratory equipment, mostly limits data collection to generic exercise, and tags on a hefty price tag. A tool that could potentially bridge this gap between performance and physiology in the CP model is near-infrared spectroscopy (NIRS); a tool growing in availability and popularity [41]. NIRS discerns muscle oxygenation ( $SmO_2$ ) and provides a user with information about oxygen supply and demand at the local level, in real-time [2]. Of the physiological metrics described above, all can be linked to oxidative metabolism and the ability to supply and utilize oxygen. Systemically, the link between oxygen supply and demand is the very basis of perhaps the most widely accepted physiological performance measure: maximally attainable  $VO_2$  ( $VO_{2peak}$ ). As mentioned before the first component of CP is the highest metabolic rate that can be achieved through oxidative means [42]. Therefore, it is a modest assumption that NIRS could provide insight into the CP model from the physiological perspective. Applying phosphorus magnetic resonance spectroscopy (P-MRS) assessing muscle metabolite development [9,29], Vanhatalo and colleagues [60] showed that changes in  $SmO_2$  track changes in PCr, Pi, ADP, and pH in relation to CP. PCr dynamics and  $SmO_2$  have been cross-validated by Ryan and colleagues [45], identifying NIRS as a valid alternative. This is underscored physiologically by the fact that PCr availability and recovery are dependent on oxygen availability [21]; highlighting the importance of oxygen availability for even high-intensity bouts of exercise above CP. The second component of the CP model,  $W'$ , is also acknowledged to be associated with oxidative metabolism [42]. While previously  $W'$  has been described as an anaerobic reserve, scrutiny by scientific study reveals that  $W'$  and CP are in fact two integrated systems that rely on oxygen availability [5,57].

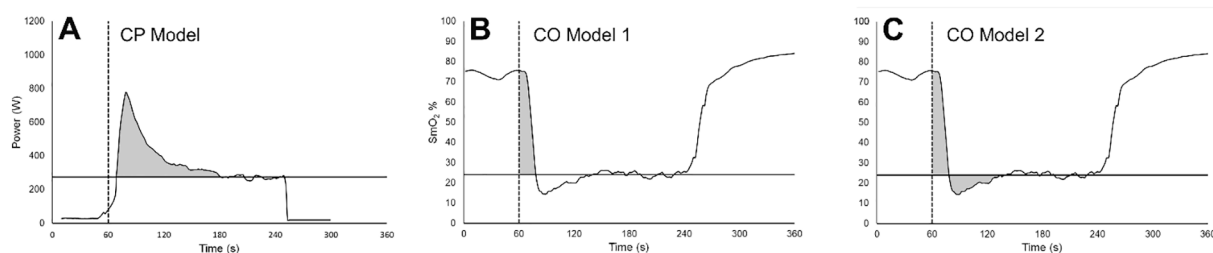
Broxterman and colleagues [5] nicely show that blood flow occlusions result in a drastic change in CP and  $W'$ . In the study, CP in occluded limbs was reduced to less than 0 w, which was to be expected if CP is dependent on oxidative metabolism. However,  $W'$  increased in the occluded limb, which cannot be explained if  $W'$  is an anaerobic reserve constant. This shift in CP and  $W'$  could additionally be seen in the  $SmO_2$  data presented directly in the study.

**Hypothesis**

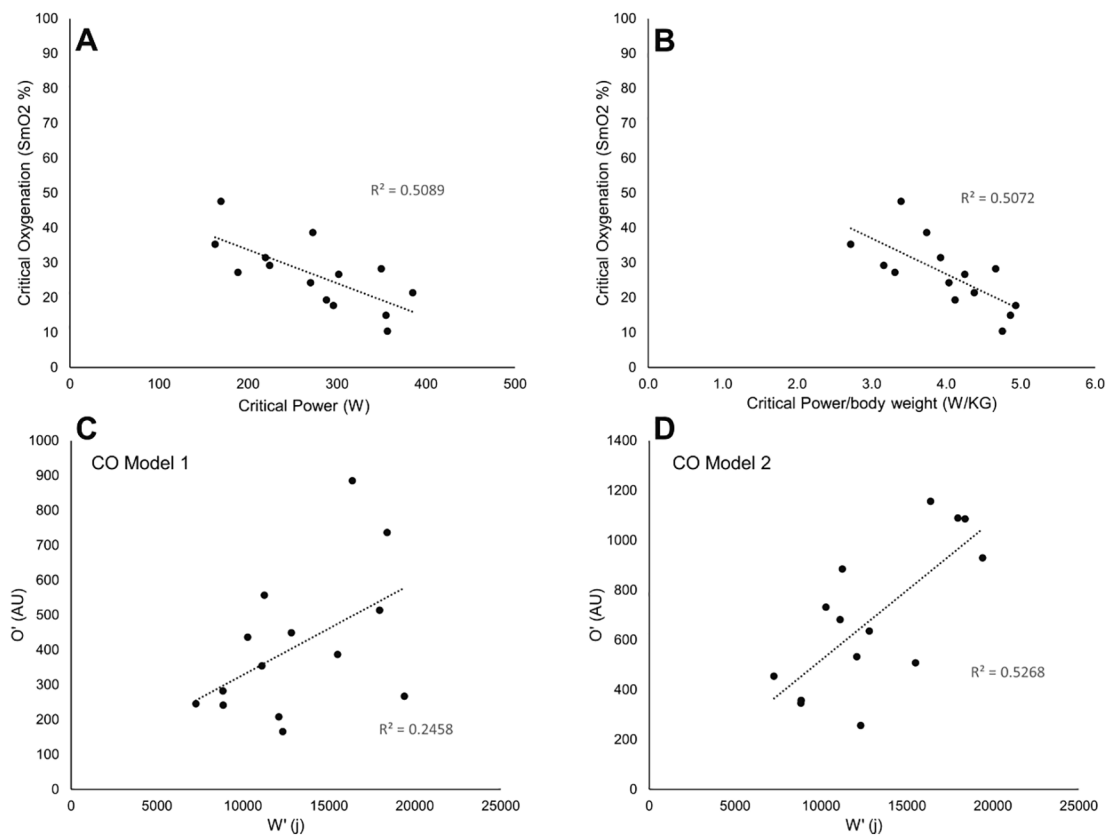
Muscle oxygen availability as well as oxygen supply and demand mechanics, are primary factors in the ability to both maintain exercise steady-state and perform exercise intensity above CP. Therefore, it is plausible to assume that  $SmO_2$  as measured by NIRS could help identify CP and  $W'$ .  $SmO_2$  has been identified by two underlying components, oxygen supply and demand, and oxygen tissue reserve capacity [41]. In both the expected response and in underlying principles, these two components of  $SmO_2$  reflect the two components of the CP model: CP and  $W'$ . One is a steady-state principle, while the other is a reserve capacity, which crystallizes the hypothesis proposed: CP can be evaluated through  $SmO_2$  steady state, while  $W'$  can be assessed through  $SmO_2$  reserve capacity. Thereby, a physiological approximation of the CP model can be generated, with the advantage of direct physiological feedback as opposed to a physical framework. As an homage to the performance model, the physiological model proposed will be termed critical oxygenation (CO) as the counterpart to CP, and then logically follows the term  $O'$  as the counterpart to  $W'$ .

**Exploratory data**

Fourteen participants (nine males and five females; weight:  $67.6 \pm 9.9$  kg; height:  $175.9 \pm 6.9$  cm; age:  $23.9 \pm 5.5$  years [mean  $\pm$  SD])



**Fig. 2.** A) CP model as proposed by Vanhatalo et al. (59) to calculate CP (horizontal line) and  $W'$  (shaded area). B) CO model 1 was proposed to calculate CO (horizontal line) and  $O'$  (shaded area), excluding the area below CO. C) CO model 2 proposed to calculate CO (horizontal line) and  $O'$  (shaded area), including the area below CO.



**Fig. 3.** A) correlation between critical oxygenation (CO) and critical power (CP);  $r(13) = 0.713, p = .004$ . B) correlation between CO and CP as a ratio of bodyweight;  $r(13) = 0.712, p = .004$ . C) correlation between  $O'$  and  $W'$  using CO model 1;  $r(13) = 0.496, p = .071$ . D) correlation between  $O'$  and  $W'$  using CO model 2;  $r(13) = 0.726, p = .003$ .

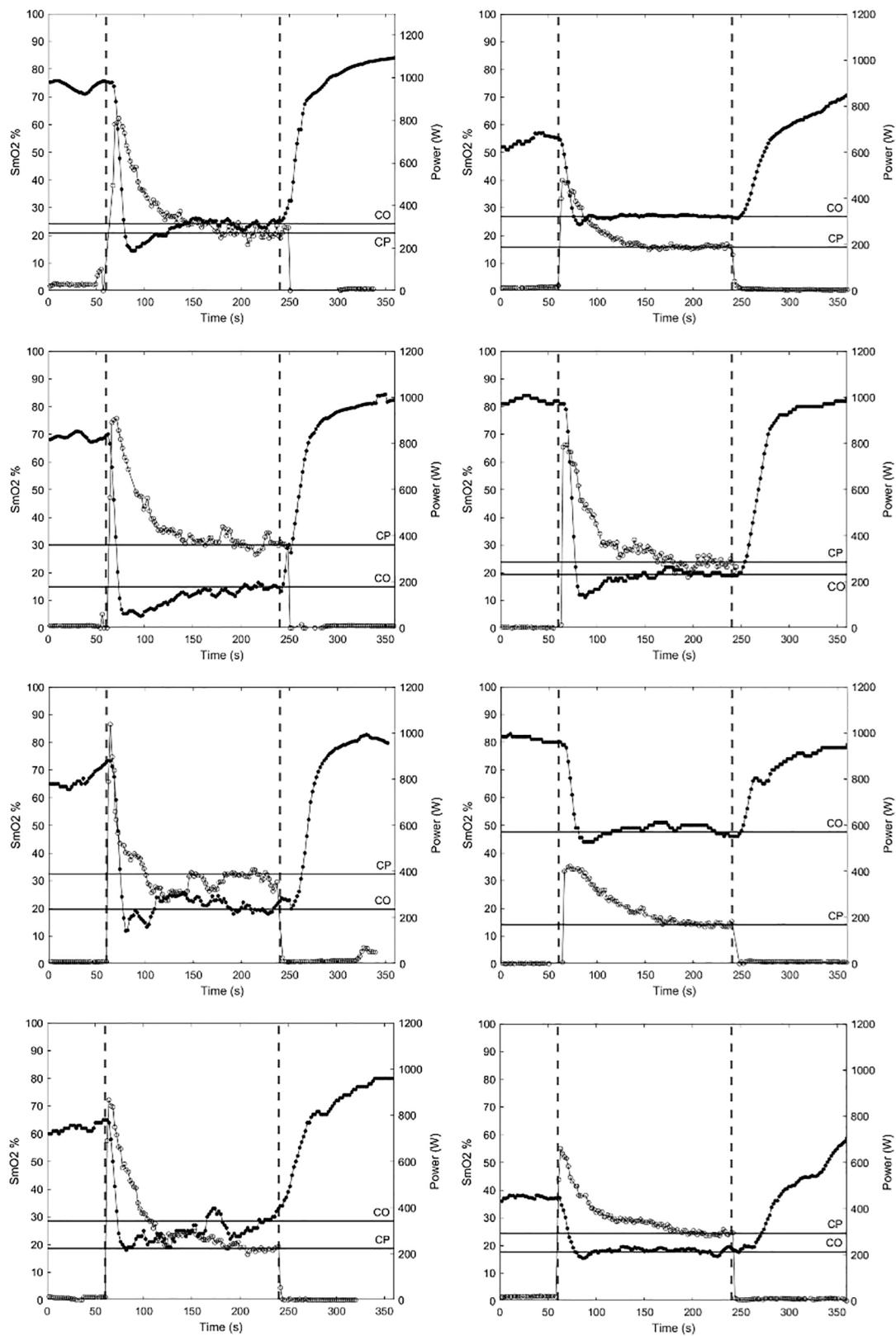
completed a 3-min all-out test [56] to determine CP and  $W'$ . Participants first performed a warm-up at 100 W, followed by 5 min of rest. Participants then started 3 min of unloaded baseline pedaling, followed by the all-out 3-min effort. The procedure followed the example of Vanhatalo and colleagues [56]. Visual analysis of the  $SmO_2$  data quickly identified the similarity between the power profile and the  $SmO_2$  profile (see Fig. 1). This realization yielded a simple analysis and results. CP and  $W'$  were analyzed as prescribed by Vanhatalo et al. [56] and the principle of the path of least resistance demanded that the  $SmO_2$  data be analyzed in the same manner (see Fig. 2). This generated model 1 with the following results, assessed at a significance level set at an alpha of 0.05. For CP and CO, results show a significant negative correlation,  $r(13) = 0.713, p = .004$  (see Fig. 3). However, for  $W'$  and  $O'$ , model 1 was not significant,  $r(13) = 0.496, p = .071$  (see Fig. 3). When taking a closer look at the power curve and the  $SmO_2$  curve, the largest descriptive difference between the two is the fact that  $SmO_2$ , in eight of fourteen cases, dips below the CO threshold, an effect not seen in a power-duration curve (see Figs 1, 4 and 5). This phenomenon appears to be consistent and deserves an explanation. More importantly, it may be a determining factor to a successful model. If  $SmO_2$  is a tissue reserve capacity as proposed, then this additional  $SmO_2$  extraction should be an important characteristic to consider. Therefore, model 2 is proposed, which further integrates the area under the CO threshold (see Fig. 2). Model 2, when adding the area under CO yields a significant positive correlation between  $W'$  and  $O'$ ,  $r(13) = 0.726, p = .003$  (see Fig. 3). Ensuring that the complete  $SmO_2$  extraction is integrated into a model appears to be a vital characteristic for the correlation between  $O'$  value and  $W'$ . When rank-ordering participants by  $W'$  magnitude, the largest values of  $W'$  are generated using model 2 (inclusion of negative dip below CO). These are also the cases, that in the results of model 1 fall out of the correlation as a result of  $W'$  being too small, without the area below CO. It would thus

appear that a large  $W'$  is associated with an  $O_2$  extraction capacity beyond CO, similar to other findings identifying an  $O_2$  extraction reserve [23,24] (see Fig. 4).

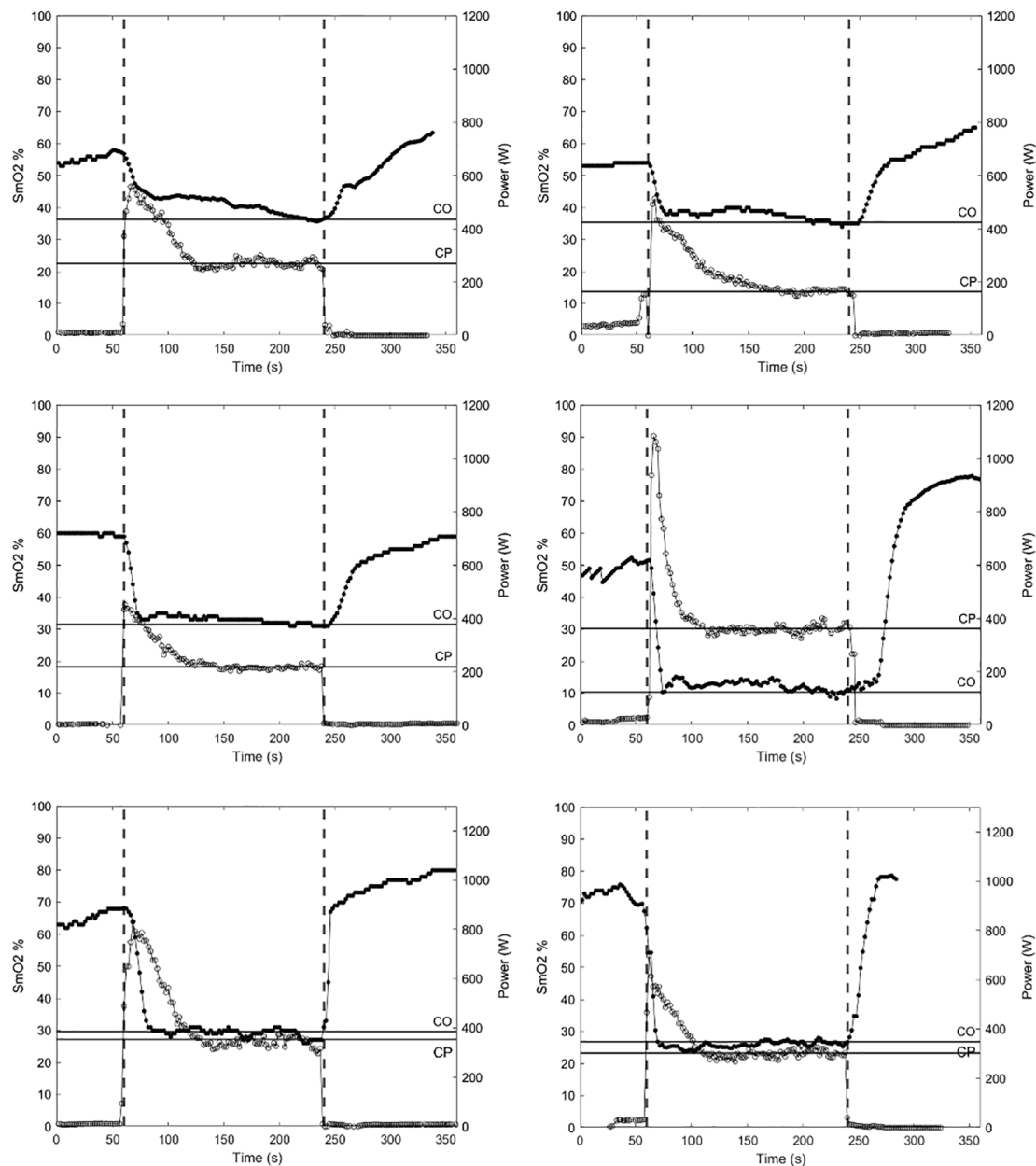
## Discussion

The CP model is a consistent and intriguing model to predict athletic performance. However, the model is not without its limitations. The fact that the model is based exclusively on performance output results, without the scrutiny of the physiological system behind the performance output, can result in rather large and accumulating error sources. If the basic phenomena of the CP model were to be united with a reliable physiological measure, this could greatly increase the predictive power of the model. The hypothesis presented offers such a unification using  $SmO_2$  as a physiological source.

The presented data set is meant to first identify an observational source for the generation of the hypothesis, and second to underscore the physiological framework proposed.  $SmO_2$  illustrates two key components of muscle oxidative capacity: 1) oxygen supply and demand through the trending information of the NIRS signal and 2) oxygen reserve capacity through the magnitude of the possible change in the NIRS signal [41]. The ability to maintain adequate oxygen supply to match oxygen demand is the basis of exercise steady-state theory [17,28]. In the CP model, CP represents “the greatest metabolic rate that results in wholly oxidative energy provision, where wholly oxidative considers the active organism *in toto* and means that energy supply through substrate-level phosphorylation reaches a steady-state” [42]. Considering this definition, it is but a small step to assume that, if  $SmO_2$  accurately reflects oxygen supply and demand, an assessment of changing  $SmO_2$  slopes could represent CP. Specifically,  $SmO_2$  at CP must remain constant to represent the steady-state; as is seen in the



**Fig. 4.** Eight of fourteen examples of the exploratory data collection to assess the relationship between CP and SmO<sub>2</sub>. These eight examples exhibit the O<sub>2</sub> extraction reserve (area below CO) which requires model 2 in order to fit the CO model. The specific selection criteria of this consideration were the predictable pattern of a strong SmO<sub>2</sub> decrease, this decrease exceeding the CO threshold, followed by a recovery to the CO threshold. In several cases this was not an exceedingly large area, nonetheless, advanced the success of model 2. Dashed vertical lines represent the start and stop of cycling protocol; 3-min all-out test. Closed circles (●) represent SmO<sub>2</sub>. Open circles (○) represent power output. Horizontal lines represent CO and CP respectively.



**Fig. 5.** Six of fourteen examples of the exploratory data collection to assess the relationship between CP and SmO<sub>2</sub>. These six examples do not exhibit the O<sub>2</sub> extraction reserve (area below CO) and therefore showed considerable success applying model 1. These examples did not show a decrease in SmO<sub>2</sub> below the CO threshold following the initial decrease in SmO<sub>2</sub> with the onset of exercise. The top right and top left examples show an additional peculiar pattern, in that they both have a much longer decreasing trend in SmO<sub>2</sub> until the CO threshold is truly reached. When looking at the power curve, this could be a result of a non-faithful execution of the 3-min all-out test. Nonetheless, this data does not harm the CO model. Dashed vertical lines represent the start and stop of cycling protocol; 3-min all-out test. Closed circles (●) represent SmO<sub>2</sub>. Open circles (○) represent power output. Horizontal lines represent CO and CP respectively.

exploratory data presented. While this is perhaps somewhat pedestrian, of greater interest may be the fact that, when considering in terms of CO, a greater muscle deoxygenated steady state, is correlated to a higher CP. This prediction again is seconded in other findings, such as in the relationship between CP and VO<sub>2</sub> an often-discussed topic [28,33]; which links CP to VO<sub>2</sub>peak. It is then reasonable to assume that greater CP would result in greater oxygen extraction as depicted by SmO<sub>2</sub>. This assumption is upheld by experimental data showing a correlation between greater deoxygenation and higher VO<sub>2</sub>peak [38,54]. W' is also associated with VO<sub>2</sub>peak, though to a smaller extent than CP [40,53]. Nonetheless, the ability to supply and extract oxygen is a component of W'. While classically W' was referred to as a form of anaerobic reserve, experiments contradict these findings. More accurately stated: "CP and

W should not be considered as separate "aerobic" and "anaerobic" entities but rather as components of an integrated bioenergetic system" [42]. If W' can be manipulated through experiments by changing oxygen supply with hyperoxic gas [40] or blood flow restriction [5], the available oxygen – or lack thereof – determines W'; emphasizing its direct relationship to oxygen availability. Therefore, SmO<sub>2</sub> levels could be seen as helpful in determining changing W'. The fact that this could be tracked in real-time using cost-effective and portable tools greatly enhances its application in the field.

The physiological laboratory is now increasingly mobile, and a CO model derived from portable NIRS sensors would lend itself as a powerful accessory to static and often unwieldy laboratory settings. It would provide the ability simply determine the boundaries of

performance, on par with gold standard evaluations [31], on a daily basis. Additionally, it provides a wide-range of feedback to athletes and coaches, from cadence efficiency when cycling [59] to potential muscular overload following a trail running race [20]. Finally, its ability to not only track systemic changes, but also isolate single working muscles makes it an attractive tool for sporting disciplines that currently lack classical testing methods. For example, such utility would be invaluable in rock climbing, as mentioned in the introduction. When applied to assess  $\text{SmO}_2$  during critical force testing with elite level climbers, NIRS shows substantial predictive power in determining the time to task failure during high effort bouts [13,16]. Moreover, CP has been promoted as a useful tool to understand fatigue in not only health, but also in patient populations [42]. If CO is useful in the understanding of CP, then this would advance its application into the field of Sports Medicine and general practice as well.

It should be recognized that a single NIRS device collects  $\text{SmO}_2$  data from a very specific measurement site that is limited to a single muscle. This would imply that a single muscle measurement could reflect a whole-body activity. While the NIRS and  $\text{VO}_2$  peak comparisons made earlier would imply that this is – at least to a certain degree – plausible, caution is warranted. For example, Wang and colleagues [58] show discrepancies in  $\text{SmO}_2$  breakpoints between the vastus lateralis and the gastrocnemius during incremental cycling exercise. Furthermore, it should be noted that even within single muscles, heterogeneity in terms of NIRS derived oxygenation measures can be seen [39]. The data presented is from a cycling exercise, which is a highly localized quadriceps dominant activity, that can be easily assessed using NIRS. Increasing the complexity of movement, already to something like running raises questions about the straightforward implementation of the model presented.

#### Further directions

The results of this study need to be verified and the findings applied to varying conditions. Numerous questions arise in the discussion of this comparison. Clearly, there is a floor effect for  $\text{SmO}_2$  when considering its relation to  $W'$ . The  $\text{SmO}_2$  integral calculated cannot assume an area below 0% and therefore, perhaps, arbitrary units of deoxy- and oxyhemoglobin may be better suited for this calculation. However, considering that local muscle extraction and  $\text{VO}_2$  peak are closely related [48], the floor effect of  $\text{SmO}_2$  may represent a physiological truism [14,15]. Furthermore, the experimental design forced a mostly negative or falling trend in  $\text{SmO}_2$ , as with performance. In other words, the model proposed does not comment on the reconstitution of  $W'$  and how  $\text{SmO}_2$  would reflect this.  $W'$  does not recover in a linear fashion, as is the case with its depletion [50], and it would be reasonable to assume that the recovery of  $O'$  would be similar. This begs the question if  $O'$  reconstitution can simply be calculated in the reverse function of its depletion; this is unlikely. The final point of interest, for consideration in future experiments, is that  $\text{SmO}_2$  can be altered through training interventions, both acutely and chronically [20,30,37]. This is also true for CP and  $W'$  [32,47]. If the relationship proposed is true, an intervention should show the same predetermined shift in CO and  $O'$  as it does in CP and  $W'$ .

#### Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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#### References

- [1] Barker T, Poole DC, Noble ML, Barstow TJ. Human critical power-oxygen uptake relationship at different pedalling frequencies. *Exp Physiol* 2006;91:621–32. <https://doi.org/10.1113/expphysiol.2005.032789>.
- [2] Barstow TJ. Understanding near infrared spectroscopy and its application to skeletal muscle research. *J Appl Physiol* 2019;126(5):1360–76. <https://doi.org/10.1152/jappphysiol.00166.2018>.
- [3] Bergstrom HC, Housh TJ, Zuniga JM, Traylor DA, Camic CL, Lewis RW, et al. The relationships among critical power determined from a 3-min all-out test, respiratory compensation point, gas exchange threshold, and ventilatory threshold. *Res Q Exerc Sport* 2013;84(2):232–8. <https://doi.org/10.1080/02701367.2013.784723>.
- [4] Bishop D, Jenkins D, Howard A. The critical power function is dependent on the duration of the predictive exercise tests chosen. *Int J Sports Med* 1998;19(02): 125–9. <https://doi.org/10.1055/s-2007-971894>.
- [5] Broxterman RM, Ade CJ, Craig JC, Wilcox SL, Schlup SJ, Barstow TJ. Influence of blood flow occlusion on muscle oxygenation characteristics and the parameters of the power-duration relationship. *J Appl Physiol* 2015;118(7):880–9. <https://doi.org/10.1152/jappphysiol.00875.2014>.
- [6] Broxterman RM, Ade CJ, Craig JC, Wilcox SL, Schlup SJ, Barstow TJ. The relationship between critical speed and the respiratory compensation point: Coincidence or equivalence. *Eur J Sport Sci* 2015;15:631–9. <https://doi.org/10.1080/17461391.2014.966764>.
- [7] Burnley M, Jones AM. Oxygen uptake kinetics as a determinant of sports performance. *Eur J Sport Sci* 2007;7(2):63–79. <https://doi.org/10.1080/17461390701456148>.
- [8] Burnley M, Jones AM. Power-duration relationship: Physiology, fatigue, and the limits of human performance. *Eur J Sport Sci* 2018;18:1–12. <https://doi.org/10.1080/17461391.2016.1249524>.
- [9] Chidnok W, Fulford J, Bailey SJ, Dimenna FJ, Skiba PF, Vanhatalo A, et al. Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the “critical power”. *J Appl Physiol* 2013;115:243–50. <https://doi.org/10.1152/jappphysiol.00334.2013>.
- [10] Dekerle J, Baron B, Dupont L, Vanvelcenaher J, Pelayo P. Maximal lactate steady state, respiratory compensation threshold and critical power. *Eur J Appl Physiol* 2003;89(3):281–8. <https://doi.org/10.1007/s00421-002-0786-y>.
- [11] Dekerle J, Sidney M, Hespel JM, Pelayo P. Validity and reliability of critical speed, critical stroke rate, and anaerobic capacity in relation to front crawl swimming performances. *Int J Sports Med* 2002;23:93–8. <https://doi.org/10.1055/s-2002-20125>.
- [12] Dotan R. Critical power: what is being measured and why? *Eur. J. Appl. Physiol* 2014;114:2677–8. <https://doi.org/10.1007/s00421-014-2961-3>.
- [13] Feldmann A, Erlacher D, Pfister S. Predict Failure: Muscle Oxygen Dynamics In Elite Climbers During Finger Hang Tests. Orlando: ACSM Annual Meeting; 2019.
- [14] Feldmann A, Schmitz R, Erlacher D. NIRS on a functional scale of 0–100%: Establishing practicality of the Moxy Monitor for sport science. Prague: ECSS Annual Meeting; 2019.
- [15] Feldmann A, Schmitz R, Erlacher D. Near-infrared spectroscopy-derived muscle oxygen saturation on a 0% to 100% scale: reliability and validity of the Moxy Monitor. *J Biomed Opt* 2019;24:1. <https://doi.org/10.1117/1.jbo.24.1.115001>.
- [16] Feldmann AM, Erlacher D, Pfister S, Lehmann R. Muscle oxygen dynamics in elite climbers during finger-hang tests at varying intensities. *Sci Rep* 2020;10:3040. <https://doi.org/10.1038/s41598-020-60029-y>.
- [17] Ferretti G, Fagoni N, Taboni A, Bruseghini P, Vinetti G. The physiology of submaximal exercise: The steady state concept. *Respir Physiol Neurobiol* 2017; 246:76–85. <https://doi.org/10.1016/j.resp.2017.08.005>.
- [18] Gaesser GA, Carnevale TJ, Garfinkel A, Walter DO, Womack CJ. Estimation of critical power with nonlinear and linear models. *Med Sci Sports Exerc* 1995;27: 1430–8.
- [19] Giles D, Chidley JB, Taylor N, Torr O, Hadley J, Randall T, Fryer S. The determination of finger-flexor critical force in rock climbers. *Int J Sports Physiol Perform* 14: 972–979, 2019. [10.1123/ijsp.2018-0809](https://doi.org/10.1123/ijsp.2018-0809).
- [20] Giovannelli N, Biasutti L, Salvadego D, Alemayehu HK, Grassi B, Lazzar S. Changes in skeletal muscle oxidative capacity after a trail-running race. *Int J Sports Physiol Perform* 2020;15:278–84. <https://doi.org/10.1123/ijsp.2018-0882>.
- [21] Haseler LJ, Hogan MC, Richardson RS. Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on O<sub>2</sub> availability. *J Appl Physiol* 1999;86(6):2013–8. <https://doi.org/10.1152/jappl.1999.86.6.2013>.
- [22] Hill PA V. The Physiological Basis of Athletic Records. [Online]. *Nature* 116, 1925. <https://www.nature.com/articles/116544a0.pdf> [17 Jun. 2020].
- [23] Iannetta D, Okushima D, Inglis EC, Kondo N, Murias JM, Koga S. Blood flow occlusion-related O<sub>2</sub> extraction “reserve” is present in different muscles of the quadriceps but greater in deeper regions after ramp-incremental test. *J Appl Physiol* 2018;125(2):313–9. <https://doi.org/10.1152/jappphysiol.00154.2018>.
- [24] Inglis EC, Iannetta D, Murias JM, Calaine Inglis E, Iannetta D, Murias JM, et al. The plateau in the NIRS-derived [HHb] signal near the end of a ramp incremental test does not indicate the upper limit of O<sub>2</sub> extraction in the vastus lateralis. *Am J*

- Physiol - Regul Integr Comp Physiol 2017;313:R723–9. <https://doi.org/10.1152/ajpregu.00261.2017>.
- [25] Jones AM, Burnley M, Black MI, Poole DC, Vanhatalo A. The maximal metabolic steady state: redefining the 'gold standard'. *Physiol Rep* 2019;7(10):e14098. <https://doi.org/10.14814/phy2.14098>.
- [27] Jones AM, Vanhatalo A. The 'Critical Power' Concept: Applications to Sports Performance with a Focus on Intermittent High-Intensity Exercise. *Sport Med* 2017;47:65–78. <https://doi.org/10.1007/s40279-017-0688-0>.
- [28] Jones AM, Vanhatalo A, Burnley M, Morton RH, Poole DC. Critical Power: Implications for Determination of VO<sub>2</sub>max and Exercise Tolerance. *Med Sci Sport Exerc* 2010;42:1876–90. <https://doi.org/10.1249/MSS.0b013e3181d9cf7f>.
- [29] Jones AM, Wilkerson DP, DiMenna F, Fulford J, Poole DC. Muscle metabolic responses to exercise above and below the "critical power" assessed using 31 P-MRS. *Am J Physiol Integr Comp Physiol* 2008;294(2):R585–93. <https://doi.org/10.1152/ajpregu.00731.2007>.
- [30] Jones B, Hamilton DK, Cooper CE, Hug F. Muscle Oxygen Changes following Sprint Interval Cycling Training in Elite Field Hockey Players. *PLoS ONE* 2015;10(3):e0120338. <https://doi.org/10.1371/journal.pone.0120338>.
- [31] Keir DA, Fontana FY, Robertson TC, Murias JM, Paterson DH, Kowalchuk JM, et al. Exercise Intensity Thresholds: Identifying the Boundaries of Sustainable Performance. *Med Sci Sports Exerc* 2015;47:1932–40. <https://doi.org/10.1249/MSS.0000000000000613>.
- [32] Kendall KL, Smith AE, Graef JL, Fukuda DH, Moon JR, Beck TW, et al. Effects of four weeks of high-intensity interval training and creatine supplementation on critical power and anaerobic working capacity in college-aged men. *J Strength Cond Res* 2009;23:1663–9. <https://doi.org/10.1519/JSC.0b013e3181b1fd1f>.
- [33] Kolbe T, Dennis SC, Selley E, Noakes TD, Lambert MI. The relationship between critical power and running performance. *J Sports Sci* 1995;13(3):265–9. <https://doi.org/10.1080/02640419508732236>.
- [34] Kranenburg KJ, Smith DJ. Comparison of critical speed determined from track running and treadmill tests in elite runners. *Med Sci Sports Exerc* 1996;28(5):614–8. <https://doi.org/10.1097/00005768-199605000-00013>.
- [35] Mattioni Maturana F, Fontana FY, Pogliaghi S, Passfield L, Murias JM. Critical power: How different protocols and models affect its determination. *J Sci Med Sport* 2018;21:742–7. <https://doi.org/10.1016/j.jsams.2017.11.015>.
- [36] Monod H, Scherrer J. The work capacity of a synergic muscular group. *Ergonomics* 1965;8(3):329–38. <https://doi.org/10.1080/00140136508930810>.
- [37] Patrick Neary J, McKenzie DC, Bhambhani YN. Effects of short-term endurance training on muscle deoxygenation trends using NIRS. *Med Sci Sports Exerc* 2002;34(11):1725–32. <https://doi.org/10.1097/00005768-200211000-00006>.
- [38] Okushima D, Poole DC, Barstow TJ, Rossiter HB, Kondo N, Bowen TS, et al. Greater VO<sub>2</sub>peak is correlated with greater skeletal muscle deoxygenation amplitude and hemoglobin concentration within individual muscles during ramp-incremental cycle exercise. *Physiol Rep* 2016;4:1–12. <https://doi.org/10.14814/phy2.13065>.
- [39] Okushima D, Poole DC, Rossiter HB, Barstow TJ, Kondo N, Ohmae E, et al. Muscle deoxygenation in the quadriceps during ramp incremental cycling: Deep vs. superficial heterogeneity. *J Appl Physiol* 2015;119:1313–9. <https://doi.org/10.1152/jappphysiol.00574.2015>.
- [40] Parker Simpson L, Jones A, Skiba P, Vanhatalo A, Wilkerson D. Influence of hypoxia on the power-duration relationship during high-intensity exercise. *Int J Sports Med* 2014;36(02):113–9. <https://doi.org/10.1055/s-0000002810.1055/s-005-2842810.1055/s-0034-1389943>.
- [41] Perrey S, Ferrari M. Muscle Oximetry in Sports Science: A Systematic Review. *Sport Med* 2018;48:597–616. <https://doi.org/10.1007/s40279-017-0820-1>.
- [42] Poole DC, Burnley M, Vanhatalo A, Rossiter HB, Jones AM. Critical Power: An Important Fatigue Threshold in Exercise Physiology. *Med Sci Sports Exerc* 2016;48:2320–34. <https://doi.org/10.1249/MSS.0000000000000939>.
- [43] Poole DC, Ward SA, Gardner GW, Whipp BJ. Metabolic and respiratory profile of the upper limit for prolonged exercise in man. *Ergonomics* 1988;31(9):1265–79. <https://doi.org/10.1080/00140138808966766>.
- [44] Pringle J, Jones A. Maximal lactate steady state, critical power and EMG during cycling. *Eur J Appl Physiol* 2002;88(3):214–26. <https://doi.org/10.1007/s00421-002-0703-4>.
- [45] Ryan TE, Southern WM, Reynolds MA, McCully KK. A cross-validation of near-infrared spectroscopy measurements of skeletal muscle oxidative capacity with phosphorus magnetic resonance spectroscopy. *J Appl Physiol* 2013;115(12):1757–66. <https://doi.org/10.1152/jappphysiol.00835.2013>.
- [46] Salam H, Marcora SM, Hopker JG. The effect of mental fatigue on critical power during cycling exercise. *Eur J Appl Physiol* 2018;118(1):85–92. <https://doi.org/10.1007/s00421-017-3747-1>.
- [47] Sawyer BJ, Stokes DG, Womack CJ, Morton RH, Weltman A, Gaesser GA. Strength training increases endurance time to exhaustion during high-intensity exercise despite no change in critical power. *J Strength Cond Res* 2014;28:601–9. <https://doi.org/10.1519/JSC.0b013e31829e113b>.
- [48] Skattebo Ø, Calbet JAL, Rud B, Capelli C, Hallén J. Contribution of oxygen extraction fraction to maximal oxygen uptake in healthy young men. *Acta Physiol* 2020;230. <https://doi.org/10.1111/apha.13486>.
- [49] Skiba PF, Chidnok W, Vanhatalo A, Jones AM. Modeling the expenditure and reconstitution of work capacity above critical power. *Med Sci Sports Exerc* 2012;44:1526–32. <https://doi.org/10.1249/MSS.0b013e3182517a80>.
- [50] Skiba PF, Jackman S, Clarke D, Vanhatalo A, Jones AM. Effect of work and recovery durations on W' reconstitution during intermittent exercise. *Med Sci Sports Exerc* 2014;46:1433–40. <https://doi.org/10.1249/MSS.0000000000000226>.
- [51] Smith JC, Hill DW. Stability of parameter estimates derived from the power/time relationship. *Can J Appl Physiol* 1993;18(1):43–7. <https://doi.org/10.1139/h93-005>.
- [52] Townsend NE, Nichols DS, Skiba PF, Racinais S, Périard JD. Prediction of critical power and W' in hypoxia: Application to work-balance modelling. *Front Physiol* 2017;8:180. <https://doi.org/10.3389/fphys.2017.00180>.
- [53] Valli G, Cogo A, Passino C, Bonardi D, Morici G, Fasano V, et al. Exercise intolerance at high altitude (5050m): Critical power and W'. *Respir Physiol Neurobiol* 2011;177:333–41. <https://doi.org/10.1016/j.resp.2011.05.014>.
- [54] Van Der Vaart H, Murgatroyd SR, Rossiter HB, Chen C, Casaburi R, Porszasz J. Selecting constant work rates for endurance testing in COPD: The role of the power-duration relationship. *COPD J Chronic Obstr Pulm Dis* 2014;11:267–76. <https://doi.org/10.3109/15412555.2013.840572>.
- [55] Vandewalle H, Vautier JF, Kachouri M, Lechevalier JM, Monod H. Work-exhaustion time relationships and the critical power concept [Online]. *J Sports Med. Phys. Fitness* 37: 89–102, 1997. [https://www.researchgate.net/publication/13979872\\_Work-exhaustion\\_time\\_relationships\\_and\\_the\\_critical\\_power\\_concept\\_A\\_critical\\_review](https://www.researchgate.net/publication/13979872_Work-exhaustion_time_relationships_and_the_critical_power_concept_A_critical_review) [15 Jun. 2020].
- [56] Vanhatalo A, Doust JH, Burnley M. Determination of critical power using a 3-min all-out cycling test. *Med Sci Sports Exerc* 2007;39:548–55. <https://doi.org/10.1249/mss.0b013e31802dd3e6>.
- [57] Vanhatalo A, Fulford J, DiMenna FJ, Jones AM. Influence of hyperoxia on muscle metabolic responses and the power-duration relationship during severe-intensity exercise in humans: a 31P magnetic resonance spectroscopy study. *Exp Physiol* 2010;95:528–40. <https://doi.org/10.1113/expphysiol.2009.050500>.
- [58] Wang B, Xu G, Tian Q, Sun J, Sun B, Zhang L, et al. Differences between the Vastus Lateralis and Gastrocnemius Lateralis in the Assessment Ability of Breakpoints of Muscle Oxygenation for Aerobic Capacity Indices During an Incremental Cycling Exercise. [Online]. *J Sports Sci Med* 2012;11:606–13.
- [59] Zorgati H, Collomp K, Boone J, Guimard A, Buttelli O, Mucci P, et al. Effect of pedaling cadence on muscle oxygenation during high-intensity cycling until exhaustion: a comparison between untrained subjects and triathletes. *Eur J Appl Physiol* 2015;115:2681–9. <https://doi.org/10.1007/s00421-015-3235-4>.