# Insights into Anaerobic Power Reserve On Relationships with Exercise Tolerance, Work Above Critical Power, and Accumulated Oxygen Deficit in Endurance-Trained Male Cyclists: A Pilot Study

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**Abstract**

The anaerobic power reserve (APR) model seeks to account for the heterogeneity in athletes' anaerobic characteristics. However, its relationship with exercise tolerance across various durations and with anaerobic markers remains unclear. Therefore, we investigate the relationship between APR, exercise tolerance, work above critical power (W'), and maximal accumulated oxygen deficit (MAOD) in male cyclists. We further analyzed this relationship replacing maximal aerobic power (MAP) with critical power (CP) as the lower boundary of the power reserve, defining a so-called maximal power reserve (MPR). After preliminary tests, 19 endurance-trained male cyclists performed five trials to exhaustion (Tlim) at 130%, 115%, 100%, 85%, and 80% of maximal aerobic power (MAP) and Wingate test. APR and MPR correlated with all Tlim values ($r > 0.511$, $p < 0.03$), except at 80%MAP. After fixing CP or MAP, only correlations with supramaximal Tlim remained significant ($r > 0.703$, $p < 0.002$). When PPO was fixed, only MPR correlated with Tlim at 130% and 115%MAP ($r > 0.508$, $p = 0.037$). Both APR and MPR were associated with MAOD and W′ ($r = 0.480–0.542$, $p = 0.045$), but only MPR remained significantly related to MAOD after adjusting for lower boundary ($r = 0.488$, $p = 0.040$). Our findings showed that in endurance-trained male cyclists, both power reserves relate to exercise tolerance, however their influence decreases for longer efforts. MPR exhibited a stronger link to anaerobic capacity compared to APR. The association between exercise tolerance and APR/MPR appears largely driven by peak power output (PPO), rather than the choice of lower boundary.

**Keywords:**

Cycling, Modelling, Power-Duration, Critical Power, Anaerobic Power Reserve

## *Introduction*

In the last years, the anaerobic power reserve (APR) has emerged as a potential tool to prescribe training within the severe and extreme intensity domains as well as a predictive model for sprint performance (Sandford et al. 2021). The APR is generally defined as the difference between the maximal instantaneous power output (PPO) and the last power output attained during an incremental test, often referred to as maximal aerobic power (MAP), disregarding any significant anaerobic contribution above critical power (Sandford et al. 2021). This model seeks to account for the heterogeneity in athletes' anaerobic characteristics and potentially leads to a more homogenous acute training response (Sandford et al. 2021). At the same relative intensity, prescribed as MAP percentage, athletes with higher PPO will engage a smaller fraction of their APR. This could lead to reduced metabolic stress and consequently delayed exhaustion compared to athletes with lower PPO (Buchheit and Laursen 2013). According to this theory, a previous study reported a strong relationship between running time to exhaustion within the severe intensity domain and anaerobic speed reserve (ASR), suggesting this parameter can be a potential proxy for anaerobic characteristics (Blondel N et al. 2001). Similarly, the APR model in cycling has been shown to predict sprint performance across efforts lasting between 5 and 180 seconds in both recreational (Weyand et al. 2006) and well-trained male road cyclists (Sanders and Heijboer 2019). However, the aforementioned approach relies on mathematical models, which can only be applied following several unconventional maximal efforts (Weyand et al. 2006; Sanders and Heijboer 2019). Consequently, some researchers have explored whether a simpler method, such as prescribing exercise intensity based on the simple difference between PPO and MAP (i.e., APR), could account for the variability observed during exhaustion tests based on MAP (Barnett et al. 1996; Blondel N et al. 2001; Buchheit and Laursen 2013; Boullosa and Abreu 2014). Barnett et al. (1996) prescribed a cycling work rate based on individual APR in male athletes, trying to reduce the variability in time to exhaustion. However, this approach did not yield the expected results, suggesting instead that APR might represent an oversimplification of the metabolic and physiological mechanisms involved in anaerobic pathways. Moreover, recent studies on male team players have reported

only minimal, and never statistically significant, reductions in intersubject variability in physiological responses and exercise tolerance outcomes during interval training when using APR-based intensity prescriptions, compared to traditional methods (Julio et al. 2020; Collison et al. 2021; Bok et al. 2023). While the APR construct holds theoretical validity, its lack of physiological validation may stem from the use of MAP as its lower boundary. MAP, in addition to being protocol-dependent, also requires substantial anaerobic contributions to be sustained; thereby, this overlap complicates the isolation of a 'reserve' that clearly defines the boundary between sustainable and unsustainable power domains. Therefore, taking these factors together, some doubts arise about the actual effectiveness of APR as a prescription method to uniform exhaustion times and physiological responses during high-intensity exercise.

Currently, the most accurate method to achieve uniform exercise tolerance among athletes is to consider the individual power-duration relationship (Dotan 2022). This relationship enables the determination of the critical power (CP) and the finite amount of work (W') that can be performed above it (Jones et al. 2019; Drake et al. 2024). Bearing in mind the analogy between the APR model and the power-duration relationship, the former presents a potentially simpler alternative to uniform exercise stimulus compared with the more complex and demanding procedures required for CP and W' determination (Sanders and Heijboer 2019; Leo et al. 2022). Although the APR appears to be a useful and appealing tool for exercise prescription, there is no evidence that it correlates with exercise tolerance during constant cycling within the severe-intensity domain based on MAP in male cyclists. Additionally, no research investigated the possible correlation between APR and well-established measures of anaerobic capacity like, the maximal accumulated oxygen deficit (Medbo et al. 1988; Medbø and Welde 2022) or W' in male cyclists. Therefore, in order to assess the construct validity of the APR model, it is essential to investigate its correlation with established measures of anaerobic characteristics.

Hence, the aim of this pilot study was to investigate, in endurance-trained male cyclists: (i) the relationship between cycling exercise tolerance during severe-intensity constant work rates MAP-based and APR;(ii)

the association between APR and W';(iii) the relationship between APR and maximal accumulated oxygen deficit.

Furthermore, given the inherent protocol dependency of MAP and the high reliance on anaerobic pathways to meet energy demands at this intensity, we further analyzed this relationship using CP instead of MAP as the lower boundary of the power reserve, defining a so-called maximal power reserve (MPR).

## ***Materials and Methods***

### *Participants*

A *priori* sample size calculation was performed through G*Power considering a positive relationship between APR and time to exhaustion as the main outcome (Blondel N et al. 2001). A bivariate correlation with a significance level set at 0.05, two tails, statistical power of 0.80, and *r* equal to 0.6 were applied. This computation generated a desired sample size of at least 17 participants. Therefore, 19 endurance-trained male cyclists were recruited (mean ± SD; age: 34 ± 9 years; height: 1.79 ± 0.07 m; mass: 74 ± 10 kg). Inclusion criteria required that participants had trained consistently for at least 6 hours per week in the last year. Any muscular or orthopedic injuries and health problems in the previous six months were considered exclusion criteria. To standardize testing conditions, participants were asked to refrain from food and caffeine at least 3-4 hours prior to each test, visit the laboratory consistently at the same time of the day (±1), avoid strenuous exercise for at least 36 hours beforehand, and keep their dietary habits unchanged throughout all the intervention period. During all visits, participants were allowed to drink water *ad libitum*. All participants were provided with a thorough explanation of the study's aim and procedures before providing written informed consent. The research adhered to the Declaration of Helsinki, with prior approval from the Ethics Committee of Università degli Studi di Milano (University of Milan. N. 2020/49).

### *Design*

Participants visited the laboratory seven times over four weeks. The sessions included familiarization sessions, a ramp incremental test to determine maximal oxygen uptake ($VO_2$max) and MAP, a Wingate test, and five severe-intensity trials to exhaustion (Tlim) to estimate CP and W'. Additionally, submaximal constant work rates (CWR) below the respiratory compensation point were conducted before each Tlim session to estimate accumulated oxygen deficit (AOD). An electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, Netherlands) was used, adjusted to individual anthropometry and positioning habits. All tests, except for familiarization and the initial ramp test, were randomized across visits.

*Pre-testing and familiarization*

During their first visit, participants underwent anthropometric assessments and familiarization with all procedures. Body composition was estimated using a bioelectrical impedance device (Tanita BC-420 MA, Tokyo, Japan) following the manufacturer's recommendations. Height was measured using a stadiometer (Seca 217, Vogel & Halke, Hamburg, Germany). Then, participants completed a torque-velocity profile test and a Wingate test. These tests served two purposes: 1) to familiarize participants with sprinting on a stationary ergometer and 2) to determine the optimal cadence (through the ergometer linear factor) for achieving PPO during a subsequent Wingate test (Driss and Vandewalle 2013). After thirty minutes participants performed a familiarization ramp test and a Tlim lasting ~5 minutes. A further familiarization Tlim lasting ~15 minutes was conducted at the end of the second visit.

*Ramp-incremental Test*

Participants completed a ramp incremental test to determine $VO_2$max and the MAP following the step-ramp-step protocol proposed by Iannetta et al. (2020). The test began with a 2-minute warm-up at 20 W, followed by a first square wave increase at 100 W lasting 6 minutes, within the moderate domain (MOD), followed by 4 minutes at 50 W. Then, an increase of ~1 W every 5 seconds (25 W every 2 minutes) started. The slope of this ramp test was chosen to be comparable to that used by one of the pivotal manuscripts on

the relationship between APR and Tlim, and thus be able to compare the results (Blondel N et al. 2001). Participants were instructed to maintain a self-selected cadence between 70 and 90 rpm throughout the test. The test ended when participants were not able to maintain their cadence above 70 rpm despite strong verbal encouragement. $VO_2$max was determined as the highest 30-second rolling average and was considered achieved when participants met at least two of the three of the following criteria: heart rate exceeding 95% of predicted maximal heart rate (210 – (0.65 × age)); respiratory exchange ratio exceeding 1.1; blood lactate exceeding 8.0 mmol·$L^{-1}$ (Poole and Jones 2017). MAP was considered the final power output achieved during the ramp test. After thirty minutes a heavy intensity constant work rate (HVY), corresponding to 75% of the second ventilatory threshold, lasting 15 minutes was performed. Mean oxygen uptake ($VO_2$) of the last 2 minutes of MOD and HVY were used to left-shift the power output corresponding to gas exchange threshold (GET) and respiratory compensation point (RCP) according to the recommended procedure (Iannetta et al. 2020).

*Wingate Test*

The Wingate test began with a 5-minute warm-up cycling between 50 and 100 W. Participants then performed three 4-5 second preparation sprints followed by a 5-minute passive rest period. Afterward, participants pedaled (unloaded) at 65 rpm for 10 seconds to allow a flying start. Upon completion of these 10 seconds, the linear ergometer mode was applied, and the participants were instructed to accelerate maximally and perform a 30-second sprint while maintaining a seated position (Driss and Vandewalle 2013). The resistance applied during the test was determined based on the torque-velocity test conducted during the familiarization session. Participants were blinded to power output and elapsed time during the test to minimize pacing strategies. PPO was considered the highest 1-second power output.

*Severe intensity trials to exhaustion*

Five Tlim were conducted to estimate CP and W'. These trials employed a range of pre-determined power outputs corresponding to 80% (Tlim80), 85% (Tlim85), 100% (Tlim100), 115% (Tlim115), and 130%

(Tlim130) of MAP. The specific power outputs were chosen to elicit a range of time-to-exhaustion durations between 2 and 20 minutes as recently suggested (Mattioni Maturana et al. 2018). Each Tlim commenced with a 4-minute warm-up at 50 W, followed by a square wave increase to the pre-determined power output. CP and W' were modeled using the following equations (Hugh Morton 1996):

1) Three parameters hyperbolic model: $t = W'/(PO-CP) + W'/(CP-P_{max})$
2) Two parameters hyperbolic model: $t = W'/(PO-CP)$

Where $t$ is time to exhaustion, $P_{max}$ is the theoretical maximal instantaneous power, $W'$ is work (Joules) and $PO$ is power output. Both sets of estimated values were retained for statistical analysis.

*Accumulated oxygen deficit*

Thirty minutes before each Tlim, participants performed two CWRs, 10 minutes long separated by a 10-minute rest period. The intensity of each CWR spanned from 30% of MAP to the RCP (i.e. 30, 35, 40, 45, 50, 55, 60, 65, 70, 75% of MAP). The mean $VO_2$ from the final two minutes of each CWR test was used to establish the relationship between PO and $VO_2$. Participants were instructed to pedal at their preferred cadence (80 ± 5 rpm) and maintain the same cadence throughout the sessions. The theoretical oxygen demand for each Tlim was estimated by multiplying the extrapolated $VO_2$ (from the PO-$VO_2$ relationship) by the time to exhaustion for that specific trial. The AOD was then calculated for each trial by subtracting the measured $VO_2$ consumed during the trial from the $VO_2$ demand area (Medbo et al. 1988; Medbø and Welde 2022). Finally, the highest AOD (MAOD) obtained from these trials was retained for further correlation analysis. The contribution of anaerobic metabolism was calculated as the ratio between the AOD and the $VO_2$ demand and expressed in percentage (Medbø and Welde 2022).

*Anaerobic Power Reserve and Maximal Power Reserve*

APR has been calculated as PPO minus MAP. Additionally, considering that MAP, and *ipso facto* the APR, are protocol dependent, the MPR, that is PPO minus CP, has been calculated. We specifically opted for the CP derived from the 3-parameter model ($CP_{3\text{-}hyp}$) due to its lower values compared to the 2-parameter

188 hyperbolic model ($CP_{2\text{-}hyp}$). This selection reflects the greater sustainability of exercise intensities based on
189 the $CP_{3\text{-}hyp}$, potentially resulting in a lower contribution from anaerobic pathways and consequently
190 generating an MPR with less overlapping energy systems contributions (Mattioni Maturana et al. 2018).

191 *Data collection and analysis*

192 Pulmonary gas exchange was continuously monitored breath-by-breath during all testing sessions using a
193 wearable metabolic cart (Cosmed K5, Rome, Italy) and a mask (Hans Rudolph, INC. Shawnee, KS, USA)
194 with a dead space of 30 mL. Before the test, the ergospirometer was calibrated with a 3-L syringe (Cosmed,
195 Rome, Italy), with ambient air and a standardized gas mixture (20.0% O2 and 4.0% CO2). The laboratory
196 temperature was maintained at 20 ± 1 °C and relative humidity at 53 ± 4 %. Raw gas exchange was analyzed
197 using Matlab R2023b (The Mathworks Inc., Natick, MA, USA). A custom-built script was used to remove
198 outliers (i.e. errant breath or coughing). Subsequently, data were interpolated to a one-second time base and
199 filtered using a low-pass forward-backward Butterworth filter (3rd order, 0.04 Hz cut-off). The $VO_2$ at
200 GET during the ramp test was identified by the first non-linear increase in both ventilation (VE) and carbon
201 dioxide output that deviated from the initial linear rise in $VO_2$ (Beaver et al. 1986). The $VO_2$ at RCP was
202 identified by the subsequent, distinct rise in ventilation and an initial decrease in the end-tidal partial
203 pressure of carbon dioxide following the isocapnic buffering phase (Keir et al. 2018). Afterward, employing
204 VO2 of the last two minutes of MOD and HVY, the power outputs at GET and RCP were retrospectively
205 adjusted. This adjustment was performed using a MATLAB custom-built script following the procedures
206 outlined by Iannetta et al. (2020) to account for the $VO_2$ mean response time during the ramp test (Iannetta
207 et al. 2019; Iannetta et al. 2020).

208 Blood lactate concentration ([La]) was measured from earlobe samples (5 μL) using a Lactate Pro 2
209 (Arkray, Kyoto, Japan). Following the ramp incremental test to exhaustion, samples were obtained at 2 and
210 3 minutes post-exhaustion. Similarly, for the severe-intensity trials, blood was drawn 2, 3, and 4 minutes
211 after exhaustion and the peak value was retained.

*Statistical analysis*

Data normality was assessed using the Shapiro-Wilk test. Descriptive statistics are presented as mean ± standard deviation (SD). Pearson's correlation and 95% confidence intervals (CIs) were used to examine the relationship between all measured parameters with APR and MPR, and a correlation coefficient of >.1, >.3, >.5, and >.7 was considered small, moderate, large, and very large (Hopkins et al. 2009). Moreover, the standard error of estimation (SEE) has been calculated to assess the Tlim prediction accuracy of APR/MPR. Since a high APR/MPR can be obtained with a high PPO, as well as a low MAP/CP, or both, a partial correlation analysis was employed to better understand the effects of APR/MPR on exercise tolerance and other variables. The approach is therefore divided into two categories: 1) evaluating the relationship between APR/MPR and the other parameters controlling for PPO (thus evaluating the effect of APR/MPR as MAP/CP decreases); 2) evaluating the relationship between APR/MPR and the other parameters, controlling for MAP/CP (thus evaluating the effect of APR/MPR as PPO increases). A lack of correlation between APR/MPR and both CP and MAP prevented us from directly performing the latter. However, the significant relationship between PPO and both CP and MAP enabled us to employ an indirect approach to assess the influence of APR/MPR. In particular, by performing partial correlations between PPO with other variables while fixing either CP or MAP we could isolate the contribution of APR as PPO increases. To compare the physiological and perceptual responses attained during all Tlim an analysis of variance for repeated measures was applied, and in case of significant differences between intensities, pairwise comparisons were calculated with the Bonferroni correction. La, VE, AOD, and RPE were not normally distributed, thus the Friedman test was performed. Significance was set at 0.05 (two-tailed). All analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp. Armonk, NY, USA).

## ***Results***

Participants' characteristics are presented in **Table 1.**

236 ***Table 1***

237 The mean duration, PO, and physiological responses attained during each Tlim are shown in **Table 2** and
238 **Figure 1**. There were no significant differences between $VO_2$max reached during the incremental test and
239 $VO_2$peak attained during Tlims ($p > 0.49$). However, a significantly higher $VO_2$peak has been observed
240 during Tlim100 compared to Tlim115 and Tlim130 ($p = 0.01$ and $p = 0.009$, respectively). 59% of
241 participants achieved the MAOD during Tlim115, 26% during Tlim100, and 16% during Tlim130. Indeed,
242 ANOVA revealed a significant difference in AOD among Tlims, and pairwise comparisons with Bonferroni
243 correction indicated that AOD during Tlim115 was significantly higher than during Tlim85 and Tlim80 ($p$
244 = 0.007 and $p = 0.045$, respectively) (**Table 2** and **Figure 1**).

245

246 ***Table 2***

247 *** Figure 1***

248 APR and MPR showed a large to very large significant positive correlation with all Tlims except Tlim80,
249 as shown in **Figures 1** and **2**. Notwithstanding when APR/MPR were normalized to body mass, only the
250 relationships with (supra)maximal Tlims remained significant (Table 3). Similarly, after controlling for CP
251 or MAP, these relationships remain significant for Tlim130, Tlim115, and Tlim100 while becoming non-
252 significant for Tlim85 and Tlim80. After controlling for PPO, only the relationship between MPR, Tlim130
253 and Tlim115 remained statistically significant (**Table 3)**. Similarly, PPO showed a large to very large
254 significant positive correlation with all Tlim (**Figures 1** and **2**). When expressing the data normalized to
255 body mass, only the relationships with Tlim100 remained significant (Table 3).

256 *** Insert Figures 2 and 3 and Tables 3 and 4***

257 APR displayed a correlation with MAOD ($r = 0.502$, $p = 0.029$), and with W' derived by the 2-parameter
258 hyperbolic model ($W'_{2\text{-hyp}}$) ($r = 0.464$, $p = 0.045$), but not with W' derived by the 3-parameter model ($W'_{3\text{-}}$

259 $_{hyp}$). MPR shows significant associations with MAOD ($r$ = 0.542, $p$ = 0.016) and W'$_{2\text{-hyp}}$ ($r$ = 0.527, $p$ =
260 0.020) but not with W'$_{3\text{-hyp}}$. PPO was significantly related to both MAOD ($r$ = 0.560, $p$ = 0.013) and W'$_{2\text{-}}$
261 $_{hyp}$ ($r$ = 0.603, $p$ = 0.006) but it was not correlated with W'$_{3\text{-hyp}}$.

262 The partial correlation approach revealed that by fixing PPO the relationships between APR, MPR, and
263 MAOD became non-significant. While the previously non-significant correlations between APR and both
264 W'$_{2\text{-hyp}}$ and W'$_{3\text{-hyp}}$ became significant with negative slopes. In contrast, the association between MPR and
265 both W'$_{2\text{-hyp}}$ and W'$_{3\text{-hyp}}$ remains non-significant. When partial correlations were employed to account for
266 CP, relationships between PPO and MAOD remained significant, and a similar but not significant trend
267 was observed when MAP was fixed. Finally, when CP or MAP was fixed, the relationships between PPO
268 and W'$_{2\text{-hyp}}$ became non-significant (**Table 4**).

269 *****Table 4*****

270 ***DISCUSSION***

271 *APR/MPR and exercise tolerance*

272 The present pilot study investigated the relationship between APR/MPR and exercise tolerance across
273 various exercise intensities in endurance-trained male cyclists. We further explored potential associations
274 between APR/MPR, anaerobic capacity and work above critical power. Our initial findings revealed
275 significant correlations between both APR/MPR (in absolute values) and all Tlims but Tlim80. These
276 results align with an early study that observed a large correlation between running time to exhaustion at
277 intensities spanning from 90% to 140% of maximal aerobic speed with both ASR and maximal speed
278 reserve (the running counterpart of our MPR) (Blondel N et al. 2001). Since a high APR/MPR can be
279 achieved through either a high PPO or a low MAP/CP, a partial correlation approach was employed to
280 better understand their independent contributions to exercise tolerance. Fixing PPO, only the relationship
281 between MPR, Tlim130 and Tlim115 remained statistically significant, and all the relationships between

APR and Tlims became non-significant. Fixing MAP/CP instead revealed that a higher PPO, and *ipso facto***,** higher APR/MPR were positively associated with greater exercise tolerance for Tlim100, Tlim115, and Tlim130. Moreover, positive relationships between Tlim130 and Tlim115 emerged with their corresponding AOD, indicating a greater reliance on anaerobic pathways at these intensities (see also *supplementary materials*). Collectively, these findings support the rationale for APR/MPR as a metric to assess anaerobic characteristics in endurance male cyclists and the strong correlation between APR/MPR and supramaximal Tlims aligns with previous research demonstrating the high accuracy of the APR in predicting sprint and supramaximal efforts in healthy individuals (Weyand et al. 2006) and professional male road cyclists (Sanders and Heijboer 2019). Additionally, these results reinforce the notion that MAP, an aerobic marker, is insufficient to explain exercise tolerance when anaerobic contributions exceed approximately 20% of the total energy demand, as seen during supra-maximal Tlims (Blondel N et al. 2001). In contrast, incorporating APR/MPR, indirect markers of anaerobic characteristics, offers a more comprehensive explanation for the observed variance in exercise tolerance. Regarding Tlim85 and Tlim80, a trend toward significant relationships highlighted how fixing MAP/CP, having a higher APR/MPR likely leads to a greater exercise tolerance also during longer trials. This aligns with a recent study in well-trained track cyclists, in which a very large positive correlation between PPO and various maximal mean powers up to 20 minutes was observed (Ferguson et al. 2023). Nevertheless, the magnitude of these correlations decreases during sub-maximal Tlims, likely due to their lower relative anaerobic contribution (<10%).

Noteworthy, the positive association between MPR and Tlim80 became negative after controlling for PPO using partial correlations, although it did not reach statistical significance. This seemingly counterintuitive finding could be explained by the influence of this approach. By fixing PPO, athletes with higher CP would inherently have lower MPR. Consequently, the observed negative correlation might indirectly reflect the well-established principle that, for longer exercise durations, athletes with higher CP will demonstrate greater exercise tolerance when intensity is based on MAP. However, it has also been observed that for these submaximal Tlims, the body mass plays a substantial role in driving such correlation, given that when

APR/MPR or PPO are normalized to body mass, the relationships become non-significant. Therefore, the correlation between APR/MPR and submaximal Tlim must be interpreted with caution.

Finally, despite these power reserves seeming to account for a substantial proportion of the variance in supramaximal Tlims, the wide confidence intervals and elevated SEE caution against relying solely on these metrics for individualized exercise prescription. Therefore, using this parameter as a yardstick for prescribing Tlims may not completely resolve the high variance classically associated with Tlims.

Pearson correlations between PPO and Tlim were also significant for all Tlims. Moreover, although it would have been more predictable that the correlation strength would decrease between PPO and Tlim as the power of Tlim decreases, we observed the strongest correlation at Tlim100. While the reason remains unclear, we propose that task familiarity may underpin this finding. Cyclists more often perform high-intensity efforts near 100% MAP, which typically elicit exhaustion within ~5 minutes (matching Tlim100 durations). In contrast, supramaximal efforts (Tlim115 and Tlim130) are less familiar, both physiologically and psychologically, potentially introducing variability in exercise tolerance.

*APR/MPR, anaerobic capacity and work above critical power*

MPR displayed a stronger relation with $W'_{2hyp}$, compared to APR. This discrepancy might be due to the neglect of anaerobic contribution by the APR approach within the power range between CP and the MAP achievement during the ramp test. In fact, this zone can be considered as a sort of anaerobic work capacity. Since exhaustion during the ramp test theoretically occurs only when the finite work capacity is depleted (beyond CP), using MAP to define the lower boundary of APR might be a less rigorous approach. Somewhat counterintuitively, when PPO's influence was fixed, the previous positive significant correlation between APR and $W'_{2\text{-}hyp}$ became negative. This can be explained by the positive correlation between $W'_{2\text{-}hyp}$ and MAP (see *Online Resource*). Indeed, fixing PPO, athletes with higher MAP will have lower APR and simultaneously greater $W'_{2\text{-}hyp}$ and $W'_{3\text{-}hyp}$. In addition, when controlling for MAP/CP, higher APR/MPR values in our endurance male cyclist sample were not associated with higher $W'_{2\text{-}hyp}$. Therefore,

these results confirm that the work capacity above CP and APR/MPR are two distinct parameters and highlight caution when interpreting APR/MPR as a measure of work that can be performed within the severe intensity domain. This result may also explain why no significant reduction in heterogeneity of exercise tolerance and physiological responses was observed in previous studies, involving male participants, when intensity is prescribed according to APR (Barnett et al. 1996; Julio et al. 2020; Collison et al. 2021; Bok et al. 2023).

Despite both APR and MPR displayed significant positive correlations with MAOD, the partial correlations revealed that these results were primarily driven by the influence of PPO. This suggests that when PPO is fixed, variations in CP or MAP do not significantly influence MAOD. Conversely, when CP, but not MAP, is fixed, a higher PPO is likely associated with a greater MAOD. This discrepancy could again be attributed to the mixed metabolic contributions at MAP.

Finally, considering these results collectively, the divergence between MPR and APR probably stems from the latter's reliance on MAP as a lower boundary. MAP is not only inherently protocol-dependent but also represents an intensity that cannot be sustained beyond ~5 minutes due to its substantial anaerobic pathway involvement (Blondel et al. 2001). These dual limitations conflate sustainable and unsustainable domains in the "grey zone" between CP and MAP. In contrast, MPR circumvents this ambiguity by anchoring its lower boundary to CP, a well-established threshold for sustainable metabolic steady state, thereby providing a more stable and physiologically coherent model for distinguishing power domains.

The present study has some limitations. First, we enrolled exclusively male cyclists. However, considering the well-documented sex-related differences in anaerobic capacity (Weber and Schneider 2000; Hunter 2016; Ansdell et al. 2020), muscle fiber type composition (Vanhatalo et al. 2016; McDougall et al. 2023), and neuromuscular recruitment patterns (Hunter 2016; Ansdell et al. 2020), it is plausible that female athletes may display different responses. Although males have a greater W', several studies also show greater variability in male W' than in female W', even when LBM-normalized CP and its variability is not different between sexes (Caswell et al. 2024; Bourgois et al. 2023; McDougall et al. 2023). Therefore,

further studies investigating sex differences surrounding the APR/MPR are needed. Second, a limited number of familiarization sessions. Due to the high physical demands placed on participants, who were required to complete seven laboratory sessions (all until exhaustion), we limited the number of Tlim familiarization sessions to two. These sessions were designed to approximate the conditions of the actual trials but may not have fully replicated them. This could explain the unexpected finding of a stronger correlation between PPO and Tlim100 compared to Tlim130.

Finally, this study focused only on endurance-trained cyclists; therefore, our results cannot be broadened to other cycling disciplines involving distinct physiological demands characterized by a higher reliance on anaerobic metabolism and explosive power output, such as track cycling.

*Conclusions*

This pilot investigation revealed that, in endurance-trained male cyclists, both APR and MPR are closely related to exercise tolerance at various intensities, particularly for shorter efforts (under 5 minutes). However, for longer durations, their role decreases. MPR displayed a stronger link with both mechanical and physiological measures of anaerobic capacity compared to APR. This suggests a potential bias when using MAP as the lower boundary for APR calculations. Furthermore, the association between exercise tolerance and APR/MPR seems mainly driven by the role of the PPO rather than the lower boundary used to define the power reserve. This study, however, included a single sex, thus limiting the generalizability of results and highlighting the need for future research including female cyclists.

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**Competing interests**

The authors declare there are no competing interests.

379 **Author contributions**

380 SDG, EF, and MP conceived the research and designed the experiment; LF and MP helped to design the
381 experiment. SD recruited participants and performed the experiment and performed the statistical analysis;
382 SDG, LF, MP, GG, PR, and EF interpreted the results, revised and edited the manuscript; SDG prepared
383 tables and figures; SDG drafted the manuscript; SDG, LF, MP, GG, PR, EF approved the final version of
384 the manuscript.

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387 **Data availability statement**

388 The data supporting this study's findings are available from the corresponding author, [MP], upon
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390

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**Table 1 . Participants' Characteristics**

| **Anthropometry** | | **Power-Duration Relationships** | |
|---|---|---|---|
| Age (years) | 34 ± 9 | $CP_{3\text{-}hyp}$ (W) | 249 ± 36 |
| Height (m) | 179 ± 7 | $W'_{3\text{-}hyp}$ (kJ) | 41 ± 15 |
| Mass (kg) | 74 ± 10 | $P_{max}$ (W) | 1177 ± 1124 |
| Lean mass (kg) | 63 ± 8 | $CP_{2\text{-}hyp}$ (W) | 257 ± 34 |
| Body Fat (%) | 13 ± 2 | $W'_{2\text{-}hyp}$ (kJ) | 29 ± 9 |
| **Incremental Ramp Test** | | **Wingate Test** | |
| $VO_2$peak ($ml \cdot kg^{-1} \cdot min^{-1}$) | 61 ± 8 | PPO (W) | 1064 ± 190 |
| MAP (W) | 350 ± 46 | **Anaerobic/Maximal Power Reserve** | |
| GET (W) | 176 ± 32 | APR (W) | 714 ± 171 |
| RCP (W) | 243 ± 35 | MPR (W) | 815 ± 176 |

***Notes*** $VO_2$max, maximal oxygen uptake; MAP, maximal aerobic power; GET, gas exchange threshold; RCP, respiratory compensation point; $CP_{3\text{-}hyp}$, Critical power derived from the 3 parameters hyperbolic model; $W'_{3\text{-}hyp}$, anaerobic work capacity derived from the 3 parameters hyperbolic model; $P_{max}$, the theoretical maximal istantaneous power output estimated by the 3 parameters model; $CP_{2\text{-}hyp}$, Critical power derived from the 2 parameters hyperbolic model; $W'_{2\text{-}hyp}$, anaerobic work capacity derived from the 2 parameters hyperbolic model; PPO, peak power output; APR, anaerobic power reserve; MPR, maximal power reserve.

**Table 2. Descriptive data of the Time to exhaustion trials**

| | Tlim130 | Tlim115 | Tlim100 | Tlim85 | Tlim80 |
|---|---|---|---|---|---|
| Power Output (W) | 455 ± 60 | 403 ± 53 | 350 ± 46 | 298 ± 39 | 280 ± 37 |
| Durations (s) | 107 ± 26 | 152 ± 31 | 289 ± 80 | 717 ± 17 | 1126 ± 275 |
| $VO_2$peak (ml□kg$^{-1}$□min$^{-1}$) | 58 ± 8 | 59 ± 8 | 64 ± 9 | 61 ± 7 | 61 ± 8 |
| Hrpeak (bpm) | 175 ± 11 | 178 ± 11 | 181 ± 11 | 183 ± 11 | 183 ± 12 |
| [La] (mmol□L$^{-1}$) | 11.8 ± 4.1 | 12.6 ± 3.9 | 12.8 ± 3.7 | 10.7 ± 4.0 | 8.9 ± 3.5 |
| VEpeak (L□min$^{-1}$) | 157 ± 29 | 168 ± 25 | 173 ± 20 | 166 ± 23 | 156 ± 23 |
| AOD (L) | 4.4 ± 1.3 | 4.9 ± 1.5 | 4.6 ± 1.9 | 3.9 ± 1.8 | 3.6 ± 1.3 |
| RPE (a.u.) | 19.2 ± 1.1 | 19.3 ± 0.9 | 19.3 ± 0.6 | 19.3 ± 0.8 | 19.4 ± 0.6 |

***Notes*** Data are expressed as mean ± standard deviation. $VO_2$peak, peak oxygen uptake; Hrpeak, peak heart rate; [La], peak blood lactate concentration; VEpeak, peak ventilation; AOD, accumulated oxygen deficit; RPE, rate of perceived exertion; Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP

**Table 3. Pearson correlations between Anaerobic/Maximal Power Reserve normalized to body mass and Exercise Tolerance**

| | APR (W□kg$^{-1}$) | | MPR (W□kg$^{-1}$) | | PPO (W□kg$^{-1}$) | |
|---|---|---|---|---|---|---|
| | *r (95% CI)* | *p* | *r (95% CI)* | *p* | *r (95% CI)* | *p* |
| Tlim130 | 0.597 (0.232 to 0.836) | 0.009 | 0.584 (0.207 to 0.827) | 0.011 | 0.356 (-0.111 to 0.721) | 0.147 |
| Tlim115 | 0.606 (0.174 to 0.858) | 0.008 | 0.602 (0.146 to 0.853) | 0.008 | 0.407 (-0.112 to 0.757) | 0.093 |
| Tlim100 | 0.622 (0.078 to 0.849) | 0.006 | 0.643 (0.047 to 0.860) | 0.004 | 0.594 (-0.081 to 0.844) | 0.009 |
| Tlim85 | 0.276 (-0.196 to 0.677) | 0.257 | 0.264 (-0.248 to 0.676) | 0.290 | 0.264 (-0.323 to 0.705) | 0.290 |
| Tlim80 | 0.284 (-0.366 to 0.681) | 0.253 | 0.269 (-0.415 to 0.665) | 0.280 | 0.333 (-0.369 to 0.715) | 0.177 |

***Notes*** Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve; PPO, peak power output.

**Table 4. Partial Correlations Between Anaerobic/Maximal Power Reserve, Exercise Tolerance, Anaerobic Capacity and Work Above Critical Power**

| | Partial Correlation | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | APR (Controlling for PPO) | | MPR (Controlling for PPO) | | PPO (Controlling for CP) | | PPO (controlling for MAP) | |
| | *r (95% CI)* | *p* | *r (95% CI)* | *p* | *r (95% CI)* | *p* | *r (95% CI)* | *p* |
| Tlim130 | 0.467 (-0.192 to 0.853) | 0.059 | 0.606 (0.055 to 0.884) | 0.010 | 0.746 (0.542 to 0.901) | <0.001 | 0.703 (0.406 to 0.903) | 0.002 |
| Tlim115 | 0.363 (-0.241 to 0.759) | 0.152 | 0.508(-0.021 to 0.811) | 0.037 | 0.757 (0.594 to 0.888) | <0.001 | 0.728 (0.505 to 0.873) | 0.001 |
| Tlim100 | -0.253 (-0.637 to 0.197) | 0.328 | -0.151 (-0.519 to 0.276) | 0.564 | 0.729 (0.522 to 0.916) | 0.001 | 0.792 (0.610 to 0.930) | <0.001 |
| Tlim85 | -0.255 (-0.716 to 0.293) | 0.323 | -0.261 (-0.765 to 0.346) | 0.312 | 0.348 (-0.113 to 0.656) | 0.157 | 0.451 (0.041 to 0.779) | 0.070 |
| Tlim80 | -0.333 (-0.684 to 0.026) | 0.192 | -0.435 (-0.768 to -0.139) | 0.081 | 0.279 (-0.273 to 0.647) | 0.263 | 0.428 (-0.277 to 0.774) | 0.086 |
| MAOD | -0.222 (-0.616 to 0.218) | 0.376 | -0.143 (-0.608 to 0.383) | 0.934 | 0.488 (-0.033 to 0.791) | 0.040 | 0.428 (-0.270 to 0.880) | 0.076 |
| $W'_{3\text{-hyp}}$ | -0.531 (-0.770 to 0.147) | 0.023 | -0.335 (-0.671 to 0.052) | 0.314 | 0.148 (-0.386 to 0.534) | 0.559 | -0.016 (-0.548 to 0.426) | 0.949 |
| $W'_{2\text{-hyp}}$ | -0.506 (-0.860 to 0.108) | 0.032 | -0.269 (-0.695 to 0.141) | 0.100 | 0.430 (-0.006 to 0.749) | 0.070 | 0.358 (-0.062 to 0.690) | 0.145 |

***Notes*** Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP; MAOD, maximal accumulated oxygen deficit; $W'_{3\text{-hyp}}$, work above crtitical powerderived from the 3 parameters hyperbolic model; $W'_{2\text{-hyp}}$, work above critical powerderived from the 2 parameters hyperbolic model; APR, anaerobic power reserve; MPR, maximal power reserve; CP, Critical power derived from the 3 parameters hyperbolic model; PPO, peak power output.

## Figure Captions

**Fig. 1** Physiological and perceptual responses during the time to exhaustion trials across intensities

***Notes*** Tlim130, Time to exhaustion (Tlim) at 130% of maximal aerobic power (MAP); Tlim115, Tlim at 115% of MAP; Tlim100, Tlim at 100% of MAP; Tlim85, Tlim at 85% of MAP; Tlim80, Tlim at 80% of MAP; $VO_2$, oxygen uptake; [La], blood lactate concentration; VE, ventilation; RPE, rate of perceived exertion.

**Fig.2** Relationships between anaerobic/maximal power reserve and time to exhaustion at 130%,115%, and 100% of maximal aerobic power.

***Notes*** Tlim130, Time to exhaustion (Tlim) at 130% of maximal aerobic power (MAP); Tlim115, Tlim at 115% of MAP; Tlim100, Tlim at 100% of MAP; Tlim85, Tlim at 85% of MAP; Tlim80, Tlim at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve. PPO, peak power output; SEE values represent the estimated standard error associated with the time estimate using APR/MPR/PPO as the independent variable

***Fig 3*** Relationships between anaerobic/maximal power reserve and time to exhaustion at 85 and 80% of maximal aerobic power.

***Notes*** Tlim85, Time to exhaustion at 85% of maximal aerobic power (MAP); Tlim80, Time to exhaustion at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve. PPO, peak power output; SEE values represent the estimated standard error associated with the time estimate using APR/MPR/PPO as the independent variable.

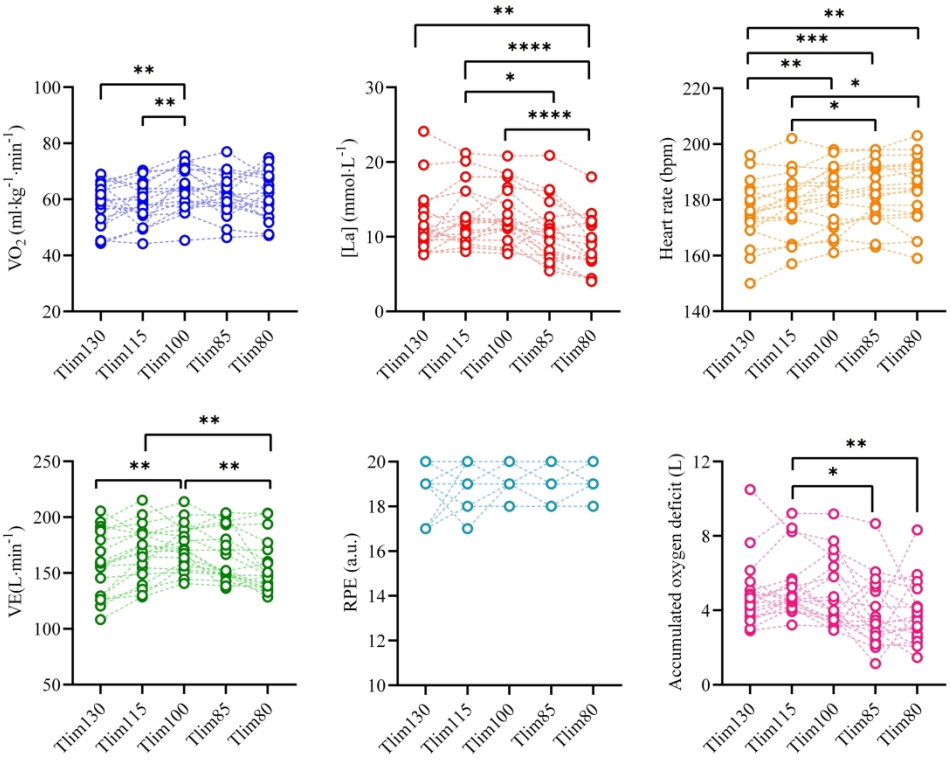


Figure 1

260x211mm (300 x 300 DPI)

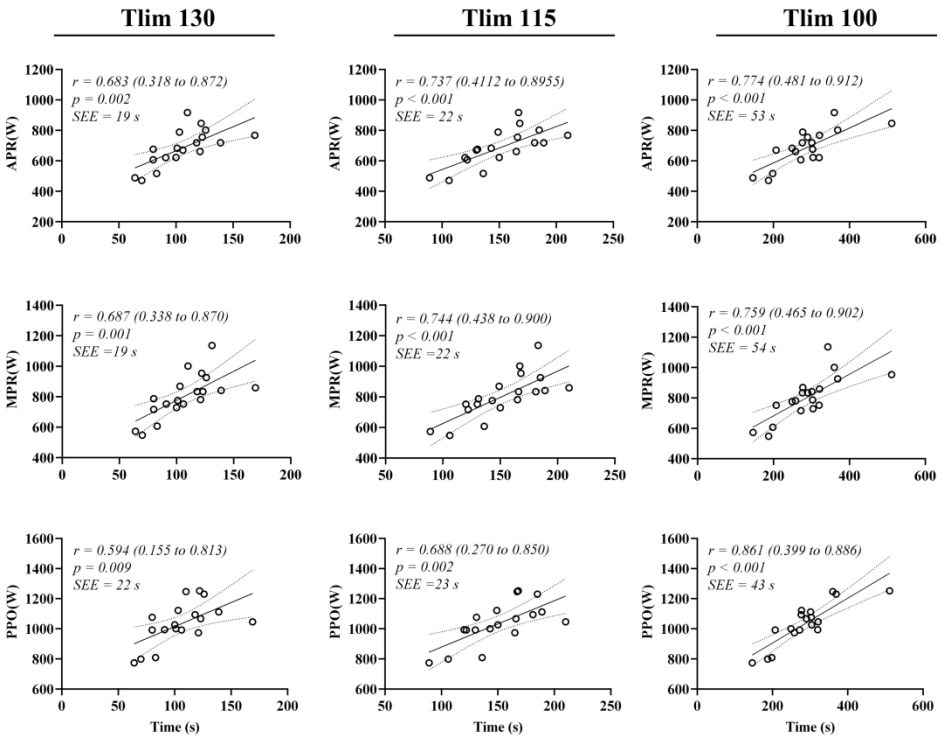


Figure 2

282x216mm (300 x 300 DPI)

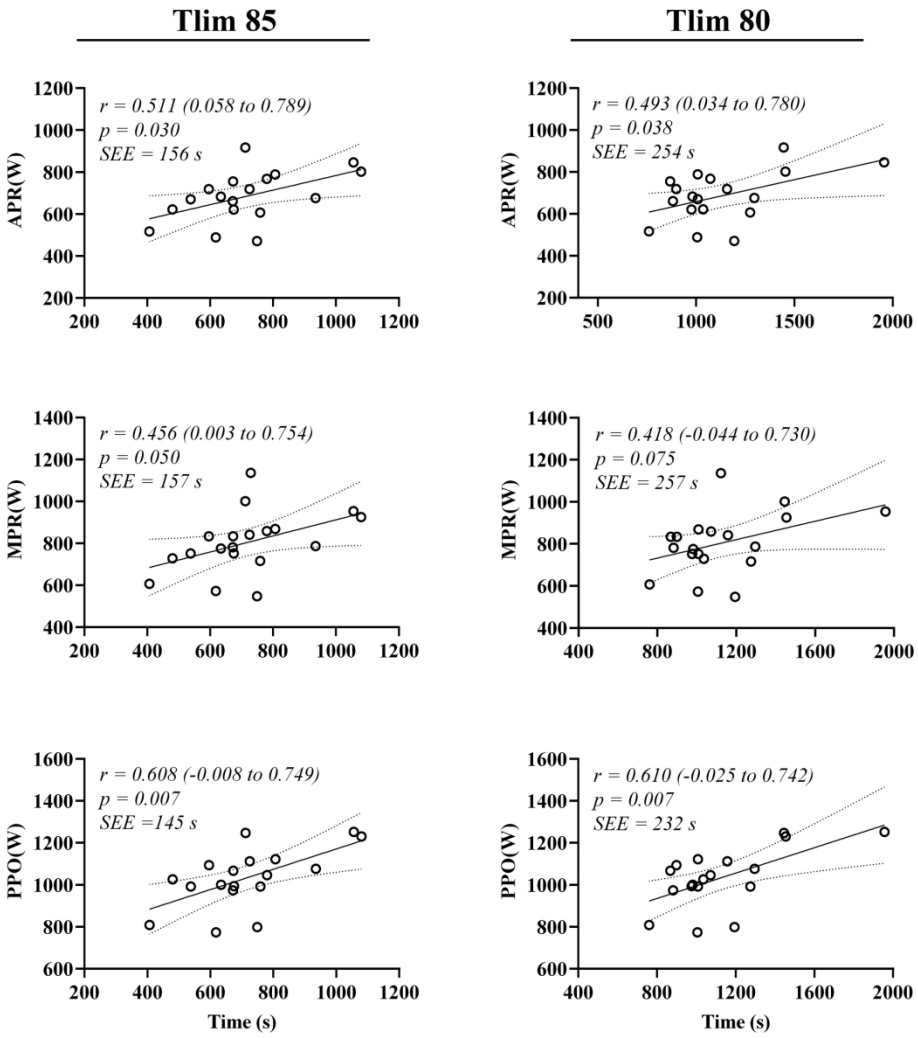


Figure 3

191x207mm (300 x 300 DPI)