

Beyond $\dot{V}O_{2\max}$ sex-stratified contributions of maximal glycolytic flux to metabolic threshold power in trained cyclists

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1 Introduction

The flux-based framework proposed by Mader offers a mechanistic explanation for threshold genesis: metabolic thresholds emerge from the interaction between maximal oxidative and maximal glycolytic flux, such that athletes with similar $\dot{V}O_{2\max}$ may nonetheless differ substantially in threshold power output because of differences in glycolytic capacity (Mader and Heck 1986; Wackerhage et al. 2022). A practical estimate of maximal glycolytic flux is the peak lactate accumulation rate (vLa_{peak}), derived from the increase in blood lactate following a short all-

out sprint. Recent work in trained cyclists suggests that higher vLa_{peak} is associated with lower power output at multiple lactate- and gas-exchange-derived thresholds, independent of $\dot{V}O_{2\max}$. To examine whether this relationship is similarly evident in male and female cyclists, we performed sex-stratified analyses in a heterogeneous cohort of trained athletes. Rather than focusing primarily on between-sex comparisons, this approach was chosen to describe the contribution of vLa_{peak} to threshold power within each sex separately and thereby assess the consistency of the flux–threshold relationship across sexes.



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2 Material and Methods

Fifty trained cyclists (30 male, 20 female; >3 years cycling experience, >2 sessions per week) completed three laboratory procedures on separate visits: a ramp test with verification phase to determine $\dot{V}O_{2\max}$ and peak power output; a 15-s isokinetic all-out sprint (130 rpm) followed by repeated capillary blood sampling over 9 min to determine peak lactate accumulation (ΔLa) and derived vLa_{peak} ($= \Delta La / 15 \text{ s}$); and a 3-min incremental step test with simultaneous capillary lactate and breath-by-breath gas-exchange measurements. From the step test, power output was determined at the minimal lactate equivalent (P_{MLE}), fixed blood lactate concentrations of 2 and 4 mmol·L⁻¹ ($P_{2\text{mmol}}$, $P_{4\text{mmol}}$), individual lactate thresholds at minimum lactate + 0.5 and +1.5 mmol·L⁻¹ ($P_{\text{min}+0.5}$, $P_{\text{min}+1.5}$), ventilatory thresholds 1 and 2 (P_{VT1} , P_{VT2}), and maximal fat oxidation (P_{FatMax}).

Sex-stratified partial Pearson correlations between vLa_{peak} and each threshold, controlling for $\dot{V}O_{2\max}$, were computed separately in males and females. Sex-stratified general linear models were fitted with $\dot{V}O_{2\max}$ as the sole predictor (M_0) and with vLa_{peak} subsequently added (M_1); the incremental model contribution was quantified as ΔR^2 and tested by partial F-test. Additionally, thresholds were expressed relative to fat-free mass (W·kg FFM⁻¹) and as a percentage of ramp peak power output (%PPO) to assess whether absolute sex differences in threshold power persisted after normalization.

3 Results

Males and females did not differ in $\dot{V}O_{2\max}$ normalized to fat-free mass (63.2 ± 8.6 vs. $61.8 \pm 7.2 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg FFM}^{-1}$, $p = 0.57$), indicating comparable oxidative capacity when expressed relative to metabolically active tissue. Males showed higher absolute vLa_{peak} (0.45 ± 0.11 vs. $0.38 \pm 0.08 \text{ mmol} \cdot \text{L}^{-1} \cdot \text{s}^{-1}$, $p =$

0.02 , $d = 0.69$) and higher absolute threshold power across all outcomes ($d = 1.80\text{--}2.30$). These differences were markedly attenuated after normalization to fat-free mass (e.g., $d \leq 0.07$ for P_{VT1} and P_{VT2}), indicating that much of the sex difference in absolute threshold power was attributable to differences in fat-free mass.

In males, partial correlations between vLa_{peak} and threshold power (controlling for $\dot{V}O_{2\max}$) were significant and moderate-to-strong across all outcomes ($r = -0.44$ to -0.73 , all $p \leq 0.017$), including P_{VT1} ($r = -0.59$), P_{VT2} ($r = -0.69$), and P_{FatMax} ($r = -0.61$). Sex-stratified GLMs confirmed that adding vLa_{peak} to $\dot{V}O_{2\max}$ significantly improved model fit for all eight outcomes ($\Delta R^2 = 0.109\text{--}0.287$, all $p \leq 0.017$), with the largest incremental contributions observed for $P_{2\text{mmol}}$ ($\Delta R^2 = 0.287$) and P_{FatMax} ($\Delta R^2 = 0.241$).

In females, partial correlations were significant for P_{MLE} , $P_{\text{min}+0.5}$, $P_{\text{min}+1.5}$, $P_{4\text{mmol}}$, and P_{FatMax} ($r = -0.41$ to -0.65 , $p \leq 0.041$) but not for P_{VT1} ($r = -0.35$, $p = 0.136$) or P_{VT2} ($r = -0.16$, $p = 0.517$). Correspondingly, vLa_{peak} significantly improved GLM fit for P_{MLE} , $P_{\text{min}+0.5}$, $P_{\text{min}+1.5}$, $P_{4\text{mmol}}$, and P_{FatMax} ($\Delta R^2 = 0.107\text{--}0.383$, $p \leq 0.041$), but yielded non-significant improvements for $P_{2\text{mmol}}$, P_{VT1} , and P_{VT2} . The absence of significant associations for ventilatory thresholds in females coincided with higher baseline R^2 values for models including $\dot{V}O_{2\max}$ alone ($R^2(M_0) = 0.64\text{--}0.74$ for $P_{\text{VT1}}/P_{\text{VT2}}$ in females vs. $0.45\text{--}0.59$ in males), suggesting less additional variance for vLa_{peak} to explain in these outcomes.

4 Discussion

The association between maximal glycolytic flux and metabolic threshold power was evident in both sexes, but differed in consistency and outcome-specific pattern. In males, vLa_{peak} contributed substantially to threshold prediction beyond $\dot{V}O_{2\max}$ across all

threshold concepts, including ventilatory thresholds and P_{FatMax} , which is consistent with the prediction of flux-based models that glycolytic capacity modulates threshold behavior in addition to oxidative capacity (Mader & Heck, 1986; Wackerhage et al., 2022). In females, the contribution of vL_{apeak} was more selective, being apparent for lactate-derived thresholds and P_{FatMax} but not for ventilatory thresholds. This pattern does not necessarily indicate fundamentally different threshold physiology between sexes but rather suggests that the incremental explanatory value of vL_{apeak} may be lower or less consistent for some outcomes in female cyclists.

Absolute sex differences in threshold power were substantially reduced when expressed relative to fat-free mass, indicating that these differences are largely attributable to body composition rather than to oxidative or glycolytic characteristics per se. Together, these findings suggest that sprint-derived

vL_{apeak} provides meaningful physiological information beyond $\dot{V}O_{2\text{max}}$ for metabolic profiling in trained cyclists, although its incremental contribution appears stronger and more consistent in males than in females. This should be considered when applying glycolytic flux parameters in performance diagnostics and training prescription, particularly in female endurance athletes.

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