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ORIGINAL ARTICLE

Maximal glycolytic flux modulates metabolic thresholds independent of maximal oxygen uptake

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Abstract

Purpose This study examined how maximal glycolytic flux, indexed by the peak lactate accumulation rate (vLa_{peak}) from a 15-s sprint, relates to lactate- and gas-exchange derived endurance thresholds at a given $\dot{V}O_2max$ in trained cyclists.

Methods Fifty cyclists (30 men, 20 women) completed two laboratory visits: a ramp test with verification to determine $\dot{V}O_2max$, and on a separate day, a 15-s all-out sprint with repeated post-exercise capillary blood sampling to determine peak lactate accumulation (ΔLa) and derived vLa_{peak}, followed by a 3-min step-increment test with lactate and gas-exchange measurements.

Results Partial correlations controlling for $\dot{V}O_2max$ revealed moderate-to-strong negative associations between vLa_{peak} and selected lactate- and gas-exchange–derived thresholds ($r = -0.37$ to -0.59, $p<0.01$), whereas absolute and relative 15-s work showed consistently weaker or no relationships ($r=0.06$ to -0.48). No association was observed between vLa_{peak} and peak fat oxidation. In general linear models, adding vLa_{peak} to models with $\dot{V}O_2max$ and sex as predictors significantly increased the explained variance in threshold power ($\Delta R^2 \approx 0.06$ to 0.20). Sex contributed additional but consistently smaller effects.

Conclusion These findings indicate that the power output at several metabolic thresholds originates from the interaction of maximal oxidative and glycolytic flux. As such, vLa_{peak} seems to be a promising parameter that complements traditional measures like $\dot{V}O_2max$ and helps to improve metabolic profiling and exercise prescription. However, longitudinal studies are needed to verify these findings.

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Abbreviations

BM	Body mass
Fat_{max}	Power output associated with maximal fat oxidation
FFM	Fat-free mass
LT	Lactate threshold
MAD	Mean absolute deviation
MLSS	Maximal lactate steady state
$P_{+0.5}$	Power at minimum value + 0.5 mmol·L^{-1} lactate concentration
$P_{+1.5}$	Power output at minimum value + 1.5 mmol·L^{-1} lactate concentration
P_2	Power output at 2 mmol·L^{-1} lactate concentration
P_4	Power output at 4 mmol·L^{-1}
PFO	Peak fat oxidation rate
P_{LaWmin}	Power output at minimal lactate equivalent

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P_{VT1}	Power output at VT_1
P_{VT2}	Power output at VT_2
rpm	Revolutions per minute
vLa_{max}	Maximal lactate accumulation rate
VT_1	Ventilatory threshold 1
VT_2	Ventilatory threshold 2
ΔLa	Maximal capillary blood lactate accumulation

Introduction

Maximal oxygen uptake ($\dot{V}O_2max$) reflects the highest rate to produce adenosine triphosphate (ATP) through oxidative phosphorylation and is widely recognized as a primary determinant of endurance performance (Lucía et al. 1998; Joyner 1991; Hargreaves and Spriet 2020). Mechanistically, $\dot{V}O_2max$ emerges from the integrated limits of pulmonary diffusion capacity, cardiac output, blood oxygen-carrying capacity, and skeletal muscle oxidative capacity (Bassett and Howley 2000).

However, for many endurance sports, maximal lactate steady state (MLSS) and associated lactate (LT) or ventilatory thresholds (VT) are often more critical for performance differentiation than $\dot{V}O_2max$ per se (Lucía et al. 1998; Joyner 1991; Hagberg and Coyle 1983; Farrell et al. 1979; Coyle et al. 1988, 1991; Yoshida et al. 1987; Faude et al. 2009). Over the last decade, the mechanistic interpretation of lactate and gas-exchange thresholds has shifted away from the original "anaerobic" or dysoxic view (Poole et al. 2021). It has been argued that the rise in blood lactate concentration and excess carbon dioxide (CO_2) at the so-called anaerobic threshold is better explained by an increased rate of glycolysis and lactate release into the blood relative to lactate removal, rather than by widespread muscle anoxia (Poole et al. 2021). In this view, thresholds emerge when glycolytic flux and the associated proton load exceed the rate of oxidative and buffering processes to stabilize blood lactate and acid–base status during progressively increasing exercise (Brooks 2010, 2018; Ferguson et al. 2018).

This flux-based perspective highlights glycolysis as a key determinant of threshold behaviour. A plausible explanation is that glycolytic flux at submaximal intensities is not constant across individuals, but scales with each person's maximal glycolytic capacity – analogous to expressing oxygen uptake at threshold as a fraction of $\dot{V}O_2max$ (Mader 2003; Mader and Heck 1986). Consequently, athletes with a higher maximal glycolytic flux may reach a given threshold at a lower external power output because, at the same submaximal workload, their glycolytic system is recruited more readily and provides a greater fraction of total ATP.

The metabolic model proposed by Mader (Mader 1984; Mader and Heck 1986) provides a mechanistic-mathematical model for the interaction between (maximal function of) glycolytic and oxidative pathways (Brooks et al. 2022a; Ferguson et al. 2018) to explain the genesis of thresholds (Heck and Wackerhage 2024). It describes lactate steady-state equilibrium based on the interplay between two primary rates: the maximal rate of oxidative phosphorylation ($\dot{V}O_2max$) and maximal glycolytic flux (vLa_{max}). In practice, conceptual vLa_{max} is estimated from the empirical maximal lactate accumulation rate during a short, maximal sprint (termed vLa_{peak}) and is regarded as a proxy for maximal glycolytic activity in the absence of direct cellular measurements (Wackerhage et al. 2022, 2025; Heck et al. 2003). Within this framework, higher vLa_{max} is predicted to shift the lactate–power curve leftward at a given $\dot{V}O_2max$, lowering the power sustainable at lactate-defined thresholds and altering substrate-use patterns (Wackerhage et al. 2022, 2025; Beneke 2003b).

These model predictions have important implications for metabolic flexibility (San-Millan and Brooks 2018) and for the prevention and treatment of non-communicable diseases (Esteves and Stanford 2024), yet the connection between maximal glycolytic flux and thresholds determined during incremental exercise tests has so far only been addressed in simulations, not in experimental human data. In particular, the model predicts that a lower maximal glycolytic flux reduces pyruvate availability at lower intensities, thereby promoting greater fat oxidation as long as oxidative lactate clearance exceeds lactate production at a given workload (Beneke 2003a, b; Brooks et al. 2022b; Sablain et al. 2025).

Consequently, as stated above, a lower maximal glycolytic flux should theoretically be associated with higher peak fat oxidation (PFO) and with a rightward shift of the workload eliciting maximal fat oxidation (Fat_{max}) (Jeukendrup and Achten 2001; Beneke 2003a, b; Mader 2003), but this has not been demonstrated empirically using direct experimental estimates of maximal glycolytic flux.

One practical and increasingly used method to estimate maximal glycolytic flux in vivo is based on a short-duration all-out sprint test. In this case, a 10–30 s sprint (Heck et al. 2003) is combined with repeated post-exercise blood lactate sampling to determine the maximal peak lactate accumulation (ΔLa) and the corresponding peak accumulation rate (vLa_{peak}, $mmol \cdot L^{-1} \cdot s^{-1}$), which can be interpreted as an empirical marker of maximal whole-body glycolytic flux (Margaria et al. 1964; di Prampero and Ferretti 1999; Ferretti 2015, 2023).

In contrast to the modelling approach of metabolism, incremental step tests provide an accessible method for identifying metabolic thresholds and substrate oxidation patterns in many endurance sports, thereby informing training interventions and performance diagnostics (Faude et al. 2009; Jamnick et al. 2020). Yet, the theoretical link

between vLa_{peak} and thresholds derived from such tests, and its consequences for substrate-use profiles, remains largely untested. Recent work even reported that $\dot{V}O_2max$ was the strongest predictor of a fixed lactate threshold, whereas vLa_{peak} showed little independent explanatory value (Fischer et al. 2025), raising questions about how maximal glycolytic flux and oxidative capacity jointly shape endurance exercise metabolism. vLa_{max} and the related empirically determined index vLa_{peak} have been consistently linked to sprint performance, with strong correlations reported between maximal glycolytic flux and short-duration power output (Clark and Macdermid 2025; Held et al. 2023; Meixner et al. 2024b, 2025b, c). Based on Mader's model, however, markers of maximal glycolytic flux should also help explain endurance-related variables, potentially providing more physiologically meaningful information than mechanical metrics alone despite their close interdependence (Meixner et al. 2024b).

Moreover, several studies have highlighted potential sex differences in metabolic profiles, including distinct patterns of fat oxidation, lactate thresholds relative to peak power, and glycolytic capacity between men and women (Esbjörnsson-Liljedahl et al. 2002; Benitez-Muñoz et al. 2024a, 2024b; Benítez-Muñoz et al. 2025; Thron et al. 2024; Meixner et al. 2024b; Quittmann et al. 2022). These disparities likely reflect differences in hormonal milieu, muscle fiber composition, and enzyme activities that influence substrate utilization and glycolytic flux. Accordingly, sex may modify the relationship between maximal glycolytic flux, thresholds, and substrate use, with implications for performance diagnostics and training prescription (Oosthuyse and Bosch 2006; Devries 2016; Boisseau and Isacco 2022; Tarnopolsky and Ruby 2001).

Given that $\dot{V}O_2max$ on its own is a strong independent predictor of metabolic flexibility (San-Millan and Brooks 2018) and exercise metabolism (Burtscher et al. 2023; Millet et al. 2023) in general, it may mask relationships with glycolytic parameters, it is essential to account for its influence in statistical analyses. Therefore, this study aims to: (i) evaluate correlations between sprint-derived glycolytic flux parameters (i.e. vLa_{peak}, as an empirical marker for maximal glycolytic flux) and various established lactate (P_2; $P_{+0.5}$; P_4; $P_{+1.5}$; P_{LaWmin}), ventilatory (P_{VT1}; P_{VT2}) thresholds and fat oxidation (Fat_{max}); (ii) quantify the explanatory power of a 15-s sprint test and therefrom derived vLa_{peak} for these metabolic thresholds when added to models that already include $\dot{V}O_2max$; and (iii) examine whether these relationships differ between men and women.

We tested the hypothesis that the implications of maximal glycolytic flux extend beyond short-duration exercise, i.e. that maximal glycolytic flux, indexed by vLa_{peak} from a 15-s sprint, would be negatively associated with first and second thresholds, independent of $\dot{V}O_2max$, and would provide greater explanatory value than purely mechanical measures of sprint performance. We further hypothesized that these associations would be present in both sexes and across a selection of metabolic thresholds, but that their magnitude might differ between men and women.

Table 1 Anthropometric data for total study population and female and male group

Age [y]	Total ($n=50$)	Male ($n=30$)	Female ($n=20$)
	31.2 ± 7.8	34.3 ± 7.8	26.4 ± 5.0
Height [cm]	177 ± 9.3	183 ± 7.0	170 ± 6.6
Body mass [kg]	71.7 ± 12.4	78.6 ± 10.3	61.4 ± 6.6
Fat-free mass [kg]	61.9 ± 11.5	69.2 ± 7.9	50.9 ± 5.9

Methods and materials

Participants

A cohort of fifty ($n=30$ male, $n=20$ female) healthy and experienced cyclists with more than 3 years of cycling exercise (> 2 sessions per week) was recruited for this study. All participants were experienced in road cycling with clipless pedals and cycled regularly as exercise. Prior to the study, the participants were informed of the protocol and gave their written informed consent to participate. All procedures were approved by the ethical committee of Exercise Science & Training of the Faculty of Human Sciences (EV2024/1–1004) and conducted in accordance with the Declaration of Helsinki (Harriss and Atkinson 2009). Participant characteristics are given as Mean ± SD in Table 1.

Study design

Two experimental visits (T1 and T2) to the laboratory were required, which were at least 48 h apart and completed within a 7-day period. Participants underwent a sprint test as familiarization and a ramp protocol during the first visit; a sprint test and a step incremental test during the second visit. All testing was performed in the laboratory of the department of sport science and sport in Erlangen from April to December of 2022. The overall study design is illustrated in Fig. 1.

An a priori target of $N=50$ participants (≥ 1/3 female) was set based on feasibility constraints (McCrum et al. 2022). In addition, for the planned multiple linear regression analyses adjusting for $\dot{V}O_2max$ and sex, assuming $\alpha=0.05$ (two-sided), $N=50$ provides approximately 80% power to detect an incremental effect of vLa_{peak} of $f^2 \approx 0.18$ when added to the covariate model (Abt et al. 2025; Lakens 2022).

All participants were instructed to keep a nutrition diary and to repeat their usual diet for each visit within the 24 h before each experimental visit (Jeacocke and Burke 2010). In addition, all were instructed to stay adequately hydrated,

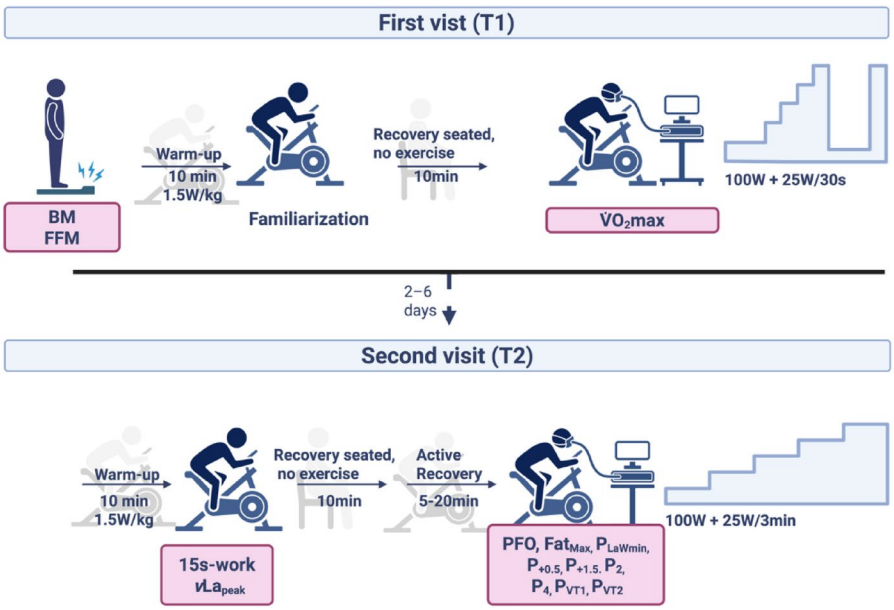

Fig. 1 Time course of study design

to eat a carbohydrate-rich meal (i.e., a banana and a jam sandwich) no less than 3 h before each visit, and to refrain from caffeine consumption on the day of testing. Each participant received 35 g of a carbohydrate mixture (IsoFast, DextroEnergy, Krefeld, Germany) dissolved in 500 ml of water to drink *ad libitum* during warm-up and recovery periods. The main intent of this procedure was to ensure adequate hydration and carbohydrate availability after the warmup, sprint and recovery period for the step test in line with recommendations (Thomas et al. 2016); based on previous work, minimal to no influence on thresholds is to be expected (Rotstein et al. 2007; Aandahl et al. 2021).

All cycling tests were conducted on a Cyclus2 ergometer (RBM, Leipzig, Germany) and their own personal road bike. The Cyclus2 is an electromagnetically braked ergometer and measures power with an accuracy error of 2% according to the manufacturer. Considering that all participants performed the tests in a similar gear, between-bike losses of mechanical power output induced by drivetrain friction are considered minimal (Dahmen et al. 2011; Lanaspeze et al. 2025). All cyclists used their own shoes and pedals for all tests. A warmup of 10 min at a load of 1.5 W · kg^{-1} BM was used for all visits to the laboratory.

Body composition

During the first visit, stature was measured as well as body mass (BM) and fat-free mass (FFM) of all participants, employing eight-electrode impedance analysis (InBody 720, Biospace, Des Moines, Iowa, USA)(McLester et al. 2020)

15s-sprint

The 15 s all-out cycle sprint was performed in a seated position utilizing the large chainring (if applicable) of the participant's bike and the 15-tooth cog of the ergometer. A short, unloaded pedalling phase < 3 s and rpm < 30 was employed before the sprint, recording of the test started when cadence surpassed 30 rpm. The ergometer software was set to isokinetic mode and 130 rpm (Adam et al. 2015; Nitzsche et al. 2018). Capillary blood samples of the left earlobe were sampled twice during the resting period, after the warm-up and once directly after the sprint as well as every minute for 9 min after the 15-s cycle sprint. We ensured capillary blood lactate values reached peak values by continuing measurement if no clear peak was reached by the end of the 9th minute. Resting lactate concentration was defined as the mean of the two pre-exercise samples. νLa_{peak} was calculated as indicated in the formula (Heck et al. 2003; Wackerhage et al. 2025)

$$\nu La_{peak} = \frac{La_{peakpost} - La_{pre}}{t_{test}} = \frac{\Delta La}{15s}$$

Ramp protocol

To determine $\dot{V}O_2max$ and peak power output (PPO_{ramp}), all participants performed a ramp protocol in T1. The participants began cycling at 100 W for 2 min with a freely chosen cadence, after which the load increased by 25 W every 30 s (Adam et al. 2015). The ramp ended when volitional exhaustion was reached, or the cadence dropped by more than 10 rpm. Following 3-min active recovery at 75 W, a verification phase at 100% of the load at the last fully

completed 30 s until volitional exhaustion was performed (Midgley and Carroll 2009).

Incremental step protocol

To determine power output at various endurance thresholds, participants underwent an incremental step protocol (T2). The participants began the test with a freely chosen cadence but were advised to maintain their regular cadence. The test started at 100 W, with the load increasing by 25 W every 3 min. Three participants wished to start at 75 W based on previous results. The test concluded when volitional exhaustion was reached or when cadence consistently dropped by more than 10 rpm. Capillary blood samples of the left earlobe were sampled before the start of the step test and in the last 15 s of every step.

Gas exchange and fat oxidation measurement

Participants were fitted with a Hans Rudolph V2 mask (Hans Rudolph, Inc, Shawnee, KS, USA), and expired gases along with breathing volume were analyzed using a Cosmed Quark CPET system (Cosmed Srl, Rome, Italy). Gas and volume analyzers were calibrated before each test using precision gas (16% O_2 and 5% CO_2) and a volume pump, following the manufacturer's instructions (Airgas Therapeutics, Plumsteadville, PA, USA). Gas exchange parameters were averaged over 10-s intervals.

In line with the manufacturer's recommendations, we employed a mixing chamber setup for the ramp test and breath-by-breath measurements for the step incremental test. $\dot{V}O_2$max was calculated as the highest moving average over 30 s in the ramp test as indicated in Omnia software (v2.4.2., Cosmed Srl, Rome, Italy) (Nolte et al. 2023; Iannetta et al. 2018), matching the step duration of ramp increments.

Only the final 30 s of each step were considered for analysis and fat oxidation rates were determined using standard nonprotein stoichiometric equations for moderate to high intensity according to Jeukendrup and Wallis (2005). VT_1 and VT_2 were determined following the method described by (Keir et al. 2022) and assessed by two independent experienced researchers in exercise testing and prescription who were blinded to the identity of the athletes and the aim of the study. Power output at VT_1 and VT_2 was linearly interpolated based on the test duration.

Lactate concentration measurement and threshold determination

Lactate concentration was measured amperometric-enzymatically employing Biosen C-Line (EKF Diagnostics, Barleben, Germany) from 20 µL earlobe capillary samples. Lactate values were interpolated using third-order polynomial regression analysis, with all regressions attaining adequate goodness of fit ($R^2 \geq 0.97$). Based on the regression, the following thresholds were calculated as power output: the minimal equivalent of lactate (P_{LaWmin}), lactate concentration at the minimal lactate value during the step test + 0.5 mmol·L^{-1} ($P_{+0.5}$) respectively + 1.5 mmol·L^{-1} ($P_{+1.5}$) and thresholds at fixed lactate concentrations of 2 mmol·L^{-1} and 4 mmol·L^{-1} (P_2 resp. P_4)(Pallarés et al. 2016; Hartmann et al. 1990).

Statistical analysis

All data were collected and exported in Microsoft Excel. Statistical analysis was performed employing jamovi (The jamovi project, v2.6.26.0) and Python 3.11.7, using the numpy 1.26.4, statsmodels 0.14.0 and matplotlib 3.8.0 packages. Normality of the lactate accumulation parameters, power outputs, and $\dot{V}O_2$max was assessed using the Shapiro–Wilk test, without requiring further transformation, except for power output at Fat_{max}. Median and mean absolute deviation (MAD) are therefore given for Fat_{max} (Bouza 2013). The level of significance (α) was set to 0.05 for all statistical analyses.

Partial Pearson correlations were conducted to examine the relationship between parameters derived from the sprint test (primarily $vLa_{peak)}$ and endurance thresholds, while controlling for $\dot{V}$O2max.

We fitted ordinary least squares linear regression models for all thresholds through a base model of $\dot{V}O_2$max and sex. The categorical predictor sex was coded with 0 = females, 1 = males for inclusion in the model. We expanded this model by adding $vLa_{peak,}$ 15s-work and 15s-work/FFM in separate models. Incremental model contribution of each sprint-derived predictor beyond the base model was quantified as the change in explained variance (ΔR^2) between nested models (e.g., M_0 vs. M_1). Statistical significance of ΔR^2 was tested using partial F-tests comparing the full and reduced ordinary least squares models. As secondary analyses, we evaluated whether 15s-work or 15s-work/FFM provided additional explanatory value when added to the vLa_{peak} model.

Results

Results of sprint test and selected endurance thresholds for male, female and the all participants are presented in Table 2.

Table 2 Mean ± SD for all relevant parameters determined in the sprint and incremental step test, Median ± MAD for Fat_{max}

	Total (n = 50)	Male (n = 30)	Female (n = 20)
15s-work [kJ]	11.373 ± 2.927	13.131 ± 2.325	8.736 ± 1.285
15s-work/FFM [J/kg]	182 ± 22	190 ± 24.7	172 ± 10.6
ΔLa [mmol·L^{-1}]	6.28 ± 1.57	6.69 ± 1.68	5.66 ± 1.18
νLa_{peak} [mmol·L^{-1}·s^{-1}]	0.42 ± 0.11	0.45 ± 0.11	0.38 ± 0.08
PFO [g/min]	0.380 ± 0.15	0.420 ± 0.152	0.327 ± 0.118
PFO [mg/min/kg FFM]	5.37 ± 1.96	5.38 ± 2.01	5.36 ± 1.94
Fat_{max} [W]	150 ± 25	150 ± 62.5	125 ± 0
P_{LaWmin} [W]	163 ± 40	186 ± 30	128 ± 25
$P_{+0.5}$ [W]	196 ± 48	225 ± 36	153 ± 28
P_2 [W]	208 ± 54	239 ± 43	162 ± 30
$P_{+1.5}$ [W]	224 ± 52	256 ± 38	177 ± 28
P_4 [W]	248 ± 57	283 ± 43	196 ± 30
P_{VT1} [W]	200 ± 46	224 ± 40	163 ± 24
P_{VT2} [W]	250 ± 50	279 ± 40	206 ± 24

FFM fat-free mass, *ΔLa* maximal lactate accumulation, νLa_{peak} maximal lactate accumulation rate, *PFO* peak fat oxidation, Fat_{max} power output associated with maximal fat oxidation, P_{LaWmin} minimal lactate equivalent, P_2 fixed 2 mmol·L^{-1}lactate threshold, $P_{+0.5}$individual lactate threshold at minimum value + 0.5 mmol·L^{-1}, P_4 fixed 4 mmol·L^{-1}lactate threshold, $P_{+1.5}$ individual lactate threshold at minimum value + 1.5 mmol·L^{-1}, P_{VT1} power output at ventilatory threshold 1, P_{VT2} power output at ventilatory threshold 2

νLa_{peak} displayed significant negative correlations to all thresholds but not to PFO or Fat_{max} in the partial correlation analysis (Fig. 2).

General linear models displayed a significant effect of $\dot{V}O_2max$ and of νLa_{peak}. Estimate of νLa_{peak} was negative. Sex had a significant effect for all lactate-derived thresholds except for Fat_{max} and consistently had smaller effect sizes than νLa_{peak}. For P_{VT1} and P_{VT2}, we display the influence of νLa_{peak} without consideration of sex in Fig. 3. Models containing νLa_{peak} displayed consistently higher ΔR^2 than those integrating 15s-work or 15s-work/FFM (Table 3). Nested model comparisons adding either 15s-work or 15s-work/FFM to M_1 with νLa_{peak} yielded small but significant improvements for 15s-work/FFM and thresholds P_{LaWmin}, $P_{+0.5}$, $P_{+1.5}$ and P_4 (Table 4). Figures of other thresholds as well as full model parameters are supplied in supplementary material.

Discussion

The primary aim of this study was to characterize how maximal glycolytic flux, indexed via a 15-s sprint through νLa_{peak}, relates to several endurance thresholds at a given $\dot{V}O_2max$, and thereby to describe the interplay between

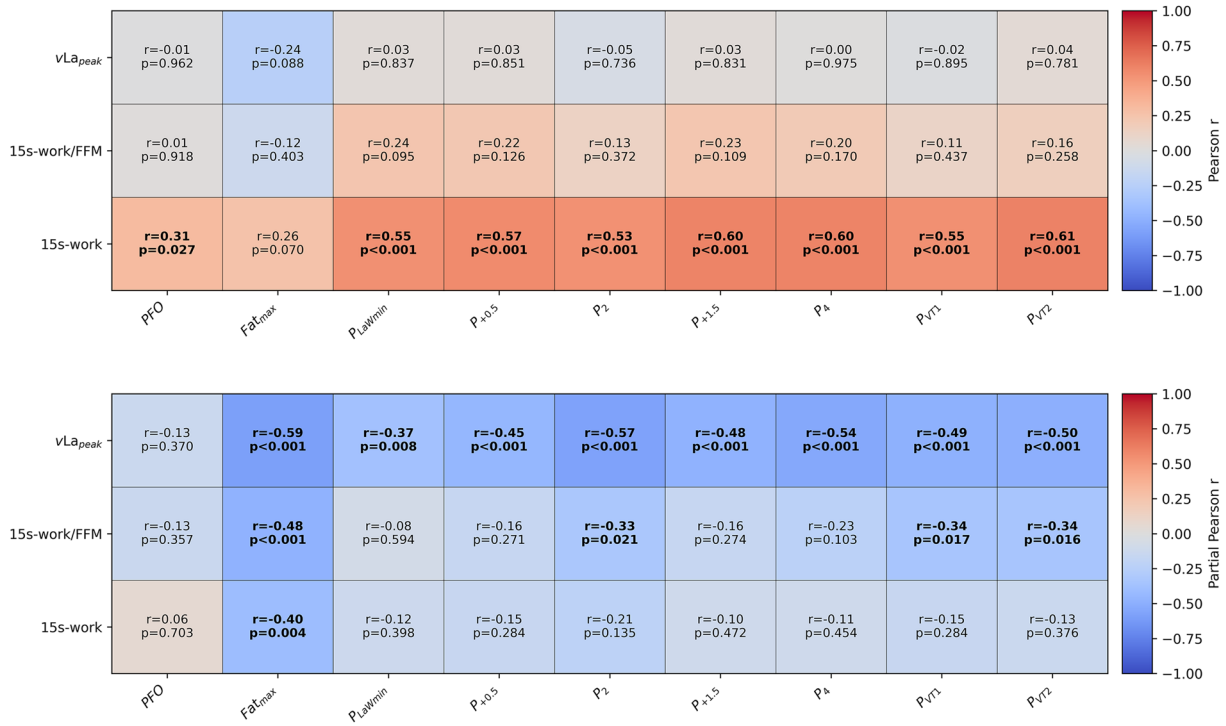

Fig. 2 Pearson and partial Pearson correlations between thresholds and sprint test parameters. *FFM* fat-free mass, νLa_{peak} maximal lactate accumulation rate, *PFO* peak fat oxidation,Fat_{max} power output associated with maximal fat oxidation, P_{LaWmin} minimal lactate equivalent, P_2 fixed 2 mmol·L^{-1}lactate threshold, $P_{+0.5}$ individual lactate threshold at minimum value + 0.5 mmol·L^{-1}, P_4 fixed 4 mmol·L^{-1}lactate threshold, $P_{+1.5}$ individual lactate threshold at minimum value + 1.5 mmol·L^{-1}, P_{VT1} power output at ventilatory threshold 1, P_{VT2} power output at ventilatory threshold 2. *indicates significant correlations

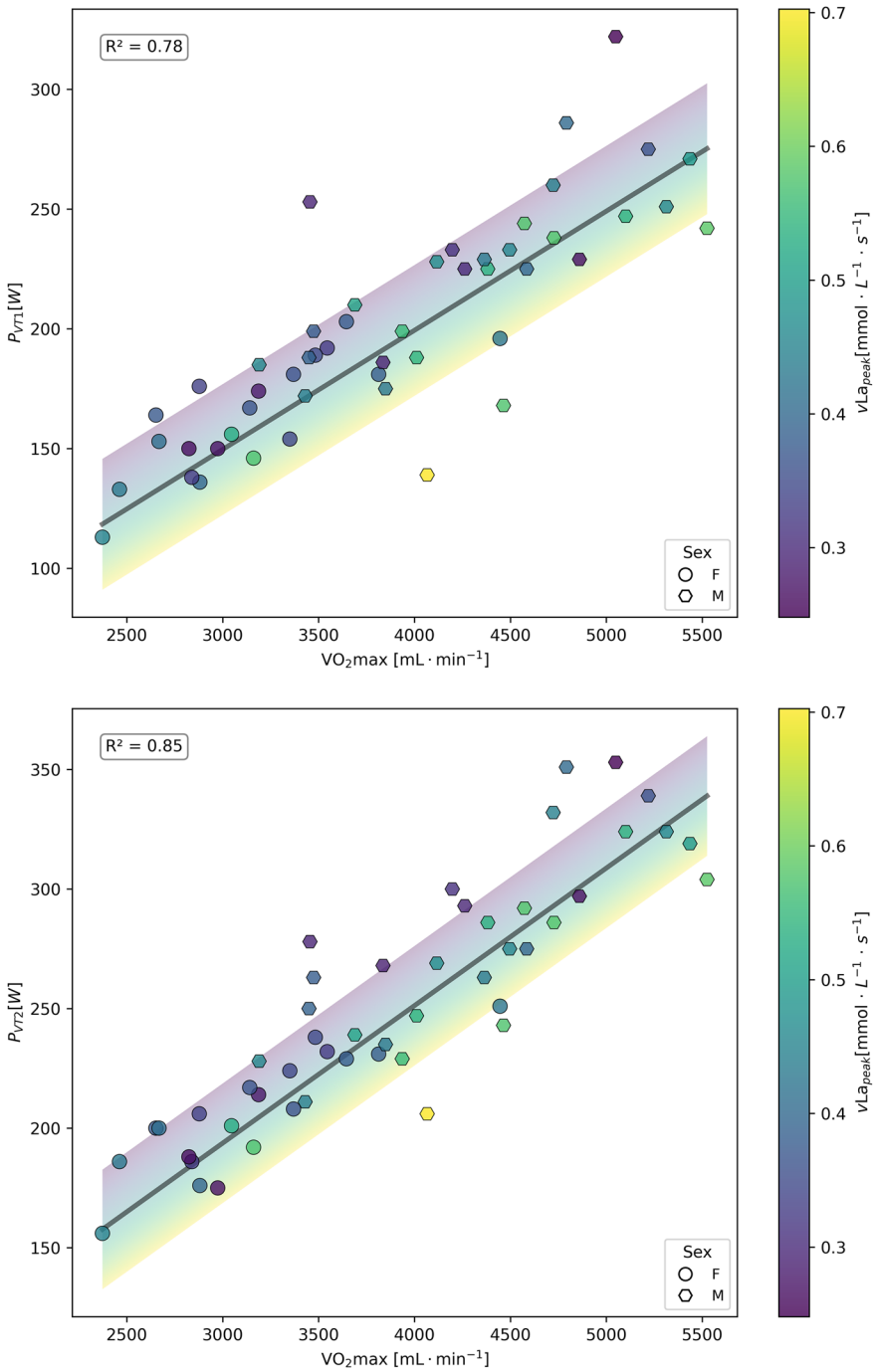

Fig. 3 Visual representation of influence of vLa_{peak} on A P_{VT1}, P_{VT2}, by including $\dot{V}O_2max$, vLa_{peak} as predictors

glycolytic and oxidative pathways in vivo. The key findings were:

i) vLa_{peak} showed stronger negative partial correlations with selected lactate- and gas-exchange–derived endurance thresholds than absolute or relative 15-s power, after statistically accounting for $\dot{V}O_2max$;
ii) including vLa_{peak} alongside $\dot{V}O_2max$ in linear models substantially increased the variance explained in threshold power, consistent with the notion that the lactate–power relationship is shaped by the combined limits of maximal oxidative and maximal glycolytic flux rather than by $\dot{V}O_2max$ alone;
iii) sex was identified as a significant independent factor for most lactate-derived thresholds, although its contribution was consistently smaller than that of vLa_{peak}, suggesting that sex-related differences in glycolytic flux, substrate use, and lactate kinetics modulate the expression of thresholds but do not replace the primary role of maximal flux parameters.

Table 3 Results for model comparison of M_0 with $\dot{V}O_2$max and sex as predictors and adding either vLa_{peak}, 15s-work or 15s-work/FFM as predictors

Threshold	Parameter added to M0	B	β	*p*	R2 (full)	ΔR2 (vs. M0)	*p*(ΔR2)
Fat_{max}	vLa_{peak}	−189.724	−0.478	< 0.001	0.629	0.203	< 0.001
Fat_{max}	15s-work	−0.008	−0.551	0.001	0.540	0.114	0.001
Fat_{max}	15s-work/FFM	−0.773	−0.409	< 0.001	0.565	0.139	< 0.001
P_{LaWmin}	vLa_{peak}	−99.643	−0.263	< 0.001	0.776	0.062	< 0.001
P_{LaWmin}	15s-work	−0.004	−0.281	0.025	0.744	0.030	0.025
P_{LaWmin}	15s-work/FFM	−0.178	−0.099	0.252	0.723	0.008	0.252
$P_{+0.5}$	vLa_{peak}	−127.403	−0.278	< 0.001	0.850	0.069	< 0.001
$P_{+0.5}$	15s-work	−0.005	−0.292	0.007	0.814	0.032	0.007
$P_{+0.5}$	15s-work/FFM	−0.301	−0.138	0.065	0.797	0.016	0.065
P_2	vLa_{peak}	−181.466	−0.352	< 0.001	0.857	0.110	< 0.001
P_2	15s-work	−0.006	−0.327	0.005	0.787	0.040	0.005
P_2	15s-work/FFM	−0.568	−0.231	0.003	0.791	0.044	0.003
$P_{+1.5}$	vLa_{peak}	−137.248	−0.279	< 0.001	0.879	0.069	< 0.001
$P_{+1.5}$	15s-work	−0.004	−0.243	0.018	0.832	0.022	0.018
$P_{+1.5}$	15s-work/FFM	−0.311	−0.133	0.056	0.825	0.015	0.056
P_4	vLa_{peak}	−167.516	−0.306	< 0.001	0.890	0.083	< 0.001
P_4	15s-work	−0.005	−0.232	0.025	0.827	0.020	0.025
P_4	15s-work/FFM	−0.438	−0.168	0.015	0.831	0.023	0.015
P_{VT1}	vLa_{peak}	−132.906	−0.306	< 0.001	0.798	0.083	< 0.001
P_{VT1}	15s-work	−0.004	−0.239	0.059	0.736	0.021	0.059
P_{VT1}	15s-work/FFM	−0.485	−0.234	0.005	0.761	0.046	0.005
P_{VT2}	vLa_{peak}	−125.579	−0.262	< 0.001	0.880	0.061	< 0.001
P_{VT2}	15s-work	−0.004	−0.209	0.037	0.836	0.017	0.037
P_{VT2}	15s-work/FFM	−0.465	−0.203	0.002	0.854	0.034	0.002

FFM fat-free mass, vLa_{peak} maximal lactate accumulation rate, *PFO* peak fat oxidation, Fat_{max} power output associated with maximal fat oxidation, P_{LaWmin} minimal lactate equivalent, P_2 fixed 2 mmol·L^{-1}lactate threshold, $P_{+0.5}$ individual lactate threshold at minimum value + 0.5 mmol·L^{-1}, P_4 fixed 4 mmol·L^{-1} lactate threshold, $P_{+1.5}$ individual lactate threshold at minimum value + 1.5 mmol·L^{-1}, P_{VT1} power output at ventilatory threshold 1, P_{VT2} power output at ventilatory threshold 2

Table 4 Results for nested model comparison of M_1 with $\dot{V}O_2$max, vLa_{peak} and sex as predictors and adding either 15s-work or 15s-work/FFM as predictors

Threshold	Parameter added to M1	B	β	*p*	R2 (full)	ΔR2 (vs. M1)	*p*(ΔR2)
Fat_{max}	15s-work	−0.002	−0.159	0.399	0.635	0.006	0.399
Fat_{max}	15s-work/FFM	−0.128	−0.068	0.667	0.630	0.002	0.667
P_{LaWmin}	15s-work	−0.001	−0.052	0.721	0.777	0.001	0.721
P_{LaWmin}	15s-work/FFM	0.497	0.275	0.021	0.801	0.025	0.021
$P_{+0.5}$	15s-work	−0.001	−0.047	0.697	0.851	0.001	0.697
$P_{+0.5}$	15s-work/FFM	0.455	0.208	0.033	0.865	0.015	0.033
P_2	15s-work	0.000	0.010	0.932	0.857	0.000	0.932
P_2	15s-work/FFM	0.303	0.123	0.203	0.862	0.005	0.203
$P_{+1.5}$	15s-work	0.001	0.034	0.749	0.879	0.000	0.749
$P_{+1.5}$	15s-work/FFM	0.522	0.223	0.010	0.896	0.017	0.010
P_4	15s-work	0.002	0.093	0.362	0.892	0.002	0.362
P_4	15s-work/FFM	0.494	0.189	0.023	0.902	0.012	0.023
P_{VT1}	15s-work	0.001	0.082	0.557	0.800	0.002	0.557
P_{VT1}	15s-work/FFM	0.051	0.025	0.830	0.798	0.000	0.830
P_{VT2}	15s-work	0.001	0.062	0.563	0.881	0.001	0.563
P_{VT2}	15s-work/FFM	0.032	0.014	0.874	0.881	0.000	0.874

FFM fat-free mass, vLa_{peak} maximal lactate accumulation rate, *PFO* peak fat oxidation, Fat_{max} power output associated with maximal fat oxidation, P_{LaWmin} minimal lactate equivalent, P_2 fixed 2 mmol·L^{-1}lactate threshold, $P_{+0.5}$ individual lactate threshold at minimum value + 0.5 mmol·L^{-1}, P_4 fixed 4 mmol·L^{-1} lactate threshold, $P_{+1.5}$ individual lactate threshold at minimum value + 1.5 mmol·L^{-1}, P_{VT1} power output at ventilatory threshold 1, P_{VT2} power output at ventilatory threshold 2

Maximal glycolytic flux and endurance thresholds

The results of our study indicate that there is a significant negative association between the sprint-derived peak lactate accumulation rate (vLa_{peak}), as a marker of maximal glycolytic flux, and selected endurance thresholds. This supports the concept outlined in Mader's model (Mader 1984, 2003; Mader and Heck 1986) that these thresholds emerge from the interplay between maximal oxidative and maximal glycolytic flux rather than from $\dot{V}O_2max$ alone and warrants further elucidation of the mechanisms behind this relationship. The comparable associations for P_{VT1}, P_{VT2} and Fat_{max} suggest the vLa_{peak} - threshold link is not specific to lactate curve fitting, but also manifests in gas-exchange–based breakpoint behavior.

In the primary modelling framework, adding vLa_{peak} to the base model containing $\dot{V}O_2max$ and sex ($M_0 \rightarrow M_1$) consistently improved model fit across all threshold outcomes, with $\Delta R^2 \approx 0.06–0.20$ (partial F-tests: all $p < 0.01$). This indicates that vLa_{peak} captures a meaningful portion of inter-individual variability in threshold power that is not explained by $\dot{V}O_2max$ alone, supporting the view that glycolytic flux capacity contributes to threshold behaviour beyond differences in maximal aerobic power.

In secondary nested comparisons, we tested whether sprint work metrics provide additional explanatory value once vLa_{peak} is already included. Adding 15-s work to the vLa_{peak} model produced negligible changes in explained variance ($\Delta R^2 \approx 0.00–0.006$) and was not significant across outcomes (partial F-tests: $p > 0.05$), suggesting that absolute sprint work does not contribute materially beyond vLa_{peak} in this dataset. By contrast, adding 15s-work/FFM yielded small but occasionally significant improvements for selected lactate-derived thresholds ($\Delta R^2 \approx 0.012–0.025$, partial F-tests: $p < 0.05$ in several outcomes), indicating a modest additional component associated with size-normalized sprint work that is not fully captured by vLa_{peak}. Overall, these results reinforce vLa_{peak} as the dominant sprint-derived predictor within the framework, while suggesting limited incremental information from work/FFM for some thresholds.

Although previous studies have shown that work performed during a 15-s all-out sprint is closely associated with body composition and lactate accumulation (Meixner et al. 2024b, 2025b, 2025c), vLa_{peak} demonstrates stronger and more consistent correlations with endurance-related markers than either absolute or relative 15-second sprint work. Therefore, we conclude that the peak lactate accumulation rate during a 15-s sprint serves as a more specific indicator of maximal glycolytic flux and is more sensitive than power output during the sprint, which depends on other factors such as body composition, mechanical efficiency, or neuromuscular activation (Bundle and Weyand 2012). This is especially noteworthy as glycolytic contribution and work accomplished during a 15-s sprint are strongly linked (Meixner et al. 2024b) and is positively related to 60 s power output (Clark and Macdermid 2025). Previously, this negative relationship between a marker of maximal glycolysis and endurance thresholds had been shown only in theory. Additionally, lactate measurement for the determination of glycolytic flux is essential, as only capillary blood lactate concentrations provide a meaningful representation of glycolytic activity across all active musculature beyond what power output in the 15s-sprint alone can indicate, despite the close linkage of the two parameters and the energetic significance of lactate accumulation (Meixner et al. 2024b; Ferretti and di Prampero 2026).

Previous studies have highlighted the utility of all-out sprint tests and the associated maximal capillary blood lactate accumulation as integrative markers of endurance performance and metabolic profile (Ji et al. 2021; Quittmann et al. 2022; Hauser et al. 2014; Poffé et al. 2024). As theorized in Mader's equations (Mader and Heck 1986), the maximal lactate accumulation rate in active muscles plays a crucial role in determining the fractional utilization of $\dot{V}O2max$. The present data extend these theoretical and modelling observations – in a simplified way and through statistical methods to alleviate methodological challenges – by showing that a directly measured vLa_{peak} relates to multiple lactate- and gas-exchange–derived thresholds after accounting for $\dot{V}O_2max$, thereby experimentally supporting the idea that submaximal glycolytic behaviour at thresholds is partly determined by an individual's maximal glycolytic flux.

Peak fat oxidation

No significant correlation was found for peak fat oxidation (PFO). PFO plays an important role in longer duration endurance exercise where the athlete is not metabolically but energetically limited. However, this finding in our data, which is not supported by the Mader model, may be caused by several factors: (i) the determination of PFO is not a very reliable testing procedure (Chrzanowski-Smith et al. 2020); (ii) the previously performed sprint test with considerable accumulation of blood lactate may influence the capacity for fat oxidation (San-Millan and Brooks 2018) even though lactate levels were lowered before the start of the incremental test; (iii) other factors such as time of day, habitual nutrition and muscle glycogen levels as well as age may influence the measurement of PFO more than $\dot{V}O_2max$ and vLa_{peak} (Amaro-Gahete et al. 2018; Maunder et al. 2018; Purdom et al. 2018). Thus, the absence of a significant association between maximal glycolytic flux and PFO in the present study should be interpreted cautiously and

may reflect methodological noise and acute test sequencing rather than a true absence of interaction between glycolytic and oxidative pathways at lower intensities.

Statistical instead of physiological modeling

We tested a sprint-derived marker of maximal glycolytic flux (νLa_{peak}) as an informative descriptor of the oxidative–glycolytic interplay at endurance thresholds, rather than to fully implement the original physiological modelling approach of Mader (Mader 1984, 2003). We rely on statistical modelling instead of full physiological modelling as described by Mader (Mader 2003; Mader and Heck 1986) to show that νLa_{peak} is more strongly related to threshold power than power-based sprint parameters, supporting its value as a glycolytic flux marker. Several reasons were responsible for this approach. The νLa_{max} parameter is theorized in the level of muscle metabolism and a necessity for the model simulation, but cannot readily be measured directly and is instead assumed to be approximated by νLa_{peak} (Wackerhage et al. 2025).

Further, the determination of an alactic time component remains unresolved. As power-based parameters have been shown to exhibit only poor to moderate reliability (Meixner et al. 2024a), a fixed alactic time frame could in principle resolve this issue (Wackerhage et al. 2025). However, there is still debate about this approach and about which fixed value to use, especially in relation to total test duration (Meixner et al. 2024a; Langley et al. 2024, 2025; Dunst et al. 2023). Nonetheless, an assumption of this time frame (even if set to 0 s) is needed to attain a model-based rate of maximal lactate accumulation in addition to total test time and ΔLa.

Further methodological issues persist in the estimation of maximal glycolytic rate as a proxy measure for maximal glycolytic activity: it remains unclear whether the test should be optimized for maximal lactate accumulation or for maximal mechanical performance (Dunst et al. 2025; Haase et al. 2024). The optimal duration for the test is also unresolved, as some authors consider durations of 10 to 12 s better suited because higher νLa_{peak} values are attained (Quittmann 2025; Langley et al. 2025; Porter and Langley 2025). We consider not the highest possible blood lactate values to be optimal, but those with the highest informational value across metabolic demands.

While conceptual markers of muscle glycolytic activity (νLa_{max}) are required for simulation and insertion into the model, experimental studies can only access peak parameters of lactate accumulation at the whole-body level through concentration measures in capillary blood (Wackerhage et al. 2025). Since its inception, it has been suggested that the actually measured parameter is only an estimation (Heck et al. 2003). Nonetheless, our νLa_{peak}, calculated as ΔLa divided by sprint duration, follows the recently proposed calculation method (Wackerhage et al. 2025) and therefore represents an empirical marker of maximal net glycolytic flux rather than a fully modelled νLa_{max} of the active muscle.

It is also important to recognize that capillary blood lactate accumulation following the sprint test may be influenced by cardiovascular and hemodynamic function. The transport of lactate from muscle to blood is mediated by monocarboxylate transporters and changes in their expression may alter the appearance of lactate in capillary blood (Dubouchaud et al. 2000; Thomas et al. 2005). Additionally, systemic lactate uptake and clearance by tissues such as the heart, brain, liver, and skeletal muscle may contribute to variations in capillary blood lactate accumulation and are themselves influenced by endurance training status (Brooks 2009; Dubouchaud et al. 2000; Leija et al. 2025). Therefore, our measurements of maximal glycolytic flux reflect net accumulation rather than true production of lactate, and a direct measure of intramuscular lactate production rate is not possible through capillary blood measurements alone. Furthermore, lactate clearance capacity might be a confounding factor for both endurance thresholds and post-sprint lactate accumulation. It also needs to be mentioned that lactate accumulation after an all-out sprint test does not represent a peak instantaneous value of glycolysis, but merely the mean value over the duration of the test.

The original approach by Margaria (Margaria et al. 1964) to determine maximal lactate accumulation rate used a series of supramaximal exercise tests. Interestingly enough, this rate was similar across participants and uphill running grades and comparatively close the average value of our male participants. The onset of lactate accumulation depended on Phosphocreatine (PCr) stores, which is something we could not replicate in our own study in the sprint test under creatine supplementation (Meixner et al. 2025c). Nonetheless, it may be that the sprint test is sensitive enough to accurately display a maximal rate of glycolysis in a specific exercise mode irrespective of PCr stores to achieve predictive value for endurance exercise. Therefore, we refrained from the calculation of a fully parameterized νLa_{max} for insertion into simulation models and opted for a simplified statistical modelling approach, using νLa_{peak} as an empirical marker of maximal glycolytic flux and examining its relationship with thresholds and fat oxidation at a given $\dot{V}O_2max$. In this sense, the present work should be viewed as an experimental constraint and augmentation of physiological modelling, rather than as a replacement for it. In this context, our results complement recent predictive modelling work in cycling (e.g., Wahl & Ji's MuDo-PD) by providing an empirical constraint on whether a sprint-derived glycolytic marker adds explanatory information beyond $\dot{V}O_2max$

and threshold anchors (Wahl and Ji 2026). In line with the discussion and potential influence of alactic time on $v\mathrm{la}_{max}$ (Dunst et al. 2023) and hence simulation results, we chose to forgo this discussion and eliminate this parameter by setting it equal to zero. For physiological modelling, alactic time remains a primary calibration problem while this is not a problem in regression analysis.

Due to these methodological issues, the Mader model and the parameter $v\mathrm{La}_{max}$ so far have failed to gain broader international scientific recognition and application, preventing wider understanding and exploration of the mechanisms described in the model. The results of our study show that a simple empirically derived glycolytic flux parameter $v\mathrm{La}_{peak}$ already provides substantial information for constructing a metabolic profile of an athlete. Furthermore, it provides experimental evidence that metabolic behaviour under incremental loads reflects the interplay between maximal oxidative and maximal glycolytic rates. We stress that our findings, obtained through statistical modelling, should be interpreted cautiously as qualitative support for flux-based models of thresholds and as a starting point for the refinement and wider application of physiological models of exercise metabolism.

Sex differences

The results of our study indicate that sex is an additional factor, independent of $v\mathrm{La}_{peak}$ and $\dot{V}O_2max$, in explaining variation in endurance thresholds. In the general linear models, men showed higher threshold power than women at a given $\dot{V}O_2max$ and $v\mathrm{La}_{peak}$, although the effect size of sex was consistently smaller than that of $v\mathrm{La}_{peak}$. Previous studies have described differences in maximal lactate accumulation between males and females (Meixner et al. 2024b, 2026a; Quittmann 2025). It remains unclear whether this is predominantly influenced by muscle architecture (Esbjörnsson-Liljedahl et al. 2002), training and neuromuscular activation (Bundle and Weyand 2012), or sex-related differences in lactate distribution and elimination, together with differences in fat-free mass (FFM) between the sexes (Tripp et al. 2025).

It has also been reported that females have a higher fractional utilization of thresholds in relation to their $\dot{V}O_2max$ (Benítez-Muñoz et al. 2024a, 2024b, 2025). This could mechanistically be explained by their muscle fiber distribution (Esbjörnsson-Liljedahl et al. 2002) and their lower maximal lactate accumulation (Mader and Heck 1986; Meixner et al. 2024b). However, in our data, regression results display the opposite pattern, i.e. at the same $\dot{V}O_2max$ and $v\mathrm{La}_{peak}$, threshold power was slightly higher in men. The study by Benítez-Muñoz et al. (2025) employed only a step incremental test, whereas in our study, $\dot{V}O_2max$ was obtained from a ramp test and thresholds were determined in a separate incremental step test (Meixner et al. 2026b). The same applies other studies in this area that employ only one test for both threshold and maximal variables (Johansen et al. 2025; Meixner et al. 2026b), but interestingly enough, these studies may further benefit from including $v\mathrm{La}_{peak}$ as additional and mechanistic information. Therefore, methodological differences, including test protocols and definitions of thresholds, are potentially the source of the disparity observed in our study. In addition, our models did not explicitly adjust for mechanical efficiency or muscle mass, which may mediate part of the sex effect on threshold power.

Unlike Fischer et al. (2025), we did not assess efficiency because of the comparatively short duration of 3 min per step. We stress that we determined predictors in separate specific tests, unlike their approach of deconstructing thresholds through a single step incremental test. It is likely that efficiency could enhance statistical modelling and should also be included as a factor in physiological modelling (Sablain et al. 2025), but from our perspective, should be evaluated in an independent diagnostic procedure. Future work that combines measurements of maximal glycolytic flux, mechanical efficiency and muscle mass that consider biological sex differences may therefore better resolve how sex modulates the relationship between oxidative and glycolytic fluxes at endurance thresholds. Our data suggest that the effect of interaction of rates is larger than differences rooted in sex of individuals, but this factor may modulate the behavior of this interaction.

Physiological interpretation

At the cellular level, maximal glycolytic flux is likely governed by the combined activity of rate-limiting glycolytic enzymes, particularly phosphofructokinase (PFK) and other key control steps in glycolysis (Zuo et al. 2021; Boscá and Corredor 1984; Mor et al. 2011), together with the capacity of monocarboxylate transporters (MCT1, MCT4) to export lactate from producer fibres and import it into oxidative tissues (Kitaoka et al. 2012; Bonen 2001; Seyedi et al. 2024). These processes determine how rapidly pyruvate and cytosolic NADH are generated and how much of this reducing-equivalent load can be handled by mitochondrial oxidation versus lactate formation and export (Spriet et al. 2000; Vigh-Larsen et al. 2021). In this context, our finding that higher sprint-derived maximal glycolytic flux ($v\mathrm{La}_{peak}$) is associated with lower threshold power at a given $\dot{V}O_2max$ may represent a systems-level manifestation of glycolytic–oxidative competition for pyruvate and NADH turnover, as envisaged by contemporary lactate shuttle and threshold concepts (Brooks 2009, 2018; Poole et al. 2021; Glancy

et al. 2021). At higher maximal glycolytic flux, glycolytic activation during incremental exercise will more rapidly increase pyruvate and NADH supply, so that the point at which lactate appearance outpaces disappearance is reached at a lower external workload, consistent with a leftward shift of the lactate–power relationship.

Importantly, this *push* interpretation of glycolysis is only one plausible perspective. An alternative (and not mutually exclusive) view is that glycolytic flux is largely *pulled* by ATP demand and regulatory signals, i.e., by the mismatch between ATP demand and the combined capacity of oxidative phosphorylation and phosphagen buffering, with νLa_{peak} reflecting glycolytic responsiveness rather than a primary upstream driver. This interpretation is consistent with classical control concepts in which glycolytic rate is modulated by metabolites linked to ATP turnover (e.g., ADP/P_i) alongside constraints imposed by oxidative capacity. Thus, our data are compatible with multiple mechanistic framings while pointing to the same functional outcome – a leftward shift of the lactate–power relationship at higher νLa_{peak}.

Practical implications and perspective

The results of our study indicate experimental support for the informational value of maximal glycolytic flux and its influence on endurance-related markers at a given $\dot{V}O_2max$. Despite the methodological challenges of assessing maximal glycolytic rate, the value of the model for practice does not only lie in the simulation of metabolism but rather in the qualitative understanding of the underlying flux-based mechanisms. When recognizing maximal glycolytic flux of the working musculature as an important influence on endurance performance, training interventions can specifically aim for a directional change in this parameter. A simple 15-s sprint with post-exercise lactate sampling, yielding νLa_{peak}, may therefore complement traditional $\dot{V}O_2max$ and threshold testing when constructing an athlete's metabolic profile.

Traditional lactate curve analysis fails to provide more mechanistic understanding behind threshold genesis through the metabolic profile of an athlete (Bleicher et al. 1998). In contrast, combining $\dot{V}O_2max$ and νLa_{peak} explicitly frames thresholds as emergent properties of the interaction between maximal oxidative and maximal glycolytic fluxes, offering a more mechanistic basis for interpreting individual differences and training responses. Future studies should focus on the interplay of νLa_{peak} and $\dot{V}O_2max$ through training interventions, quantifying how much of the change in endurance performance and thresholds is attributable to adaptations in oxidative versus glycolytic flux, and whether this balance should be targeted differently across sexes, sports and competitive demands (Quittmann 2025). Furthermore, it is unclear how νLa_{peak} may develop in different training programs and if any change in νLa_{peak} is reflected in longitudinal lactate curve analysis. Although our cohort consisted of trained cyclists, the principle that the interplay of maximal aerobic flux and maximal glycolytic flux modulate the energetic contributions across intensities may also be relevant for understanding exercise intolerance and metabolic inflexibility in clinical populations.

Strengths and limitations

A key strength of this study is its sample size and inclusion of both male and female cyclists, allowing us to examine sex as an independent factor in the relationship between maximal glycolytic flux and endurance thresholds. Additionally, prior familiarization sessions enhanced the reliability of sprint test outcomes (Meixner et al. 2024a).

A wide array of endurance thresholds exists (Jamnick et al. 2020; Faude et al. 2009) and these provide information relevant to training zones, but not necessarily a direct approximation of maximal lactate steady state. Our study did not assess this equilibrium state, which also depends on definitions as well as measurement accuracy and reliability. Nonetheless, the Mader model originally calculates steady-state conditions based on lactate production and removal.

Cadence is a potential confounding factor in the determination of efficiency (Marsh et al. 2000) with additional effects on endurance thresholds, especially with regard to Mader's model (Dunst et al. 2025). This was not controlled for in our analysis, as self-selected cadence most likely reflects the participants' habitual pedalling patterns best. Furthermore, νLa_{peak} was derived from capillary blood lactate and reflects net accumulation rather than true intramuscular production flux (as discussed above), and the test order (sprint preceding the step test) as well as pre-exercise carbohydrate intake may have influenced substrate use and fat oxidation. We did not directly assess muscle enzyme activities, MCT expression or mitochondrial density, so the proposed mechanistic links between νLa_{peak}, enzyme or transporter function and threshold behaviour remain inferential. Finally, our cohort consisted of trained cyclists, which limits generalizability of the present findings to other sports and less-trained or clinical populations but represents a suitable model population for proof-of-concept.

Conclusion

νLa_{peak} was negatively associated with several endurance thresholds and improved regression models beyond $\dot{V}O_2max$ and sex, with larger incremental contributions

than 15-s work metrics when compared in separate one-marker models. Secondary analyses indicated small additional contributions of 15-s work/FFM beyond vLa_{peak} for some lactate-derived thresholds. These findings suggest that sprint-derived lactate kinetics provide information relevant to threshold power that is not captured by $\dot{V}O_2max$ alone, but confirmation in longitudinal and more diverse cohorts is required.

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Author contributions BM was involved in conceptualization, methodology, investigation, formal analysis, visualization, and writing – original draft and writing – review and editing. PL was involved in formal analysis and writing – review and editing. BS was involved in conceptualization, methodology, resources and writing – review and editing.

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Declarations

Conflict of interest Authors state no conflict of interest.

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