

Lactate threshold predicting time-trial performance: impact of heat and acclimation

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Lorenzo S, Minson CT, Babb TG, Halliwill JR. Lactate threshold predicting time-trial performance: impact of heat and acclimation. *J Appl Physiol* 111: 221–227, 2011. First published April 28, 2011; doi:10.1152/jappphysiol.00334.2011.—The relationship between exercise performance and lactate and ventilatory thresholds under two distinct environmental conditions is unknown. We examined the relationships between six lactate threshold methods (blood- and ventilation-based) and exercise performance in cyclists in hot and cool environments. Twelve cyclists performed a lactate threshold test, a maximal O₂ uptake ($\dot{V}O_{2\max}$) test, and a 1-h time trial in hot (38°C) and cool (13°C) conditions, before and after heat acclimation. Eight control subjects completed the same tests before and after 10 days of identical exercise in a cool environment. The highest correlations were observed with the blood-based lactate indexes; however, even the indirect ventilation-based indexes were well correlated with mean power during the time trial. Averaged bias was 15.4 ± 3.6 W higher for the ventilation- than the blood-based measures ($P < 0.05$). The bias of blood-based measures in the hot condition was increased: the time trial was overestimated by 37.7 ± 3.6 W compared with only 24.1 ± 3.2 W in the cool condition ($P < 0.05$). Acclimation had no effect on the bias of the blood-based indexes ($P = 0.51$) but exacerbated the overestimation by some ventilation-based indexes by an additional 34.5 ± 14.1 W ($P < 0.05$). Blood-based methods to determine lactate threshold show less bias and smaller variance than ventilation-based methods when predicting time-trial performance in cool environments. Of the blood-based methods, the inflection point between steady-state lactate and rising lactate (INFL) was the best method to predict time-trial performance. Lastly, in the hot condition, ventilation-based predictions are less accurate after heat acclimation, while blood-based predictions remain valid in both environments after heat acclimation.

heat stress; heat acclimation; heat acclimatization; critical power; endurance exercise

SEVERAL PHYSIOLOGICAL PARAMETERS, namely, maximal O₂ uptake ($\dot{V}O_{2\max}$), lactate threshold, ventilatory threshold, fraction of slow-twitch fibers, and running economy, are known to be related to (or predictive of) endurance exercise performance (12, 13, 19). As the margins for success in athletic competition are often quite small, coaches, athletes, and physiologists have long been interested in assessing an individual athlete's lactate or ventilatory threshold, in an effort to use such information to design more effective training plans, optimize an athlete's performance, or make race-day predictions. The terms "lactate threshold" and "ventilatory threshold" have generally been used to define the highest work rate or O₂ uptake ($\dot{V}O_2$) at which athletes can maintain their efforts over a specified time frame. To the best of our knowledge, no study has focused on

simultaneously determining the accuracy of lactate threshold vs. ventilatory threshold in predicting time-trial performance in hot and cool conditions in highly trained cyclists.

A variety of terms, including direct lactate measurements, such as lactate threshold (32), onset of blood lactate accumulation (46), and maximal lactate steady state, have been used to describe such critical thresholds (5). Other indirect methods to estimate such thresholds use pulmonary gas exchange data (40, 49) (i.e., ventilatory threshold) or electromyographic signals (40). Several studies have aimed to compare different methods to determine the lactate and ventilatory thresholds using different protocols (9, 11, 35, 40), although it is difficult to draw conclusions, as the results heavily depend on the specific methodologies used (e.g., cycle or treadmill, duration of exercise stage, blood sampling site). Others have attempted to investigate the relationship between lactate or ventilatory threshold and exercise performance in thermoneutral conditions (6, 19, 50). However, these studies commonly fail to standardize the comparisons to a specific environmental condition (laboratory vs. field) or activity (treadmill vs. road running) when comparing lactate or ventilatory threshold with exercise performance. In other words, the lactate or ventilatory threshold and exercise performance test may be affected differently if it is assessed on a cycle ergometer in the laboratory vs. on a bicycle outdoors. Therefore, to best determine optimal workout intensities or predict race-day performance, an accurate assessment of this critical threshold is crucial.

To our knowledge, no study has tried to determine direct (blood-based) and indirect (ventilation-based) indexes of this critical threshold and relate them to exercise performance (i.e., 1-h time trial) in highly trained athletes and documented potential differences between these direct and indirect measurements of endurance performance in cool and hot environmental conditions. Many endurance competitions take place in temperatures different from the standard thermoneutral laboratory conditions, and it is well documented that exercising in a cooler (i.e., $<15^\circ\text{C}$) or hotter (i.e., $>35^\circ\text{C}$) environment can affect lactate dynamics (41) and exercise performance (21). For instance, exercise at cold ambient temperatures results in a greater activation of the sympathoadrenal system, which may influence the nature of available substrates and, therefore, the plasma lactate response (27). On the other hand, exercise heat stress causes an increase in anaerobic glycolysis resulting from local muscle hypoxia caused by a reduced muscle blood flow and O₂ delivery (20). Therefore, documentation of any potential differences between lactate or ventilatory threshold and time-trial performance in hot and cool environmental conditions will be of great interest to endurance athletes, as they would be able to use this information to better determine optimal workout intensities or adapt their race-day strategies.

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Therefore, the purpose of this investigation was to study the relationship between exercise performance (1-h time trial) and lactate or ventilatory threshold in highly trained cyclists under two distinct environmental conditions: hot (38°C, 30% relative humidity) and cool (13°C, 30% relative humidity). As ventilatory threshold seems to occur at higher workloads than lactate threshold (22, 44), we hypothesized that endurance performance would be better correlated with lactate than ventilatory threshold in hot and cool environments. In addition, we used heat acclimation to manipulate the performance capacity in highly trained cyclists and compare how each index changed in relation to endurance performance. Heat acclimation may induce metabolic adaptations during exercise by reducing the aerobic metabolic rate (43) or decrease the rate of glycogenolysis (33), which may affect lactate or ventilatory threshold. In addition, as the ventilatory adaptations that follow a period of heat acclimation (1) might dissociate the correlation between ventilation- and blood-based indexes of lactate threshold, we hypothesized that ventilatory threshold would be less correlated with performance following heat acclimation.

METHODS

This study on measurements of lactate threshold is part of a recently published larger study of heat acclimation and performance (34). However, the current study focuses on a comparison of several methods used to measure lactate and ventilatory thresholds and predict exercise performance.

Subjects. All protocols were approved by the Institutional Review Board of the University of Oregon. Prior to participation, each volunteer gave written informed consent as set forth by the Declaration of Helsinki. Twelve highly trained endurance cyclists [10 men, 2 women; 24 ± 6 (SD) yr old, 175 ± 6 cm height, 67.7 ± 8.1 kg body wt, 22.1 ± 3.9 kg/m² body mass index] completed the heat acclimation protocol. Eight subjects (7 men, 1 woman; 26 ± 4 yr old, 174 ± 6 cm height, 70.2 ± 4.1 kg body wt, 23.1 ± 3.1 kg/m² body mass index) completed the control protocol. Of these eight controls, four subjects underwent the heat acclimation program after completing their control experiments. The heat acclimation and control groups were matched for maximal aerobic power and training experience (34).

Study design. Participants completed a lactate threshold test, a $\dot{V}O_{2\max}$ test, and a 1-h time trial (on a different day) in two environmental conditions, before and after an exercise-heat acclimation or control program. On days when the tests were performed under heat stress, the climatic chamber was set to 38°C and 30% relative humidity [wet bulb globe temperature (WBGT) = 33°C]. On days when the studies were performed in cool conditions, the climatic chamber was set to 13°C and 30% relative humidity (WBGT = 12°C). The cool environment was selected, because it approximates the thermal conditions believed to be optimal for aerobic performance (21). The order between hot and cool trials was randomized. The heat acclimation protocol consisted of 10 exposures of cycle ergometer exercise at 40°C and 30% relative humidity (WBGT = 35°C). Subjects performed two 45-min bouts at 50% of their $\dot{V}O_{2\max}$, with 10 min of rest between bouts. A matched control group exercised at the same intensity, but with the chamber set at 13°C and 30% relative humidity. The 50% $\dot{V}O_{2\max}$ exercise intensity was selected, as it would represent compensable heat stress sufficient to induce heat acclimation but would not be sufficient to induce training adaptations for our highly trained athletes (17). Subjects were instructed to maintain their normal training routines during the 10-day intervention period to maintain their fitness level. Postacclimation studies were completed within 1 wk of the conclusion of the heat acclimation period. No attempt was made to control for training during the lead-in phase of the study, although subjects were recruited from the same

club team and, thus, had the same competition schedule and essentially identical training routines.

On each study visit, subjects reported to the laboratory after a 2-h fast and well hydrated. To ensure that the subjects were properly hydrated, nude body weight and plasma osmolality were measured. Euhydration was demonstrated by nude body mass within 1% of their 5-day average and plasma osmolality <290 mosmol/kgH₂O (42). Subjects were instructed to avoid consumption of alcohol or caffeine for ≥ 8 –12 h prior to the study. In addition, they were not allowed to exercise on the day prior to the study and were told to avoid ingestion of nonprescription drugs for the duration of the multiple study visits.

Measurements. Exercise was performed while the subjects were seated on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Heart rate was monitored continuously throughout each protocol via telemetry (model RS400, Polar Electro, Lake Success, NY). Core temperature was measured using continuous recordings of rectal temperature by a thermistor (YSI 400 Series, Mallinckrodt Medical, St. Louis, MO) inserted 10 cm beyond the anal sphincter. $\dot{V}O_2$, CO₂ production ($\dot{V}CO_2$), and expired minute ventilation (\dot{V}_E) were measured breath-by-breath by custom-purposed software (KCBeck Physiological Consulting, St. Paul, MN) interfaced with a respiratory mass spectrometer (Marquette MGA 1100, MA Tech Services).

Prior to initiation of the lactate threshold and $\dot{V}O_{2\max}$ tests in the hot condition, subjects were immersed in a water bath ($\sim 41^\circ\text{C}$) for ~ 30 min to increase their rectal temperature by 0.8–1.0°C. For the protocols in cool environmental conditions, subjects were immersed in a thermoneutral ($\sim 34^\circ\text{C}$) water bath for 30 min to maintain normothermia. The water immersion allowed us to manipulate the subject's rectal and skin temperatures without employing exercise prior to the tests, which could potentially act as a confounding variable. Therefore, we could examine the impact of heat acclimation state on the different exercise tests in a standardized heat stress condition.

Lactate threshold protocol. At ~ 30 min after water immersion, subjects performed cycle ergometer exercise continuously in 3-min stages. The initial power output was selected on the basis of the subject's height, weight, and reported usual training workload. This initial power output represented a "very light" [Borg scale rating of perceived exertion (RPE) ≤ 9] workload for the subject. Power output increments were selected, such that the test concluded after four to seven stages. The test was terminated when blood lactate levels began to rise exponentially (≥ 2 mmol/l from the previous stage). Gas exchange was continuously measured by open-circuit spirometry. During the last 30 s of each stage, a capillary blood sample was taken from a fingertip and analyzed for lactate concentration (Lactate Pro, Arkray, Kyoto, Japan), and lactate values were obtained 60 s after the blood sample was taken.

$\dot{V}O_{2\max}$. At ~ 45 min after completing the lactate threshold test, subjects performed a $\dot{V}O_{2\max}$ test. This recovery time has been shown to be adequate to prevent bias in subsequent aerobic performance tests (31). To elicit $\dot{V}O_{2\max}$, subjects exercised to exhaustion on a cycle ergometer (test duration 8–15 min), with the power output increasing 20 W/min. The initial power output was selected on the basis of the subject's height, weight, and reported usual training workload and also represented a very light workload. Gas exchange was continuously measured by open-circuit spirometry.

Time-trial performance. On a separate day, the subjects performed a 1-h time trial. After a brief warm-up (5 min at 40% of maximal power on a cycle ergometer), subjects performed at their maximal effort for 60 min. Total work completed (in kJ) and mean power output (in W) after 1 h were the performance variables measured. During the test, the cycle ergometer was set to the hyperbolic mode (pedaling rate-independent), and subjects did not receive feedback (i.e., heart rate, power output, core temperature), except for total time elapsed. Subjects were allowed to modify power output as often as needed, but without knowledge of the power output. The starting power was set to 0 W, and the subjects did not know the workload at

the start of the time trial. Every 5 min, power output, cadence, work performed, heart rate, and RPE were measured. Because all the subjects were well trained and had previous experiences performing similar time-trial competitions, a potential “learning effect” was minimized.

Lactate threshold indexes. Three direct (i.e., blood-based) indexes were used to estimate the power output at lactate threshold. 1) We simultaneously fit two regression lines to the blood lactate concentration curve graphed against power output (38). One line was fit to the steady-state values for lactate (i.e., forced to a slope of zero), while the second regression line was fit to the rising lactate phase. Subsequently, the power output at the inflection point between steady state and rising phases in blood lactate (INFL) was determined as the intersection between both lines and was defined as the lactate threshold for the “INFL” method. 2) Power output at the point where blood lactate increased 1.0 mM above resting values was determined by interpolating the regression performed above and was defined as the lactate threshold for the “1mM” method (15). 3) Power output at the point where blood lactate reached 4.0 mM was determined by interpolating the regression performed above and was defined as the lactate threshold for the “4mM” method (32).

These three blood-based lactate indexes were compared with indirect (or ventilation-based) methods using pulmonary gas exchange to estimate the power output at lactate threshold. 1) The power output when the respiratory exchange ratio equaled 1.0 was determined by interpolating the regression between respiratory exchange ratio and power output and defined as the lactate threshold for the “R” method. 2) We simultaneously fit two regression lines to the $\dot{V}_E\text{-}\dot{V}_{O_2}$ curve graphed against power output. Subsequently, the power output at which there was an inflective rise (i.e., intersection between both regression lines) in the $\dot{V}_E\text{-}\dot{V}_{O_2}$ curve was determined and defined as the lactate threshold for the “ $\dot{V}_E\text{-}\dot{V}_{O_2}$ ” method (9). 3) In a similar fashion, the power output that corresponded to the departure of \dot{V}_{CO_2} from a line of identity drawn through a plot of \dot{V}_{CO_2} vs. \dot{V}_{O_2} was defined as the lactate threshold for the “VSLOPE” method (2).

Statistical analyses. The data set used for analyses contained the following numbers of observations. 1) In cool environmental conditions, we had two sets of measurements on each of 19 subjects (1 subject in the control group was excluded because of measurement errors during time trials). The two sets of measurements represented those obtained before and after the 10-day acclimation protocol or an equivalent control period. As there were changes in individual responses across those time periods, we chose to treat pre- and postobservations from a subject as independent observations, so we had 38 observations in cool conditions. 2) In the hot environmental conditions, we used only one set of measurements from each of the 19 subjects taken prior to the 10-day acclimation protocol or equivalent control period ($n = 19$). Similarly, when we compared cool with hot responses, we used 19 observations from individuals studied prior to any acclimation protocol. When we compared pre- with postacclimation responses, we used 12 observations from individuals studied pre- and postacclimation.

Correlation coefficients between lactate threshold estimates and time-trial performance (i.e., mean power output during the 1-h time trial) were calculated as Pearson’s product moment (r). To compare the lactate threshold estimates with a “gold standard” marker across

subjects, we calculated the absolute difference (in W) and the percent difference between each individual’s lactate threshold estimates (in W) and their individual time-trial mean power output (also in W). Across all the lactate and ventilatory threshold estimates under investigation, absolute difference proved to be independent of time-trial mean power output, whereas the percent difference was correlated with time-trial mean power output in some cases. Consequently, the remaining analysis was conducted using the absolute difference for each lactate threshold estimate as an indicator of bias of the estimator. When this bias was compared across all the lactate threshold estimates under investigation, it became apparent that there were differences in variance. Hence, we used Levene’s median test to compare variance across methods. All subsequent analysis and hypothesis testing relied on Proc Mixed (SAS version 9.1.3, SAS Institute, Cary, NC), which can estimate a separate residual variance within each group. Differences were considered statistically significant when $P < 0.05$. Values are means \pm SE unless otherwise noted (in Table 1, we use SD to reflect the variance across the subjects used in the study).

RESULTS

Table 1 shows $\dot{V}_{O_{2\max}}$ and time-trial results for observations in the data set used for this analysis. [For complete performance results of the experimental and control groups, see our previously published study (34).]

Figure 1 (*top*) shows the relationship between the power output at one of the lactate threshold estimates (i.e., the INFL method) and the mean power output during the 1-h time trial in the cool condition. In this example, the correlation was high ($r = 0.892$, $P < 0.05$).

Table 2 lists the correlation coefficients between the power output at each of the different lactate and ventilatory threshold estimates and mean power output during the 1-h time-trial performance in cool and hot environmental conditions. The highest correlations were observed with the blood-based lactate indexes; however, even the indirect ventilation-based indexes were well correlated with mean power during the 1-h time trial.

Figure 1 (*bottom*) shows this “bias of the estimator” vs. the mean power output achieved during the time trial. The lactate threshold estimate was derived using the INFL method. The difference between threshold and time-trial power outputs (bias) was not influenced by the individual’s exercise capacity, as expressed by the time-trial performance. In this example, the power output at the lactate threshold averaged 15.7 ± 3.5 W (or $6.3 \pm 1.4\%$) higher than the mean power output during the time trial.

Figure 2 shows box plots for the bias of the three lactate threshold (blood-based) and three ventilatory threshold (ventilation-based) methods in the cool condition. When bias across all the threshold estimates under investigation are compared, variance was greater for the ventilation- than blood-based measurements ($P < 0.001$, by Levene’s median test). There were no differences in variance within a measurement group

Table 1. Maximal O_2 uptake and time-trial performances in cool and hot environmental conditions

	Cool (13°C)	Hot (38°C)
Maximal O_2 uptake, l/min	4.47 ± 0.21 (3.00–5.51)	$3.73 \pm 0.22^*$ (2.31–4.81)
Maximal power output, W	369 ± 15 (260–430)	$328 \pm 5^*$ (240–420)
Work done during 1-h time trial, kJ	880 ± 49 (588–1,011)	$719 \pm 42^*$ (558–914)
Mean power output during 1-h time trial, W	246 ± 13 (166–305)	$201 \pm 12^*$ (153–239)
Relative power output during 1-h time trial, %maximal power output	65.7 ± 4.9 (55.2–73.8)	$60.4 \pm 6.9^*$ (46.9–74.7)

Values are means \pm SD ($n = 19$ subjects studied preacclimation); ranges are shown in parentheses. * $P < 0.05$ vs. cool.

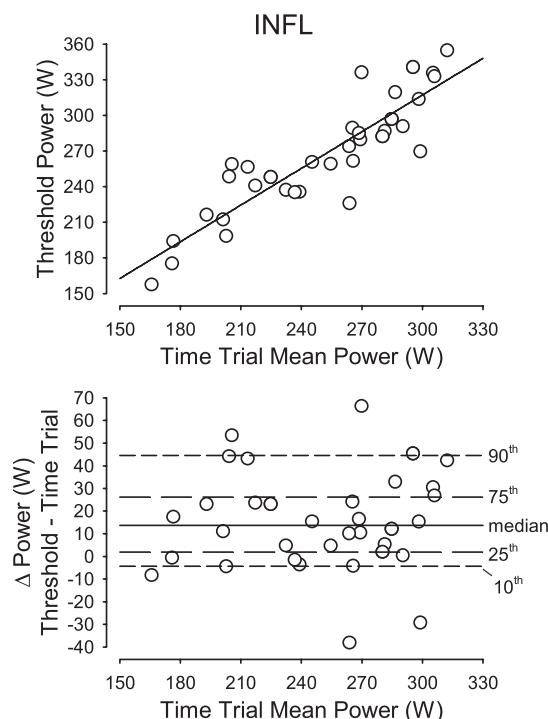


Fig. 1. Power output for INFL compared with mean power output for the 1-h time trial. *Top*: power output at the lactate threshold estimate (INFL method) vs. mean power output during the 1-h time trial. For the regression shown, $y = 1.03x + 8.17$ W, $r = 0.892$. *Bottom*: difference in power output at the lactate threshold estimate (INFL method) and the mean power output achieved during the time trial as a function of the mean power output achieved during the time trial. Each symbol denotes a single set of observations in cool conditions ($n = 38$).

(e.g., all the blood-based methods had similar variance). Mean values ranged from 15.7 to 48.4 W across the six methods. Bias averaged 15.4 ± 3.6 W higher for the ventilation- than blood-based measures ($P < 0.001$). Bias was higher for \dot{V}_E/\dot{V}_{O_2} (48.4 ± 5.0 W) than for R ($P = 0.026$) but was not significantly different from VSLOPE ($P = 0.070$). Of the blood-based methods, the only difference was between the highest (4mM: 28.6 ± 3.2) and the lowest (INFL: 15.7 ± 3.5 W) biased methods ($P = 0.008$), although the 1mM method tended to be higher than the INFL method ($P = 0.060$).

Figure 3 shows the three lactate threshold (blood-based) and three ventilatory threshold (ventilation-based) methods in cool and hot conditions. Overall, there was an increase in the bias of blood-based measures in the hot condition: the time trial was overestimated by 37.7 ± 3.6 W compared with only 24.1 ± 3.2 W in the cool condition ($P = 0.004$). There was a tendency for all the blood-based methods to reflect this elevation in bias ($P = 0.070$ – 0.154). This pattern was not reflected in the ventilation-based measurements (40.6 ± 4.5 and 37.1 ± 4.5 W in hot and cool conditions, respectively, $P = 0.543$).

Figure 4 shows the three lactate threshold (blood-based) and three ventilatory threshold (ventilation-based) methods in hot conditions prior to and after heat acclimation. Heat acclimation increased time-trial performance by 8% (34) under these conditions. While acclimation had no effect on the bias of the blood-based indexes ($P = 0.51$), which tracked the improvements in performance, it exacerbated the overestimation by an additional 34.5 ± 14.1 W for \dot{V}_E/\dot{V}_{O_2} ($P = 0.016$). VSLOPE and R did not reflect this effect of acclimation on bias. No

differences were seen for bias under cool conditions after acclimation for the blood- or ventilation-based methods (data not shown), as both tracked the improvements in time-trial performance of 6% (34).

Lastly, it is not surprising but may be worth documenting that, overall, lactate and ventilatory thresholds in cool conditions overestimate time-trial performance in hot conditions by an additional 42.7 ± 4.2 W (30.5 ± 3.0 and 73.2 ± 3.0 W in cool and hot conditions, respectively, $P < 0.001$).

DISCUSSION

This study is the first to specifically examine the relationship between lactate and ventilatory thresholds and exercise performance using direct (blood-based) and indirect (ventilation-based) methods in highly trained cyclists in two distinct environmental conditions. We also investigated the relationship between blood- and ventilation-based indexes of lactate and ventilatory thresholds and endurance performance before and after a period of heat acclimation. We studied competitive cyclists, because they are highly trained, perform consistent maximal efforts, and represent a target group that could make use of the information provided by the various indexes of this critical threshold. The main findings of this investigation are as follows. 1) Blood-based methods to determine lactate threshold had less bias and smaller variance (Fig. 2) than ventilation-based methods when predicting time-trial performance in cool environmental conditions. 2) In the cool condition, the INFL method was the best method (least bias and minimal variance) to predict time-trial performance. 3) There is an increase in the bias of the blood-based measures when we move to the hot environment (Fig. 3). This pattern is not reflected in the ventilation-based measurements, although it is worth noting that ventilation-based indexes were not better than blood-based indexes in the hot condition (i.e., they still overestimate performance, as they did in the cool condition). 4) In the hot condition, some ventilation-based predictions worsen after heat acclimation (but not in the cool condition), while blood-based predictions remain valid in both environmental conditions after heat acclimation (Fig. 4). 5) Any method (blood- or ventilation-based) assessed in the cool condition is not recommended when predicting time-trial performance in a hot environment.

Prior studies of lactate thresholds and performance. Numerous earlier studies reported an association between lactate and ventilatory thresholds and exercise performance (3, 19, 25, 26, 50). However, these studies focused on the relationships

Table 2. Correlations between power output at each lactate and ventilatory threshold estimate and mean power output during the 1-h time-trial performance in cool and hot environmental conditions

	Cool (13°C)	Hot (38°C)
INFL	0.892	0.874
1mM	0.905	0.885
4mM	0.906	0.858
R	0.797	0.765
\dot{V}_E/\dot{V}_{O_2}	0.824	0.740
VSLOPE	0.838	0.832

$P < 0.05$ for Pearson's product moment for each lactate threshold estimate vs. time-trial performance; $n = 38$ observations in cool condition and 19 in hot condition.

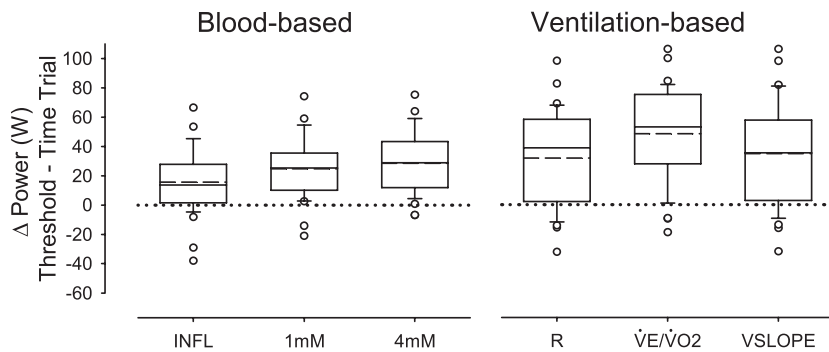


Fig. 2. Box plots of the difference in power output for the threshold estimate and the mean power output for the time trial. Boundary of the box closest to zero denotes 25th percentile, solid line within the box denotes median, dashed line denotes mean, and the boundary of the box farthest from zero denotes 75th percentile. Error bars above and below the box denote 10th and 90th percentiles. \circ , Outliers. Variance was greater for ventilation- than blood-based measurements ($P < 0.05$, by Levene's median test; $n = 38$ observations in cool conditions). $\dot{V}E$, expired minute ventilation; $\dot{V}O_2$, O_2 uptake.

among direct blood lactate measurements (19, 26, 50) or indirect methods using gas exchange (3, 25), but not between blood- and ventilation-based methods and exercise performance. Moreover, these associations have not been thoroughly explored in hot or cool environmental conditions.

Prior work has commonly used the INFL method, the 1mM method, and some of its variations (i.e., blood lactate increases of 0.5 or 0.75 mM above baseline values) (16, 35, 48), which are favored for their relative ease of application and objective nature. Furthermore, these approaches take into account individual variations in the subject's resting steady-state lactate level (which is not the case for the 4mM method). Our results are consistent with these earlier studies, which show that blood-based measures are well correlated with exercise performance (6, 26). We extended these earlier findings by showing that these approaches remain well correlated to time-trial performance in hot and cool environments, regardless of heat acclimation status.

In agreement with others (22, 44), we found that the ventilatory threshold carried at higher power outputs compared with the direct blood lactate measurements. Despite some reports suggesting that ventilation-based determination of lactate threshold is a useful index of triathlon and cycling performance (25, 51), other reports showed that this correlation depends on the duration of the stages during the incremental test (3). Such divergent outcomes may be related to the time delay between release of lactate and hydrogen ions from skeletal muscle and subsequent changes in ventilation. The time delay associated with lactate diffusion from the muscle (23), the retention of a considerable part of the lactate within the muscle (24), the difficulty associated with lactate diffusion above certain concentrations (29), and the potential dissociation between lactate and hydrogen ion clearance from the muscle (28) are factors that might disrupt these relationships. In our study, we have consistently found that the ventilatory methods were less well correlated to time-trial performance, but most importantly, they have a larger variance and are more biased toward overesti-

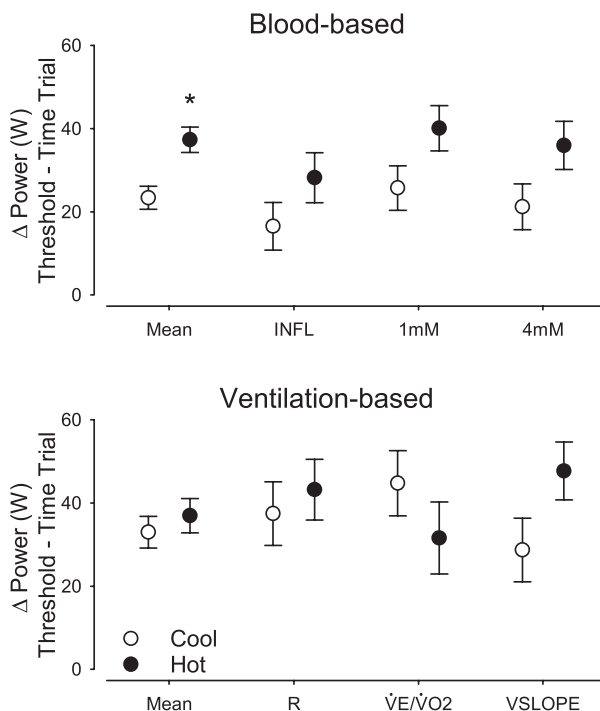


Fig. 3. Effect of environmental conditions on the difference in power output for the threshold estimate and the mean power output for the time trial. $*P < 0.05$ vs. cool condition. Values are means \pm SE ($n = 19$ observations).

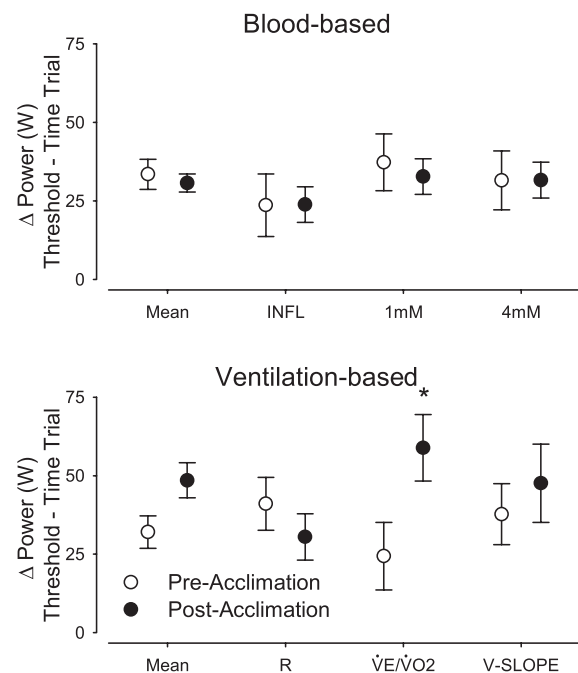


Fig. 4. Effect of heat acclimation on the difference in power output for the threshold estimate and the mean power output for the time trial. $*P < 0.05$ vs. preacclimation. No differences were seen in cool conditions with acclimation (data not shown). Values are means \pm SE ($n = 12$ observations).

mation than the direct blood lactate methods (Fig. 2), especially after a period of heat acclimation in the hot environment. We also observed that bias of blood-based thresholds was increased in the hot condition, but we can only speculate on the reasons for this. This discrepancy could be attributed, in part, to the effect of high core and skin temperatures on substrate (including lactate) metabolism via increased Q_{10} effect (37), the specific duration of the stages during the incremental test (3), differences in blood sampling site (i.e., arterial, venous, capillary), the choice of blood media analyzed (i.e., plasma or lysed or precipitated whole blood), and/or subject population.

The increased bias of the ventilation-based index (i.e., \dot{V}_E/\dot{V}_{O_2}) after heat acclimation in our study may be due to changes in ventilatory response to hyperthermia following heat acclimation. Heat acclimation could have resulted in a change in sensitivity of the respiratory center to afferent inputs from the preoptic anterior hypothalamus (7) or a change in sensitivity of the central chemosensitive areas of the ventral surface of the medulla oblongata to local temperature changes (10). Beaudin et al. (1) showed a significant decrease in esophageal temperature threshold for the onset of increases in ventilatory equivalents for O_2 (\dot{V}_E/\dot{V}_{O_2}) and CO_2 (\dot{V}_E/\dot{V}_{CO_2}) and an increase in ventilation at all levels of esophageal temperature following passive heat acclimation. Thus it is possible that the ventilatory threshold for exercise hyperventilation (i.e., \dot{V}_E/\dot{V}_{O_2}) is pushed to higher work rates during exercise in the heat after acclimation due to an elevated ventilation at lower work rates. Clearly, more research on the effects of heat acclimation on ventilatory parameters and exercise performance is warranted.

Thus, to our knowledge, no studies have simultaneously studied blood lactate and pulmonary gas exchange responses and their relationship to endurance performance in hot and cool environmental conditions, as was our intention. Our observation that the ventilation-based methods have greater bias and variance suggest that they are influenced by additional inputs that do not directly affect performance. Therefore, highly trained cyclists are likely to benefit more by using blood-based methods to determine their lactate threshold and design workout routines and for training prescription (30, 32), whereas reliance on indirect methods using pulmonary gas exchange is more likely to lead to imprecise guidance or, in some cases (e.g., following heat acclimation), large errors between prediction and performance. In addition, portable blood lactate equipment, such as that used for this project, is fairly inexpensive, provides reasonable accuracy, and is easy to use, which makes this tool a very convenient means of monitoring training improvements (8) and predicting exercise performance.

Benefits of heat acclimation. Recently, we showed that a period of heat acclimation improves exercise performance in hot and cool environmental conditions, as assessed by time-trial performance, lactate threshold, and $\dot{V}_{O_{2max}}$ in highly trained cyclists (34). Importantly, we observed that time-trial performance improved 6% in cool conditions and 8% in hot conditions. In the present analysis, we have demonstrated that blood-based indexes of lactate threshold maintain their predictive value across heat acclimation (i.e., improvements in time-trial performance in cool and hot conditions are reflective of proportional improvements in lactate threshold under these same conditions), whereas ventilation-based indexes lose much of their predictive value in the hot environment following heat

acclimation. This is likely a reflection of changes in the control of ventilation with heat acclimation (see above).

Many endurance competitions take place in locations where ambient temperatures differ from the typical “thermoneutral” laboratory environment. As exercise in a cooler (i.e., $<15^\circ C$) or hotter (i.e., $>35^\circ C$) environment can affect lactate dynamics (39, 41) and endurance performance (18, 21), better understanding of how these environments might affect the relationships between lactate threshold and endurance performance should be of great interest to athletes and coaches interested in optimizing racing performances. In particular, competitive cyclists may benefit from having their lactate threshold determined in a few representative temperature ranges, so they can best adapt training and race strategies to the conditions at hand.

The 1-h time trial. While prior studies have examined cycling over shorter (<30 min) or longer (>60 min) time-trial performances (3, 14, 36), we chose to use a 1-h time trial, because it best approximates the time required to complete a 40-km time trial in trained cyclists (4), which is one of the most common distances used in competitive cycling time-trial races. It would be naïve to presume that one specific lactate or ventilatory parameter would best predict endurance performance for all events from 30 min to 3 h (6, 47). Instead, depending on the intensity and duration of the endurance event, different lactate or ventilatory parameters may provide a better estimate of the pace that does not result in premature fatigue (6). Thus some of the indexes that had higher-than-average bias (i.e., \dot{V}_E/\dot{V}_{O_2}) in the present study may be better predictors of shorter-duration trials. However, it seems likely that the variance structure we observed (i.e., greater variance for ventilation-based measures) may generalize to events of differing durations.

In conclusion, our results suggest that direct blood lactate measurements to estimate lactate threshold are better correlated to a 1-h time-trial performance than indirect methods using pulmonary gas exchange data in hot and cool environments. Although often used in the field of exercise physiology and performance, the \dot{V}_E/\dot{V}_{O_2} method is perhaps a poor choice, whereas the INFL method is perhaps the best choice, for events of this duration. Blood lactate thresholds provide a means of monitoring training improvements (8) and predicting exercise performance but are specific to a thermal environment. Consequently, accurately measuring blood lactate threshold in a variety of conditions may provide more information than a single test under thermoneutral conditions, allowing athletes and their coaches to establish specific training programs to enhance their endurance exercise performance, regardless of the weather or degree of acclimation. Finally, these findings pertain specifically to highly trained cyclists and may not be applicable to cyclists of a lesser caliber or other sports, and more research is warranted, given that aerobic fitness can modify lactate dynamics and may cause ventilatory and lactate thresholds to behave differently (45).

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

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