Comparison of Power Outputs During Time Trialing and Power Outputs Eliciting Metabolic Variables in Cycle Ergometry

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The authors sought to compare power output at blood lactate threshold, maximal lactate steady state, and pH threshold with the average power output during a simulated 20-km time trial assessed during cycle ergometry. Participants (N = 13) were trained male and female cyclists and triathletes, all permanent residents at moderate altitude (1,525–2,225 m). Testing was performed at 1,525 or 1,860 m altitude. Power outputs were determined during a simulated 20-km time trial (PTT), at blood pH threshold (PpHT), at maximal lactate steady state (PMLSS), and at blood lactate threshold determined by 2 methods: the highest power output that did not result in consecutive and continued increases in blood lactate concentrations from exercising baseline (PLT) and the highest power output that did not result in consecutive and continued increases of ≥1 mmol/L in blood lactate concentrations from exercising baseline (PLT1). PLT, PLT1, and PMLSS were all significantly lower than PpHT (p < .05) and PTT (p < .05). No significant difference was observed between PpHT and PTT (p > .05). Significant correlations were observed between each of the metabolic variables, PLT, PLT1, PMLSS, and PpHT, compared with PTT (p < .05). The authors conclude that, of the 4 metabolic variables, only PpHT offered an accurate reflection of PTT.

Keywords: acidosis, lactate threshold, pH threshold, maximal lactate steady state

Numerous investigations have suggested that acidosis contributes to muscle fatigue and limits performance in high-intensity exercise (Adams, Fisher, & Meyer, 1991; Hultman, Del Canale, & Sjöholm, 1985; Raymer, Marsh, Kowalchuk, & Thompson, 2004; Spriet, Matsos, Peters, Heigenhauser, & Jones, 1985). Common belief about the source of exercise-induced metabolic acidosis has centered on anaerobic glycolysis and the production of lactic acid. Briefly, this theory contends that, as exercise intensity climbs, increased reliance on anaerobic glycolysis leads to increased production of lactic acid. Once formed, lactic acid is said to dissociate into lactate and a free hydrogen ion by releasing a proton from its carboxyl group (Brooks & Fahey, 1985). As work rate continues to increase, this process is said to lead to two physiological conditions: blood lactate threshold, occurring when lactic acid production rates exceed the rate of lactate removal, and metabolic acidosis, which occurs when free hydrogen ion production overwhelms buffering capacity, resulting in a drop in pH.

The proposed relationship between lactate production and acidosis has prompted exercise physiologists to examine the relationship between lactate threshold and exercise performance (Bentley, McNaughton, Thompson, Vleck, & Batterham, 2001; Bishop, Jenkins, & Mackinnon, 1998; Brooks & Fahey, 1985; Evans, Davy, Stevenson, & Seals, 1995; Farrell, Wilmore, Coyle, Billing, Costill, 1979; Kenefick, Mattern, Mahood, & Quinn, 2002; Nicholson & Sleivert, 2001). Although some have found that work rates at lactate threshold closely reflect competitive performance (Evans et al., 1995; Nicholson & Sleivert, 2001), others have observed significant discrepancies (Bentley et al., 2001; Bishop et al., 1998; Kenefick et al., 2002). These disagreements may be the result of differences in exercise mode, discrepancies in the protocols designed to obtain blood lactate data, and the criteria used to determine lactate threshold.

Some have suggested that commonly held beliefs about the relationship between lactate production and acidosis are fundamentally flawed. In their review, Robergs, Ghasvand, and Parker (2004) point out that there are no hydrogen donors to protonate the carboxyl group of the final five molecules produced during glycolysis, meaning that the terminal molecule of anaerobic glycolysis would be produced primarily as lactate, the acid salt of lactic acid. Although free H+ from the cytosol can protonate the carboxyl group of these intermediates and lead to the formation of small amounts of lactic acid, the subsequent dissociation of the H+ to form lactate would result in no net release of protons. This evidence would dismiss lactate production as a source of metabolic acidosis and call into question the use of blood lactate response during exercise as a predictor of performance during intermediate-length, high-intensity exercise.
Previous investigations (Bentley et al., 2001; Bishop et al., 1998; Kenefick et al., 2002) have compared power outputs at lactate threshold with average power outputs during cycling time trials; however, we are unaware of any investigations that have compared time-trial power outputs with power outputs at pH threshold. We are also unaware of any studies that have compared power outputs that elicit lactate and pH thresholds. This paucity of data, the questionable relationship between lactate production and acidosis, past discrepancies between lactate threshold and performance, and the role that acidosis plays in fatigue prompted us to compare blood lactate and pH responses to progressive exercise and their relationships with high-intensity performance trials in cycle ergometry. We measured the power outputs during a simulated 20-km time trial, at blood pH threshold, at blood maximal lactate steady state, and at blood lactate threshold determined by two popular criteria, all during cycle ergometry. These data were compared to determine whether there were significant differences in the power outputs of the five variables. Because current evidence suggests no direct link between lactate production and acidosis, we wanted to determine whether there were significant differences between the power outputs at the three lactate criteria and power output at pH threshold and whether there were significant differences between the power output at the three lactate criteria and power output during the time trial. Because of a more established role of acidosis in muscle fatigue, we hypothesized that power output at pH threshold would more accurately reflect the average time-trial power output than would the power outputs of the three lactate measures.

Methods

Before participant recruitment, the procedures of this study were reviewed and approved by the institutional review boards of the University of New Mexico and the U.S. Olympic Committee, Sport Science and Coaching Division. The participants selected for this study were 13 (10 male, 3 female) trained, competitive cyclists or triathletes from the front-range region of Colorado and the Albuquerque, NM, area. Minimum standards for participation in the investigation included residence at an altitude of 1,525–2,225 m for the year before participation, 1 year experience competing in cycling time trials or nondraft triathlons, and a maximal oxygen consumption of 60 ml ∙ kg−1 ∙ min−1 for men or 50 ml ∙ time trials or nondraft triathlons, and a maximal oxygen consumption of 60 ml ∙ kg−1 ∙ min−1 for men or 50 ml ∙ kg−1 ∙ min−1 for women. After being fully informed of the procedures and potential risks of the study, each participant was required to provide written consent to signify willingness to participate. Participant characteristics (M ± SD) were as follows: age 29 ± 5 years, height 177.9 ± 7.8 cm, body mass 68.3 ± 7.2 kg, and body fat 11.6% ± 5.3%. The participants’ mean maximal oxygen consumption was 63.2 ± 5.4 ml ∙ kg−1 ∙ min−1.

Design and Procedures

Three participants performed their experimental trials in the exercise physiology laboratory at the University of New Mexico, Albuquerque, and 10 participants performed their trials in the athlete performance laboratory at the U.S. Olympic Training Center in Colorado Springs, CO. The study design required each participant to perform four exercise bouts over the course of three visits to the laboratory to determine five criteria: (a) power output at lactate threshold defined as the highest power output that did not result in consecutive and continued increases in blood lactate concentrations from exercising baseline with increases in work rate (PLT); (b) power output at lactate threshold defined as the highest power output that did not result in consecutive and continued increases of ≥1 mmol/L in blood lactate concentrations from exercising baseline with increases in work rate (PLT1); (c) power output at maximal lactate steady state, defined as the highest power output that did not result in a 21-mmol/L increase in blood lactate concentration during a 9-min, constant-load exercise session (PMLSS); (d) power output at pH threshold, defined as the highest power output that did not result in a precipitous drop in blood pH from exercising baseline with an increase in work rate (PpHT); and (e) average power output produced during a simulated 20-km time trial (PTT). Specific tests included a continuous submaximal exercise test combined with a maximal test to determine PLT, PLT1, PpHT, and maximal oxygen consumption on the first visit; a discontinuous submaximal protocol to determine PMLSS on the second visit; and a simulated 20-km time trial on the final visit. On each visit to the laboratory, the participants performed a standardized warm-up followed by the placement of a 20-g catheter into a prominent antecubital vein. Once the catheter was placed, the participant remounted the ergometer and began the exercise task. Visits were separated by at least 48 hr, and all tests were completed within a 10-day period. Each test was performed on an electronically braked bicycle ergometer (Lode Excalibur, Groningen, Holland) dynamically calibrated using a dynamometer and procedure developed by Kyle (A dynamometer for ergometer calibration, U.S. Olympic Committee/Sport Science and Technology Grant 92-CYC-001, 1992) and adjusted to the exact dimensions of each participant’s racing bicycle. The participants were allowed to self-select their cadence during their initial exercise trial and were required to maintain a consistent cadence across all trials.

Participants were required to maintain consistent diets for 24 hr immediately preceding each exercise session. Dietary manipulation during the period included consumption of at least 5 g/kg body mass of carbohydrate the day before each trial and drinking at least 1 L of water the evening before each testing day and 4 ml/kg body mass of water 1 hr before each test. The participants were approximately 4 hr postprandial at the time of each exercise test. Finally, participants were required to refrain from strenuous exercise for 24 hr before each test.
Determination of PLT, PLT1, PpHT, and Maximal Oxygen Consumption

The PLT, PLT1, and PpHT were determined from blood lactate and pH responses during the continuous, graded, submaximal exercise test. The standard protocol dictated that work rates for the test began at 125 W for men and 100 W for women; however, if a recent (≤1 month) lactate threshold test revealed a lactate threshold occurring at less than 80 W above the standard initial work rate, the test began at 80 W below the work rate of the previously determined lactate threshold. After commencement of exercise, work rates were increased in step fashion by 20 W every 4 min. During the final 30 s of each stage, a 3-ml blood sample was drawn from the catheter and immediately analyzed for pH and lactate levels. The test continued until clear inflections were seen in blood lactate and pH.

Blood samples were analyzed for lactate concentrations using a YSI 2300 Stat Plus lactate analyzer (Yellow Springs Instruments, Yellow Springs, OH) in a procedure previously described (Wilber, Zawadzki, Kearney, Shannon, & Disalvo, 1997). Blood lactate values were plotted against their respective work rates to determine the thresholds. Blood pH was measured with a novel apparatus consisting of an Orion model #8175 Thermo-electro pH meter (Orion, Beverly, MA) interfaced with a National Instruments 1000 data-acquisition system with customized National Instruments LabVIEW 7.0 analysis software (National Instruments, Austin, TX) and modified 2-ml microcentrifuge tubes that served as reservoirs for the blood samples. A small hole was drilled into the cap of each microcentrifuge tube to allow the sensor of the pH probe to be inserted into the tube. A second, smaller hole was drilled near the bottom of each microcentrifuge tube, into which a short, small-bore, luer-lock-fitted plastic tube was inserted. The luer-lock-fitted tube was held in place and the hole sealed with rubber cement. Analyses of blood samples involved placing the sensor of the pH electrode through the hole in the cap and into the microcentrifuge tube. The blood sample was then injected into the microcentrifuge tube via the small-bore plastic tube. This apparatus placed the pH electrode at a uniform depth in the sample tube and allowed for the use of a uniform sample size. The system was calibrated before each test using calibration solutions (Fisher Scientific International, Hampton, NH) with pH values of 6.0, 7.0, 7.4, and 8.0. Before each sample measurement, the sensor of the pH probe was immersed in the 7.4 pH calibration solution to establish a reference baseline for all samples. In preparation for sample measurement, the sensor was removed from the calibration solution; rinsed with distilled, deionized water; and placed in the microcentrifuge sample tube. Immediately after sample collection, the blood was injected into the microcentrifuge tube and pH was measured at a rate of 10 Hz for a period of 20 s. The values for the 20-s collection period were averaged to obtain the pH for that specific blood sample. This procedure allowed for measurement of the blood sample with minimum and uniform exposure of the sample to the atmosphere, which standardized CO₂ exchange and minimized variability of CO₂ influence on blood pH measurements. The pH threshold was determined by plotting blood pH levels against their respective power outputs.

After completing the submaximal portion of the test, the participants were given a 10-min recovery period of active rest before commencing the maximal test. Work rate for the maximal test began at the power output of the penultimate stage of the submaximal test and advanced by 25 W each minute in step fashion until volitional exhaustion. Expired air was collected throughout the test and analyzed via a ParvoMedics TrueOne 2400 metabolic cart (ParvoMedics, Sandy, UT). The flow turbine of the metabolic cart was calibrated with known volumes, and the gas analyzers were calibrated with gases of known concentration in accordance with the manufacturer’s specifications before each test. Criteria for a maximum effort included a plateau in oxygen consumption with an increase in workload, a respiratory-exchange ratio greater than 1.1, and volitional exhaustion by the participant.

Determination of PMLSS

The PMLSS was determined from data obtained during the discontinuous submaximal exercise test. A series of 9-min exercise bouts was completed, with the initial bout performed at PLT determined by the continuous submaximal test. During each bout, blood samples were collected and analyzed for blood lactate at Minutes 0, 3, 6, and 9. Between stages, the blood lactates from the previous stage were analyzed to determine whether the work rate for that stage resulted in a ≥1-mmol/L rise in blood lactate between any of the three exercising blood samples. If no such increase was observed, the participant proceeded to the next stage. The work rate was increased by 10 W on each successive stage with 20 min of active rest separating the stages. PMLSS was defined as the highest power output that could be maintained without an increase of ≥1 mmol/L in blood lactate between any of the three exercising values.

Determination of PTT

The PTT was determined by having the participants perform a simulated 20-km time trial and was calculated from the total work (kJ) performed relative to the total time (s) required to complete the task. Total work for the time trial was based on each participant’s height and weight and was calculated using an equation originally presented by Sjogaard, Nielsen, Mikkelsen, Saltin, and Burke (1984) modified to reflect current aerodynamic equipment. The ergometer was placed in the cadence-independent, “hyperbolic” mode and was interfaced with a microcomputer. Accumulated work was measured by a program written with customized National Instruments Laboratory VIEW 7.0 analysis software (National Instruments, Austin, TX) and was displayed to the
participants and investigators at all times. The participants were informed of the number of kilojoules they needed to perform to complete the time trial and were instructed to complete the work task as quickly as possible. Work rates during the trial were adjusted by the participants at their discretion, but they and the investigators were blinded to the work rates at all times. The participants were allowed to practice self-selecting the work rates during the warm-up that preceded the time trial. Blood was drawn immediately before the start of the time trial and as the participant finished one third, two thirds, and all of the prescribed work. The blood samples were immediately analyzed for blood pH and lactate concentrations. Verbal encouragement was provided throughout the trial.

**Statistical Analyses**

All statistical analyses were performed using Statistica 5 software (Statsoft, Tulsa, OK). Differences in the group means of PLT, PLT1, PMLSS, PpHT, and PTT were tested for significance using a one-way ANOVA with repeated measures and a Tukey’s post hoc test when appropriate. Pearson’s correlations were performed between PTT and PLT, PLT1, PMLSS, and PpHT. The acceptable level of significance was set a priori at \( p < .05 \) for all statistical analyses. Data are reported as group \( M \pm SD \) unless otherwise noted.

**Results**

Thirteen participants completed the required protocols of the investigation. Of these, 1 did not exhibit the required 1-mmol/L break in blood lactate necessary for establishing MLSS, 1 did not exhibit a LT1, and we were unable to determine PpHT in a third participant because of equipment malfunction. The statistical software performed case-wise deletions of these participants for the repeated-measures ANOVA. Therefore, ANOVA measures were performed on 10 participants, and other analyses involving LT1, MLSS, and pH T were performed on 12 participants. All other results are from 13 participants.

Table 1 summarizes the power outputs of the five variables. Mean PLT, PLT1, and PMLSS were all significantly lower than PTT. Only PpHT did not differ significantly from PTT. PLT, PLT1, and PMLSS were also significantly lower than PpHT. No significant differences were detected between PLT, PLT1, and PMLSS. Strong and significant correlations were found between PTT and PLT, PLT1, PMLSS, and PpHT (Table 2). Mean blood lactate values at the lactate thresholds associated with PLT (2.61 ± 0.86 mmol/L) and PLT1 (2.73 ± 0.71 mmol/L) and maximal lactate steady state (2.66 ± 0.73 mmol/L) were significantly lower than mean lactate values at pH threshold (5.58 ± 1.57 mmol/L) and at the three exercising samples during the time trial (7.45 ± 3.04, 8.87 ± 3.79, and 9.79 ± 4.22 mmol/L, for the first, second, and third samples, respectively, \( p < .05 \)). No significant changes in blood pH were observed between any of the four measurement points before and during the time trial.

**Table 1 Power Outputs of Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Power output (W), ( M \pm SD )</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLT</td>
<td>219 ± 32*#</td>
</tr>
<tr>
<td>PLT1</td>
<td>226 ± 26*#</td>
</tr>
<tr>
<td>PMLSS</td>
<td>233 ± 35*#</td>
</tr>
<tr>
<td>PpHT</td>
<td>261 ± 29</td>
</tr>
<tr>
<td>PTT</td>
<td>257 ± 31</td>
</tr>
</tbody>
</table>

*Significantly different vs. PpHT (\( p < .05 \)). #Significantly different vs. PTT (\( p < .05 \)).

**Table 2 Correlations Between Metabolic Variables and PTT**

<table>
<thead>
<tr>
<th>Variable</th>
<th>( r )</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLT</td>
<td>.97*</td>
</tr>
<tr>
<td>PLT1</td>
<td>.88*</td>
</tr>
<tr>
<td>PMLSS</td>
<td>.82*</td>
</tr>
<tr>
<td>PpHT</td>
<td>.96*</td>
</tr>
</tbody>
</table>

*Significantly different vs. PPT (\( p < .05 \)).

**Discussion**

The results revealed significant differences in the PLT, PLT1, and PMLSS compared with the PpHT and PTT. No significant difference was seen between the PpHT and the PTT. Although a number of works have compared power outputs at lactate threshold with power outputs during time trials (Bentley et al., 2001; Bishop et al., 1998; Kenefick et al., 2002), to our knowledge this is the first published work to compare the power output at pH threshold with time-trial performance in trained cyclists and triathletes. We are also unaware of any works that have compared the responses of blood lactate to blood pH during prolonged, strenuous exercise of this type.

The lower power output at the lactate thresholds than with the PTT observed in the current study mirrored those seen in previous works that used time trials of similar lengths (Bentley et al., 2001; Kenefick et al., 2002). However, in previous investigations these differences in power outputs were either less, albeit still significant, or nonexistent when the time-trial durations were increased to 90 min (Bentley et al., 2001) or 60 min (Bishop et al., 1998), respectively. We chose the 20-km distance for...
mechanisms by which acidosis contributes to fatigue, H+ man et al., 1985; Spriet, Söderlund, Bergström, & Hultand Gardiner (2004) used similar preparations of rat In contrast to these findings, Karelis, Marcil, Peronnet, untreated, and glycogenolysis, presumably through enzyme inhibi- tion while exposing the muscle to low (4.2-mM) lactate concentrations. In comparison with study force production in response to electrical stimula- tion (Chasiotis, Hultman, & Sahlin, 1982; Spriet et al., 1985) noticed a greater rate of fatigue and lower oxygen uptake in artificially stimulated rat hindquarters under respiratory and metabolic acidosis than in control conditions. In human participants, ingestion of sodium bicarbonate has been shown to attenuate exercise-induced acidosis and improve exercise performance in many investigations (Gao, Costill, Horswill, & Park, 1988; Ma, Fill, Knudson, Campbell, & Coronado, 1988; McNaught, 1992; McNaughton & Cedro, 1991), whereas others have demonstrated a reduction in work capacity after acidification of the extracellular space with NH4Cl (Hultman et al., 1985; Spriet, Söderlund, Bergström, & Hult- man, 1987). Although continued debate surrounds the mechanisms by which acidosis contributes to fatigue, H+ accumulation is suspected to reduce the rates of glycolysis and glycogenolysis, presumably through enzyme inhibition (Chasiotis, Hultman, & Sahlin, 1982; Spriet et al., 1987; Sutton, Jones, & Toews, 1981; Trivedi & Danforth, 1966); reduce cross-bridge cycling via the creation of diprotonated phosphate (Nosek, Fender, & Godt, 1987); interfere with calcium release from the sarcoplasmic reticulum (Fabiatato & Fabiato, 1978; Ma et al., 1988); reduce calcium binding to troponin (Fabiatato & Fabiato, 1978; Fuchs, Reddy, & Briggs, 1970; Gao et al., 1988); and inhibit nerve impulses at the neuromuscular junction (Landau & Nachshen, 1975; Orchardson, 1978). The effects of lactate itself on muscle fatigue and exercise performance are somewhat less clear—works that have investigated these relationships have produced conflicting results. Hogan, Gladden, Kurdak, and Poole (1995) used in situ preparations of dog gastrocnemii to study force production in response to electrical stimulation while exposing the muscle to low (4.2-mM) and high (14.4-mM) lactate concentrations. In comparison with the low-lactate preparations, the high-lactate condition resulted in significantly (15%) lower tension development in response to low-intensity electrical stimulation. In contrast to these findings, Karelis, Marcil, Peronnet, and Gardiner (2004) used similar preparations of rat plantaris muscles and found no reductions in tension development muscle when 12-mM lactate infusions were performed. Furthermore, fatigue rates over the course of 60 min of electrical stimulation were significantly reduced over control conditions when lactate infusion was used. To isolate lactate as an independent variable, both studies varied the lactate levels while pH was kept constant. Other works in this area (Andrews, Godt, & Nosek, 1996; Erdogan, Kurdak, Ergen, & Dogan, 2002; Posterino, Dutka, & Lamb, 2001) have provided mixed results and, thus, have yielded no definitive conclusions on the relationship between lactate and muscle fatigue.

The results of the current investigation do not support the contention that lactate itself causes muscle fatigue. During the time trial, participants were able to maintain power outputs that not only were significantly higher than those eliciting lactate thresholds and maximal lactate steady state but also resulted in very high concentrations of blood lactate. The participants maintained fairly consistent power outputs over the course of the time trial while exhibiting mean lactate levels of 7.45, 8.87, and 9.79 mmol/L, respectively, at the first, second, and third measurement points. Furthermore, 1 participant had blood lactate levels ranging from 11.85 after the first third of the time trial to 18.39 mmol/L at the end of the time trial. Mean power outputs for this participant were 248, 248, and 246 W for the first, second, and final thirds of the time trial, respectively. Thus, it would appear that either high lactate levels did not promote muscle fatigue or that other mechanisms, such as increased neural drive, overcame the fatigue effects of lactate so that exercise performance was minimally affected.

Our results also do not support lactate production as a contributing factor to metabolic acidosis. Not only was PpHT significantly higher than PLT, PLT1, and PMLSS, but also participants were able to maintain basal levels of blood pH during the time trial despite high and con- tinually increasing blood lactate concentrations. These observations, taken together with the maintenance of high power outputs during the time trial despite high blood lactate concentrations, call into question the role of lactate production in high-intensity exercise metabolism. Traditional dogma has suggested that lactate production limits high-intensity exercise performance and contrib- utes to metabolic acidosis through the degradation of lactic acid into lactate. In contrast to this belief, Robergs et al. (2004) have argued that when glucose is completely metabolized to lactate, there is no net release of H+ and that the production of lactate from pyruvate actually con- sumes protons. If this indeed is the case, the production of lactate could be viewed as an enabler of high-intensity exercise by buffering H+ and maintaining glycolytic ATP production rates through the maintenance of cytosolic redox potential. This viewpoint appears to be supported by the current findings, which demonstrated that basal blood pH levels and high-intensity exercise could be maintained even with high rates of lactate production.

A practical application of the current results is to help athletes and coaches determine proper training intensities.
The concept of overload training enjoys support from scientific evidence (Juel et al., 2004; Sharp, Costill, Fink, & King, 1986; Weltman et al., 1992) and practical application and suggests that the most effective way to improve physiological capacity is to train at an intensity that exceeds current capacity. The use of various measures of lactate accumulation to predict competitive ability and prescribe training intensity is widespread among coaches and athletes, as many believe that power outputs at these measures accurately reflect maximum sustainable power outputs during time-trial events. Current and previous results cast doubt on the validity of this belief and suggest that an athlete who mistakenly believes that PLT, PLT1, or PMLSS accurately reflects maximum sustainable work rates during prolonged competition may risk underestimating work-rate capacity and underprescribing training intensity. To ensure the prescription of optimal training intensities, more suitable methods of assessing maximum sustainable power output should be used by athletes and coaches. These methods may include using regression equations to predict these power outputs from PLT, assessing PpHT, and measuring power output during actual competitive time trials.

In conclusion, the current study demonstrated that there are significant differences between PLT, PLT1, PMLSS, and PpHT. Of these measures, only PpHT did not differ significantly from PTT for a 20-km time trial. Future studies might determine the relationship between PpHT and PTT for time trials of other lengths. Finally, the relationships between work outputs at PLT, PLT1, PMLSS, and PpHT and the work outputs during competitive events for other exercise modes should be investigated.

Acknowledgments

We wish to thank Rob Robergs for his assistance in developing the pH-measurement device, Randy Wilber and the U.S. Olympic Committee for the use of their physiology laboratory, and USA Cycling for their support of this investigation.

Partial funding for this project was received from the University of New Mexico Graduate and Professional Student Association.

References


