The effect of three different warm-up intensities on kayak ergometer performance

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ABSTRACT

BISHOP, D., D. BONETTI, and B. DAWSON. The effect of three different warm-up intensities on kayak ergometer performance. Med. Sci. Sports Exerc., Vol. 33, No. 6, 2001, pp. 1026–1032. Purpose: The purpose of this study was to investigate the influence of warm-up (WU) intensity on supramaximal kayak ergometer performance. Methods: In the initial testing session, eight institute of sport kayak squad members performed a graded exercise test for determination of VO_{2max} and lactate (La) parameters. In a random, counterbalanced order, subjects subsequently performed WU for 15-min at either their aerobic threshold (W1), their anaerobic threshold (W3), or mid-way between their aerobic threshold and anaerobic threshold (W2). A 5-min passive rest period and then a 2-min, all-out kayak ergometer test followed the WU. Results: For the three different WU conditions, no significant differences were observed for average power, peak VO_{2}, total VO_{2}, total VCO_{2}, or accumulated oxygen deficit (AOD) during the 2-min test. However, when compared with W3, differences in average power approached significance after both W1 (P = 0.09) and W2 (P = 0.10). Furthermore, when compared with W3, average power during the first half of the 2-min test was significantly greater after W2 (P < 0.05) and approached significance after W1 (P = 0.06). After each WU period, there was a significant difference in blood pH (W1 < W2 < W3; P < 0.05) and blood [La] (W1 < W2 < W3; P < 0.05). Despite the significantly different metabolic acidemia after each WU condition, there were no significant differences in the VO_{2} responses to the 2-min test. However, the greater metabolic acidemia after W3 was associated with impaired 2-min kayak ergometer performance. Conclusions: It was concluded, that although a degree of metabolic acidemia may be necessary to speed O_{2} kinetics, if the WU is too intense, the associated metabolic acidemia may impair supramaximal performance by reducing the anaerobic energy contribution and/or interfering with muscle contractile processes. Key Words: ACCUMULATED OXYGEN DEFICIT, METABOLIC ACIDOSIS, PRIOR EXERCISE, O_{2} KINETICS

The K1 500-m sprint kayak event (K_{500}) is contested at both Olympic and World Championship level. From a stationary start, the athlete must propel their kayak as fast as possible along a 500-m course. At international level, this event takes men approximately 100 s and women approximately 110 s. The margins of victory in this event are often very small. For example, in the 1997 Canoe women approximately 110 s. The margins of victory at both Olympic and World Championship level. The effect of three different warm-up intensities on kayak ergometer performance. Med. Sci. Sports Exerc., Vol. 33, No. 6, 2001, pp. 1026–1032. Purpose: The purpose of this study was to investigate the influence of warm-up (WU) intensity on supramaximal kayak ergometer performance. Methods: In the initial testing session, eight institute of sport kayak squad members performed a graded exercise test for determination of VO_{2max} and lactate (La) parameters. In a random, counterbalanced order, subjects subsequently performed WU for 15-min at either their aerobic threshold (W1), their anaerobic threshold (W3), or mid-way between their aerobic threshold and anaerobic threshold (W2). A 5-min passive rest period and then a 2-min, all-out kayak ergometer test followed the WU. Results: For the three different WU conditions, no significant differences were observed for average power, peak VO_{2}, total VO_{2}, total VCO_{2}, or accumulated oxygen deficit (AOD) during the 2-min test. However, when compared with W3, differences in average power approached significance after both W1 (P = 0.09) and W2 (P = 0.10). Furthermore, when compared with W3, average power during the first half of the 2-min test was significantly greater after W2 (P < 0.05) and approached significance after W1 (P = 0.06). After each WU period, there was a significant difference in blood pH (W1 < W2 < W3; P < 0.05) and blood [La] (W1 < W2 < W3; P < 0.05). Despite the significantly different metabolic acidemia after each WU condition, there were no significant differences in the VO_{2} responses to the 2-min test. However, the greater metabolic acidemia after W3 was associated with impaired 2-min kayak ergometer performance. Conclusions: It was concluded, that although a degree of metabolic acidemia may be necessary to speed O_{2} kinetics, if the WU is too intense, the associated metabolic acidemia may impair supramaximal performance by reducing the anaerobic energy contribution and/or interfering with muscle contractile processes. Key Words: ACCUMULATED OXYGEN DEFICIT, METABOLIC ACIDOSIS, PRIOR EXERCISE, O_{2} KINETICS

maximal performance (8,15). Some of these differences may be attributed to variations in the WU intensity used.

WU activity is generally proposed to improve exercise performance by serving as a preparatory stimulus for the systems involved in O_{2} transport and utilization, thereby allowing an individual to reach a high level of aerobic metabolism more quickly during the subsequent task (18). This is hypothesized to reduce the initial O_{2} deficit and allow the anaerobic system to contribute to energy supply for a greater period of time (24). This is supported by research demonstrating a speeding of O_{2} kinetics when high-intensity exercise is preceded by WU (9,23).

The influence of WU upon O_{2} kinetics appears to be intensity dependent. It has been reported that supra-threshold O_{2} kinetics are speeded by a supra-threshold (~ 80% VO_{2max}) WU but not a subthreshold (~ 50% VO_{2max}) WU (16). It was proposed that this speeding of O_{2} kinetics is due to improved perfusion of the working muscles, consequent to the vasodilatory effects of metabolic acidemia from the WU. Furthermore, these authors proposed that, despite the greater acidemia induced by the more intense WU, the speeded O_{2} kinetics may improve high-intensity performance. However, it is not known whether the optimal WU intensity for speeding O_{2} kinetics will also be the optimal intensity for improving subsequent supramaximal performance.

Although the metabolic acidemia induced by prior exercise may act to speed O_{2} kinetics, it may also act to reduce
muscle contraction force. At intensities greater than the “anaerobic threshold” (AnT) the rates of anaerobic glycolysis and subsequent lactate production are very high. The accumulation of lactate (La) results in a decrease in muscle pH that can serve to reduce or inhibit the reaction velocity of phosphofructokinase (21). There is also evidence to suggest a possible direct inhibitory effect of H⁺ on force generating mechanisms within the muscle (12). Thus, if WU is too intense, the subsequent increase in H⁺ may impair supramaximal performance via inhibition of anaerobic glycolysis and/or interference with contractile processes.

This hypothesis is supported by the results of Stewart and Sleivert (24). These authors reported that supramaximal performance was improved in moderately trained rugby players after warm-up at 60% and 70% VO₂max but not after WU at 80% VO₂max. However, these authors did not report changes in blood variables or the VO₂ response to the different WU intensities. Furthermore, percent VO₂max is not considered the best means for equating exercise intensities (13). For example, 80% O₂max may be above the AnT for some athletes (especially if untrained) but below AnT for others (e.g., elite kayak athletes) (3).

The purpose of this study therefore, was to investigate the influence of WU intensity, relative to AnT, on 2-min kayak ergometer performance in well-trained kayak athletes. It was hypothesized that while a degree of metabolic acidemia may be important to speed O₂ kinetics, if the WU intensity is too high, the subsequent metabolic acidemia may impair supramaximal performance.

METHODS

Subjects. Eight Western Australian Institute of Sport (WAIS) kayak squad members (4 women and 4 junior men) were selected on the basis of their K1 500-m time (115–125s) and willingness to participate in the study. Their mean (± SD) age was 21 ± 3 y and mass 72.2 ± 7.4 kg. They were all familiar with exercising on the kayak ergometer and were currently actively participating in the WAIS kayak squad’s training and physiological testing program. During the time of the study, all subjects were encouraged to undertake their normal training and diet but to not train on the day before each test. They were instructed to be adequately hydrated and to have not eaten for 3 h before each test.

Experimental overview. After being fully informed of the risks associated with participation, each subject gave their written consent. The testing procedures were approved by the research Ethics Committee of The Western Australian Institute of Sport. The testing took place over a 2-wk period. For each subject, all tests were conducted at the same time of day and separated by at least 48 h.

In the initial testing session, each subject performed a graded exercise test (GXT) for determination of maximal oxygen uptake (VO₂max) and lactate parameters. In a random, counterbalanced order, each subject subsequently performed WU for 15 min at either the aerobic threshold (W1), the anaerobic threshold (W3), or mid-way between the aerobic threshold and anaerobic threshold (W2; sessions 2–4).

The WU was followed by a 5-min passive rest period and then a 2-min, all-out kayak ergometer test (Fig. 1).

Kayak ergometer. All physiological testing was conducted on a calibrated, wind-braked kayak ergometer (K1 Ergo, Garran, Australia). The foot-bar position of the kayak ergometer was adjusted to resemble the paddler’s own kayak before each test. The ergometer was interfaced with a computer that continuously measured, calculated, and stored accumulated work and other associated work indices using specifically designed software.

Heart rate. A Polar heart rate monitor (Polar Vantage NV, Kempele, Finland) was used to monitor and store heart rate every 5 s during the physiological tests.

Blood analysis. Arterialized capillary blood (100 μL) was sampled from a hyaeremic earlobe. Hyperemia was induced by smearing the earlobe with a cutaneous vasodilator (Finalgon, Boehringer Ingelheim) 10 min before the start of each test. Capillary blood samples were taken 5-min before the WU period, 1-min after the WU, and 1, 4, and 7 min after the 2-min test. Whole blood lactate concentration was determined using a Micro Stat LM3 (Analox Instruments Ltd., London, UK). The Micro Stat was regularly calibrated using precision standards and was routinely assessed by external quality control. Whole blood pH was determined using a Ciba Corning blood gas analyser (#865, Chiron Diagnostics, Walpole, MA).

Gas measurements. During both the GXT and the 2-min test, expired gas samples were continuously monitored for O₂ and CO₂ concentrations using Ametek gas analysers (SOV S-3A and COV CD3A, respectively; Pittsburgh, PA). Data were then averaged over 15-s intervals. Ventilation (V̇E) was also recorded every 15 s using a turbine ventilometer (Morgan, Model 096, Kent, UK). The gas analysers were calibrated immediately before and verified after each test using three certified gravimetric alpha-grade gas mixtures (BOC Gases, Chatswood, Australia); the ventilometer was calibrated preexercise and verified postexercise using a 1-L syringe in accordance with the manufacturer’s instructions.

Graded exercise test. A GXT was used to determine both VO₂max and the AnT. The GXT commenced at an initial workload of 55 W and increments of 15 W were

![FIGURE 1—Experimental testing protocol for sessions 2–4.](image-url)
applied at 5-min intervals until exhaustion. A 1-min rest period between each increment allowed for the sampling of capillary blood. The GXT continued until the athlete could no longer maintain the required power output. The VO₂ values for the last 2 min of each 5-min step were recorded and used to determine, for each subject, the linear relationship between VO₂ and power output. The highest four consecutive 15-s VO₂ values were summed to determine VO₂max. It has been suggested that, for valid VO₂max results, that test duration should not exceed 12 min (25). However, while the GXT did exceed 12 min, previous research has shown that the length of the test does not appear to influence VO₂max (4). The AnT was calculated using the modified Dmax method (LT Mod; ref. 5). The LT Mod was determined by the point on the polynomial regression curve that yielded the maximal perpendicular distance to the straight line formed by the lactate threshold (first increase in lactate concentration above the resting level (26) and the final lactate point).

Two-minute test. After first performing either W1, W2, or W3, subjects completed a 2-min all-out test on the kayak ergometer. This test length was chosen based on research reporting the validity of an all-out procedure for estimating maximal AOD (14) and research indicating that AOD is maximized when the test duration is similar to that of the criterion event (7). Individual relationships between the oxygen cost and power output were established during the last 2 min of each submaximal stage during the GXT. The estimated oxygen cost for the 2-min all-out test was then determined by extrapolation (14). The AOD was then calculated as the difference between the estimated oxygen cost of exercise and the actual oxygen uptake. Average power output, estimated oxygen cost of exercise and actual oxygen uptake were calculated over 15-s intervals. The oxygen deficit for each 15-s interval was accumulated over time to give AOD.

Statistical analysis. The data were analyzed to determine whether any significant differences existed between the 2-min test results after the three different WU conditions for any of the dependent measures taken. Analysis of the means of the data for average power, peak power, VO₂max, total VO₂, total VCO₂, aerobic contribution, AOD, peak blood lactate, and blood pH during the 2-min tests were conducted using a one-way ANOVA with repeated measures for WU intensity. Blood lactate, blood pH, and heart rates during the WU period were also analyzed using the same procedure. Where appropriate, post hoc comparisons were employed. Statistical significance was set at the P < 0.05 level of probability. All statistical analyses were conducted using the SPSS statistical package (version 8.0, SPSS, Chicago, IL).

RESULTS

Power output. Group results for the average power and peak power output recorded during each 2-min test, after the three different WU conditions, are summarized in Table 1. There were no significant differences in average power output or peak power output after the three WU conditions (P > 0.05). However, when average power output was expressed in 15-s intervals (Fig. 2), it was significantly greater after W2 than after W3 in the second 15-s interval.

![FIGURE 2—Mean (± SD) for average power recorded every 15 s during the 2-min performance tests. * P < 0.05; # P < 0.10.](http://www.acsm-msse.org)
In the third 15-s interval, average power was significantly greater after W1 than after W3 \( (P < 0.05) \). In the final 15-s interval of the 2-min test, average power was significantly greater after W3 than after W2 \( (P < 0.05) \). In the first and third 15-s intervals, the difference between average power output after W2 and W3 approached significance \( (P = 0.07 \text{ and } P = 0.06, \text{ respectively}) \). When average power was examined in the first and second half of the 2-min tests, it was found to be significantly greater after W2 than after W3 \( (P < 0.05) \) in the first half of the 2-min test. The difference between average power output in the first half of the 2-min test also approached significance between W1 and W3 \( (P = 0.06) \).

**Metabolic variables.** No significant differences were found between WU conditions for measures of peak \( \dot{V}O_2 \), total \( \dot{V}O_2 \), aerobic energy contribution, AOD, or total \( \dot{V}CO_2 \) during the 2-min test (Table 2). Mean \( \dot{V}O_2 \) utilized every 15 s in the 2-min tests is illustrated in Figure 3. There were no significant differences between WU conditions at any 15-s interval \( (P > 0.05) \). When AOD was expressed in 15-s intervals, it was significantly greater after W2 than after W3 \( (P < 0.05) \) in the fifth 15-s interval (Fig. 4).

**[La].** Blood lactate concentration was significantly greater than resting blood [La] after all three WU conditions and was significantly different after each of the WU conditions (Table 3; W3 > W2 > W1; \( P < 0.05) \). Peak blood [La] after the 2-min performance test was significantly greater after W3 than after W1 (Fig. 5). However, changes in [La] (post WU to peak) were significantly less after W3 when compared with W1 and W2.

**Blood pH.** After the 15-min WU, blood pH was significantly lower than resting blood pH after both W2 and W3 and was significantly different after all three WU conditions (Table 3; W3 > W2 > W1; \( P < 0.05) \). However, at the conclusion of the 2-min performance tests, blood pH values were not significantly different \( (P < 0.05) \).

**Heart rate.** Mean heart rates were significantly different after all three WU conditions. (Table 3; W3 > W2 > W1; \( P < 0.05) \).

**DISCUSSION**

The results of this study have demonstrated that, when the rest period is fixed, the choice of WU intensity can have a significant influence on sprint kayak ergometer performance. Although average power output was not significantly different over the entire 2-min test, when compared with W3, average power output during the first 60 s of the 2-min test was significantly greater after W2 and approached significance after W1. Furthermore, in the first three 15-s intervals, differences in average power output after W2 and W3 approached or exceeded significance. Thus, although there was no significant difference in average power output after W1 (\( \sim 55\% \dot{V}O_2\text{max} \)) or W2 (\( \sim 65\% \dot{V}O_2\text{max} \)), kayak ergometer performance during the first 60 s was impaired after W3 (\( \sim 75\% \dot{V}O_2\text{max} \)).

The results of this study are similar to those reported in a previous study by Stewart and Sleivert (24). These authors reported that, compared with no WU, there was a significant increase in treadmill time to exhaustion (63–72 s), when running was preceded by a 15-min WU at either 60 or 70% \( \dot{V}O_2\text{max} \). However, time to exhaustion was significantly less when preceded by a 15-min WU at 80% \( \dot{V}O_2\text{max} \). There was

![FIGURE 3—Mean (± SD) for \( O_2 \) utilized every 15 s during the 2-min tests.](image-url)
no significant difference in time to exhaustion when WU was performed at 60 or 70% VO_2max.

Although the present results followed a similar pattern to those of Stewart and Sleivert (24), we were unable to demonstrate a significant effect of WU intensity on 2-min performance. This may be attributed to the large variability in ergometer performance, which diminished the power of the ANOVA to detect differences, and study-design differences. For example, in contrast to the previous study (24), we elected not to include a no WU condition. Thus, we could not verify the significant improvement in supramaximal performance after a moderate-intensity WU, when compared with no WU, as reported by Stewart and Sleivert (24). It was felt that the high stresses placed on the shoulder joint, particularly when commencing the all-out ergometer test, constituted an unnecessary injury risk to our national level athletes, in the absence of a WU.

Another difference between the two studies was that our most intense WU (W3, ~ 75% VO_2max) was not as intense as the most intense WU used by Stewart and Sleivert (24) (80% VO_2max). This becomes more apparent when one considers that 80% VO_2max is likely to be well above the AnT in the moderately trained subjects recruited by Stewart and Sleivert (24). Febbraio et al. (13) reported that the AnT corresponded to ~ 74% of VO_2max in endurance-trained subjects and only ~ 57% of VO_2max in untrained subjects. Thus, the choice of a slightly more intense WU by Stewart and Sleivert (24), especially relative to the AnT of their subjects, may have increased the likelihood of a significant decrease in performance.

Although there was no significant effect of WU intensity on 2-min performance, there were significant differences between WU conditions in the present study when performance was compared over a similar time frame to that of Stewart and Sleivert (24). Average power output in the first 60 s of the 2-min test was significantly less after W3 than after W2. The significantly impaired first half performance can possibly be attributed to the significantly greater metabolic acidemia after W3 (Table 3; Fig. 5). It has previously been reported that there is a significant correlation between venous and muscle pH and that there is a similar pattern of response between venous and muscle pH (1). Therefore, assuming that changes in blood pH reflected changes in muscle pH, the significant decrease in blood pH after W3 may have been associated with the impairment of a number of contractile mechanisms.

An increase in H^+ ion concentration may inhibit the release and uptake of Ca^{2+} and compete with Ca^{2+} for the binding sites on actomyosin (12). This would result in a decrease in the number of active cross bridges, causing a decline in contraction intensity and a decrease in supramaximal performance. In addition, the activity of PFK, the rate limiting enzyme in glycolysis (17), has been shown to be reduced or inhibited by a decrease in pH (21). Thus, the greater metabolic acidemia induced by the more intense WU may have contributed to the significantly impaired first-half performance after W3 via interference with muscle contractile processes and/or inhibition of anaerobic glycolysis.

An impaired anaerobic contribution, consequent to the metabolic acidemia, is supported by the significantly reduced lactate production and the tendency for both AOD and VCO_2 to be reduced after W3 (Table 2, Fig. 4). Although not significant, it is interesting to note that the reduction in AOD appeared to be greatest during the first half of the 2-min test when differences in average power output after W2 an W3 were significantly different. Gerbino et al. (16) have suggested that a smaller increase in blood [La] was consistent with the proposal that the requirement for increased lactate production during constant-load, high-intensity exercise is less when the O_2 kinetics are speeded by prior exercise. However, as there was no significant difference in the VO_2 response in the present study,

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**TABLE 3. Mean (± SD) values for [La], blood pH and heart rate after each WU condition.**

<table>
<thead>
<tr>
<th>WU Condition</th>
<th>Blood Lactate (mmol/L)</th>
<th>Blood pH</th>
<th>Heart Rate (b/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>W1</td>
<td>1.5 ± 0.4^a</td>
<td>7.40 ± 0.01</td>
<td>144 ± 7^a</td>
</tr>
<tr>
<td>W2</td>
<td>2.2 ± 0.6^a,b</td>
<td>7.38 ± 0.02^a,b</td>
<td>158 ± 10^a,b</td>
</tr>
<tr>
<td>W3</td>
<td>5.1 ± 1.4^a,b,c</td>
<td>7.35 ± 0.02^a,b,c</td>
<td>169 ± 9^a,b,c</td>
</tr>
</tbody>
</table>

^a Significantly different from rest, P < 0.05; ^b significantly different from W1, P < 0.05; ^c significantly different from W1 and W2, P < 0.05.

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FIGURE 4—Mean (± SD) AOD recorded every 15 s during the 2-min tests. * P < 0.05.

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http://www.acsm-msse.org
it doesn’t appear that the decreased lactate production can be attributed to a speeding of O$_2$ kinetics during supramaximal exercise.

Despite the first-half differences, average power output was not significantly different during the second half of the 2-min test. It has previously been reported that 90% of the peak [La] produced during a 6-min, all-out rowing ergometer test is produced during the 1st minute of the test (19). Assuming a similar response to the 2-min, all-out test in the present study, it is likely that the [La] did not increase markedly after the 1st minute of the kayak ergometer test. Therefore, at the start of the second half of the 2-min test, acid-base balance may have been similar after all three WU conditions. Consequently, a similar metabolic acidemia may explain why average power output, during the second half of the 2-min test, was not significantly different after all three WU conditions.

The residual acidemia after WU has previously been proposed to lead to an improved muscle perfusion during exercise, an improved capillary-to-mitochondria diffusional gradient for O$_2$ and a speeding of O$_2$ kinetics (16). Although we were unable to measure O$_2$ kinetics, it would be expected that an increase in O$_2$ kinetics would be accompanied by an increase in total VO$_2$. However, in the present study, there were no significant differences between WU conditions for total VO$_2$ or peak VO$_2$, despite W3 inducing a similar [La] (~ 5 mmol·L$^{-1}$) to that reported by Gerbino et al. (16). Therefore, in contrast to results reported for submaximal exercise (16), a greater residual acidemia did not appear to speed the O$_2$ kinetics associated with supramaximal exercise in the present study.

As the O$_2$ kinetics associated with supramaximal exercise have not, to our knowledge, previously been described, it is difficult to speculate why there did not appear to be a speeding of O$_2$ kinetics in the present study. Previous studies reporting an increase in O$_2$ kinetics after WU exercise have typically not used well-trained subjects (10,16,23). Therefore, as O$_2$ kinetics have been shown to be faster in individuals possessing higher levels of aerobic fitness (6,11), it may be more difficult to speed the O$_2$ kinetics of aerobically fit athletes whose O$_2$ kinetics may be close to their physiological maximum. This is supported by the observation that, even in the absence of prior WU, the O$_2$ kinetics of trained athletes (2) are considerably faster than the O$_2$ kinetics of untrained athletes after WU (16).

In summary, the results of this study have demonstrated that, when the rest period is fixed, the choice of WU intensity can have a significant influence on supramaximal kayak ergometer performance. Although there was no significant difference between W1 (~ 55% VO$_{2\text{max}}$) and W2 (~ 65% VO$_{2\text{max}}$), kayak ergometer performance during the first 60 s was impaired after W3 (~ 75% VO$_{2\text{max}}$). Despite a significantly greater metabolic acidemia after each WU condition, there was no significant difference in the VO$_2$ response. Furthermore, the greater metabolic acidemia after W3 was associated with impaired kayak ergometer performance. It is concluded, that although a degree of metabolic acidemia may be necessary to speed O$_2$ kinetics, if the WU is too intense, the associated metabolic acidemia may impair supramaximal performance by reducing the anaerobic energy contribution and/or interfering with muscle contractile processes.

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