

Comparison of effects of two interval-training programmes on lactate and ventilatory thresholds

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Twenty-one women subjects were matched in terms of their $\dot{V}O_{2\max}$ and assigned to one of two groups: (1) training at 30 s; or (2) 2 min with a 1:1 work: relief ratio (1:1 WR) before participating in a 7-week training programme which began at an intensity of 85% $\dot{V}O_{2\max}$ and increased 5% every two weeks (90% and 95% $\dot{V}O_{2\max}$). The subjects trained to exhaustion four times per week. Maximal oxygen consumption ($\dot{V}O_{2\max}$), lactate threshold (T_{lac}) and ventilatory threshold (T_{vent}) were determined before and after the training programme. After training, there were significant increases ($P < 0.05$) in $\dot{V}O_{2\max}$ (5% and 6%), T_{lac} (19.4% and 22.4%), and T_{vent} (19.5% and 18.5%). There were no significant group differences on any dependent measure but this research adds support to previous training studies in that a strong correlation ($P < 0.05$) between T_{lac} and T_{vent} is maintained from before to after the test. It was concluded that both formats of high intensity aerobic interval-training produce similar changes in $\dot{V}O_{2\max}$, T_{lac} and T_{vent} and that these changes appear to be independent of the length of the work interval.

Keywords: Aerobic interval-training, lactate threshold ventilatory threshold

Introduction

The intensity and duration of exercise will determine the production and accumulation of lactate^{1,2}. By altering the intensity of exercise and the work:rest ratio (WR), the contribution of anaerobic and aerobic metabolism to total energy needs may be manipulated³. Skeletal muscle adapts to aerobic interval training (1:1 WR) with an increase in the capacity for aerobic metabolism and a decrease in lactate production⁴. But, as the intensity of interval work (1:1 WR) approaches and exceeds 100% $\dot{V}O_{2\max}$, the anaerobically generated acidosis becomes a major factor in contributing to muscular fatigue⁵.

The presence of lactate has been proposed to have a cause and effect relationship with T_{vent} due to the carbon dioxide produced as a consequence of lactate buffering by the bicarbonate system⁶. Research evidence concerning this relationship is contradictory. Simon and colleagues⁷ have reported that T_{vent} occurs before T_{lac} whereas Walsh and Bannister⁸

described the opposite sequence of events. Our study employing two forms of high-intensity aerobic interval-training was therefore designed to clarify the proposed cause and effect relationship between T_{lac} and T_{vent} . In addition, the metabolic responses of women subjects to two different interval training regimens were evaluated.

Subjects and methods

Informed consent was obtained from 21 women physical education students from Lakehead University to participate in the study. The group was pre-tested to establish baseline performance values for $\dot{V}O_{2\max}$, T_{lac} , T_{vent} and also to ascertain individual training work loads. Determination of $\dot{V}O_{2\max}$, T_{lac} and T_{vent} were performed on the same day.

Measurement of $\dot{V}O_2$

The determination of $\dot{V}O_{2\max}$ was performed in accordance with the procedures outlined by Hoppeler and colleagues⁹ and Tesch and colleagues¹⁰ on a Monark bicycle ergometer (Monark, Varberg, Sweden) at a cadence of 60 r.p.m. The subjects underwent a 4-min unloaded warm up at 0W. Thereafter, an increase in resistance of 30W was applied to the ergometer. This load was maintained for 2 min and was subsequently increased by 30W every 2 min until $\dot{V}O_{2\max}$ had been achieved^{9,10}. Gas analysis was carried out using a pre-calibrated Beckman MMC Horizon II system (Beckman Instruments, Anaheim, California, USA). Heart rate was monitored employing a four lead electrocardiogram interfaced to the Beckman system. During the cycling test, blood was drawn into potassium oxalate/sodium fluoride vacuum tubes by an indwelling catheter from the antecubital vein. While exercising, blood samples were taken every 30 s until a heart rate of 190 beats min^{-1} was recorded. Blood was then drawn every 60 s until the cessation of exercise. At the conclusion of the test, blood was taken immediately and the plasma analysed using a Yellow Springs 23L Lactate analyser (YSI, Yellow Springs, Ohio, USA). T_{lac} was determined using the double logarithm method of Beaver and colleagues¹¹.

Ventilatory threshold was resolved using the $\dot{V}O_{2\max}$ data obtained from the Beckman metabolic cart before and after testing. Calculation of T_{vent} employed the \dot{V} -slope method ($\dot{V}_{\text{CO}_2}/\dot{V}_{\text{O}_2}$) adapted from Wasserman and colleagues¹².

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0306-3674/94/010018-04

Table 1. Pre-training values* for subject characteristics – subjects were matched according to their $\dot{V}O_{2\max}$ and placed into one of two training groups

Group	$\dot{V}O_{2\max}$ (ml kg ⁻¹ min ⁻¹)	Age (years)	Height (cm)	Weight (kg)
Group 1 (n = 10)	40.0(1.7)	19.9(0.4)	16.5(1.4)	60.5(1.6)
Group 2 (n = 11)	39.9(1.5)	20.8(0.7)	16.5(1.3)	64.8(3.8)

*Values are mean(s.e.)

The training programme

After testing for baseline values, subjects were matched in terms of their $\dot{V}O_{2\max}$ and placed in one of two training groups to achieve homogeneity between groups. Subject characteristics for both groups are listed in Table 1. The groups consisted of: an interval-training group with a 30 s work:30 s rest ratio (group 1) and an interval-training group with a 2-min work:2-min rest ratio (group 2). Participants attended four sessions per week for a period of 7 weeks (28 training sessions). During these periods subjects cycled to voluntary exhaustion on a Monark ergometer at a cadence of 60 r.p.m. against a predetermined resistance. During the first week (conditioning period) of the training programme subjects cycled at the workload which elicited T_{lac} . For the next 2 weeks, subjects exercised at a workload set to 85% $\dot{V}O_{2\max}$. During weeks 4 and 5 of the training programme, the work load was established at 90% $\dot{V}O_{2\max}$. For the final two weeks, the workload was further increased to 95% $\dot{V}O_{2\max}$. When the programme was completed, subjects were tested using the identical protocol to that used before the test.

Statistical analysis

The dependent measures, $\dot{V}O_{2\max}$, T_{lac} and T_{vent} were analysed using a 2 (group) \times 2(test) analysis of variance with repeated measures on the last factor. The Pearson product moment correlation test was used to determine the relationship between T_{lac} and T_{vent} values before and after testing. Differences for all dependent measures were considered significant at the $P < 0.05$ level. The test analyses were carried out employing the SPSS/PC+V3 software package. (Statistical Package for the Social Sciences, SPSS, Chicago, Illinois, USA).

Results

Descriptive data of the physical and physiological characteristics of the subjects before training in both training groups are presented in Table 1. There were no significant differences between groups before the start of training for any variables.

After the 7-week training programme, both groups improved significantly in $\dot{V}O_{2\max}$. However no significant differences between groups were noted over time (Figure 1).

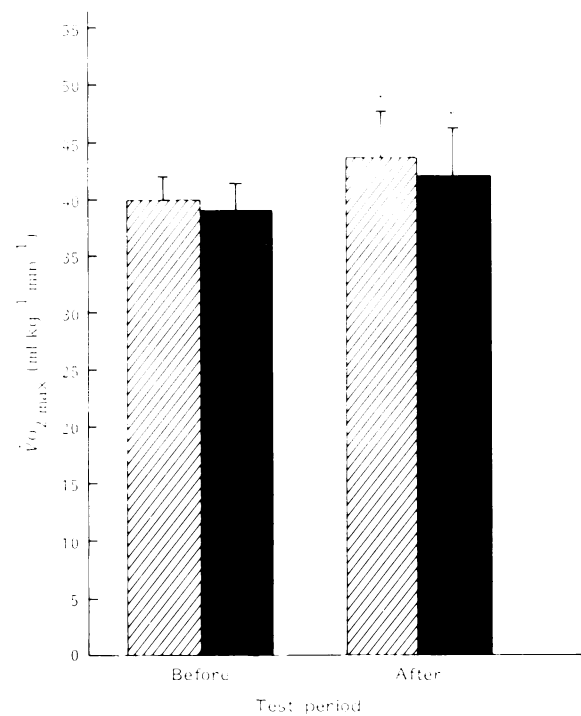


Figure 1. Mean (s.e.) changes in $\dot{V}O_{2\max}$ for group 1 (training at 30-s work: 30-s rest) and group 2 (training at 2-min work: 2-min rest) from before to after the test. No significant differences between groups were noted over time. * Within group significance for the two groups $P < 0.05$; analysis of variance. \square , group 1; \blacksquare , group 2

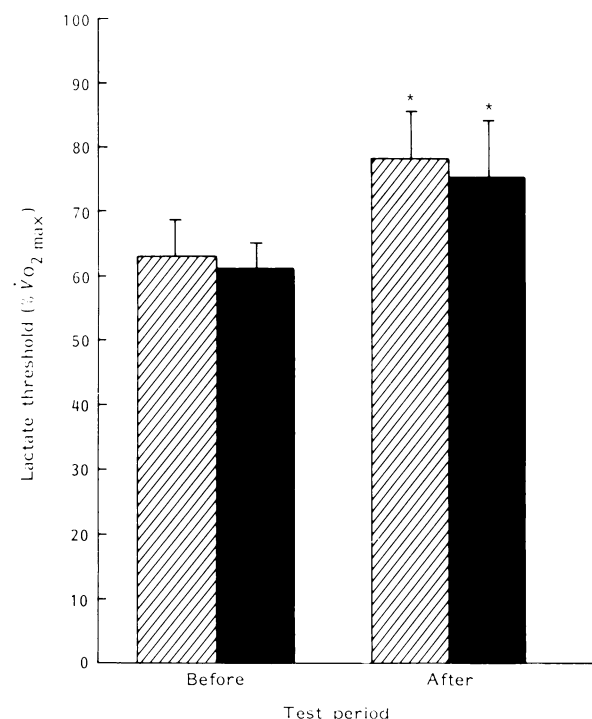


Figure 2. Mean (s.e.) changes in lactate threshold for group 1 (training at 30-s work: 30-s rest) and group 2 (training at 2-min work: 2-min rest) from before to after the test. No significant differences were noted between groups over time. *Within group significance for both groups $P < 0.05$; Pearson product moment correlation coefficient. \square , group 1; \blacksquare , group 2

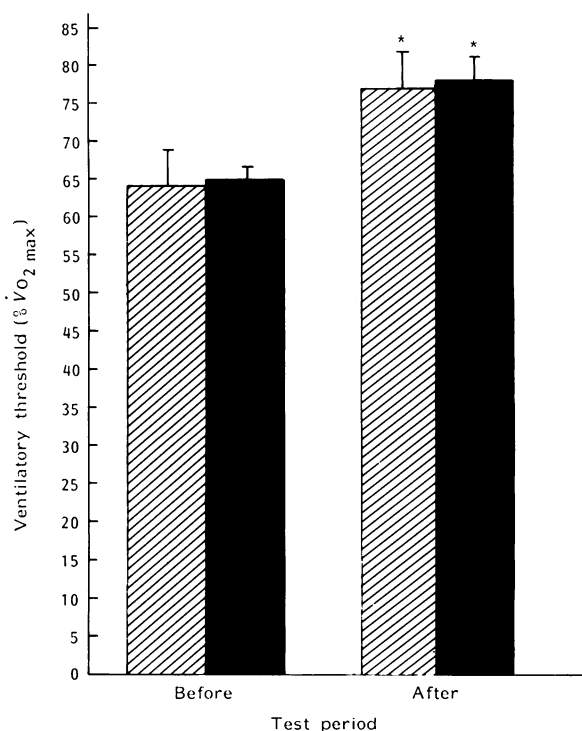


Figure 3. Mean (s.e.) changes in ventilatory threshold for group 1 (training at 30-s work: 30-s rest) and group 2 (training at 2-min work: 2-min rest) from before to after the test. No significant differences were noted between groups over time. *Within group significance for both groups $P < 0.05$; analysis of variance. ▨, group 1; ■, group 2

Table 2. Correlation between T_{lac} and T_{vent} before and after the test

Group	Condition	Correlation*
Group 1	Before	0.83
	After	0.85
Group 2	Before	0.94
	After	0.97

*All values were significant at $P < 0.05$; Pearson product moment correlation coefficient

Values for T_{lac} (Figure 2) and T_{vent} (Figure 3) after training are expressed as percentages of the post-training $\dot{V}O_{2max}$. Changes in T_{lac} and T_{vent} after training were statistically significant within groups, but not between groups (Figures 2 and 3). There were significant correlations between T_{lac} and T_{vent} before and after training for both groups 1 and 2 (Table 2).

Discussion

The increase in $\dot{V}O_{2max}$ demonstrated by both groups in the present investigation (Figure 1) is in agreement with other interval training studies which employed similar training intensities of 80–95% $\dot{V}O_{2max}$ ^{13,14}. As well, research by MacDougall and Sale⁵ revealed that the greatest changes in aerobic capacity will occur when the work intensity exceeds 90% $\dot{V}O_{2max}$.

Previous investigators have shown that the length of the work interval appears to affect the magnitude of the aerobic training response. Daniels and Scardina¹⁵ suggested that exercise bouts lasting approximately 3–5 min are considered optimal for training the aerobic energy system. While Astrand and colleagues¹ advised that a 2-min work:2-min relief interval was considered more appropriate. As well, MacDougall and Sale⁵ reported that a work period of 30s followed by a 30-s rest interval would be inadequate in stressing aerobic capacity as compared with longer 2–3 min work periods. The longer 2–3 min work intervals would provide a greater degree of hypoxia. Furthermore, both the alactate and lactate energy systems would appear to be used to a greater extent with the 30s as opposed to the 2-min work interval⁵. However, the results of the present research contradict their findings as no significant differences in aerobic capacity were revealed between the 30-s and 2-min interval-training groups.

Previously, it has been shown that the greater intensity of exercise associated with aerobic interval-training yields more pronounced peripheral than central adaptations⁵. Increases in mitochondrial volume^{9,16}, increased capillarization of muscle¹⁷, and an elevation in the concentration of the enzymes of both β - and end-terminal oxidation¹⁸ provide the mechanism for a delay in lactate production after this form of training¹⁹ and may account for the observed 19.4% (group 1) and 22.4% (group 2) increase in T_{lac} realized in this study. Formerly, it has been argued that extensive local adjustments in the oxidative potential of skeletal muscle can occur without a dramatic increase in $\dot{V}O_{2max}$ ²⁰. In our study there was a modest, but significant rise in $\dot{V}O_{2max}$ of 6% (group 1) and 5% (group 2) associated with the more dramatic elevation in both T_{lac} and T_{vent} . The smaller volume and greater exercise intensity with this type of training may account for these differences.

In the present research, T_{vent} and $\dot{V}O_{2max}$ both increased significantly after the aerobic interval training programme (Figure 3) and this is in agreement with previous work^{21,22}. However our data do not agree with the results of Acevedo and Goldfarb²³ who revealed that $\dot{V}O_{2max}$ and T_{vent} did not change in spite of lower lactate levels at greater exercise intensities. Perhaps the conflicting findings may be due to the fact that the training programme employed by Acevedo and Goldfarb²³ failed to provide the necessary training stimulus to promote a change in $\dot{V}O_{2max}$ in their well-conditioned subjects.

The hydrogen ion efflux from the muscle cell associated with lactate production is immediately buffered by the bicarbonate system¹⁹ and leads to the formation of carbon dioxide which is rapidly transferred to the lungs. Consequently, the carbon dioxide produced may be measured at the mouth and expressed in relation to $\dot{V}O_2$ as $\dot{V}CO_2/\dot{V}O_2$. With the increase in the oxidative capacity of trained muscle, T_{lac} is achieved at a greater percentage of $\dot{V}O_{2max}$. Below T_{lac} , lactate concentration for a given workload will be decreased after training and results in decreased bicarbonate buffering with the subsequent reduction in metabolically produced carbon dioxide.

Our results revealed significant correlations between T_{lac} and T_{vent} both before and after the test (Table 2) and lends support to the cause and effect relationship between lactate formation and T_{vent} previously described^{6, 24, 25}. The strong correlation has been explained on the basis of the increased carbon dioxide produced as a result of lactate buffering by the bicarbonate system^{6, 24, 25}. Furthermore Farrel and Ivy²⁶ reasoned that lactate not only stimulates ventilation via increased carbon dioxide output, but also as a result of lactate being almost completely dissociated at physiological pH²⁴. However this reported robust relationship between T_{lac} and T_{vent} has been questioned in the research literature^{23, 26, 27} and other mechanisms may therefore exist to produce the characteristic responses to incremental exercise. Further research is necessary to define the relationship between these two metabolic measures more clearly.

In summary, our results clearly demonstrate that changes in $\dot{V}O_{2max}$, T_{lac} and T_{vent} were not solely dependent on the duration of the work interval as both training groups experienced similar increases in the dependent measures. A finding unique to this study is the marked improvement in T_{lac} and T_{vent} , concomitant with only modest changes in $\dot{V}O_{2max}$. Perhaps, the greater peripheral than central adaptation associated with this type of training²⁸ may account for our findings. Finally, this investigation, in reporting a strong correlation between T_{lac} and T_{vent} before and after testing, is in agreement with previous studies which employed a similar form of training.

Acknowledgements

The authors would like to acknowledge the assistance of Mr Carlos Zerpa for his technical support. This research was supported in part by funds to Lakehead University from NSERC to Dr R. Thayer.

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