Child-adult differences in whole blood lactate responses to incremental treadmill exercise

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The aim of this study was to evaluate whether fixed blood lactate reference values of 2.5 and 4.0 mmol L⁻¹ corresponded to the equivalent intensity of exercise in prepubertal and teenage boys, and men. Twenty six prepubertal boys (mean(sd) age 11.1(0.4) years), 26 teenage boys (mean(sd) age 14.1(0.3) years), and 23 men (mean(sd) age 22.4(2.7) years) gave informed consent to participate in the study. Oxygen consumption (VO₂) and heart rates (HR) corresponding to the 2.5 and 4.0 mmol L⁻¹ fixed blood lactate reference values were used as the criterion measures during incremental treadmill exercise. At the 2.5 mmol L⁻¹ level there were no significant differences (P > 0.05) in % peak VO₂ between groups. For both prepubertal and teenage boys the 4.0 mmol L⁻¹ lactate level represented a higher mean % peak VO₂ than for the men (P < 0.05). The prepubertal and teenage values were again not significantly different (P > 0.05). Factors other than maturation during puberty influence blood lactate responses to exercise. (Br J Sports Med 1995; 29: 196–199)

Keywords: blood lactate; prepubertal; teenage; men; boy

The use of the 4.0 mmol L⁻¹ fixed blood lactate reference value to monitor and assess endurance performance in adults has received considerable attention. Although suitable for use with adults, the 4.0 mmol L⁻¹ fixed blood lactate reference value may not be appropriate for children because it corresponds to an almost maximal, rather than submaximal, effort. This finding is, however, based on only a few studies, all of which differ methodologically. The maximum lactate steady state (MLaSS), the point of equilibrium between lactate production and removal, and 4.0 mmol L⁻¹ lactate do not correlate highly in children. However, oxygen consumption (VO₂) and heart rate (HR) corresponding to 2.5 mmol L⁻¹ in children are not significantly different from those measured at the MLaSS. For this reason, 2.5 mmol L⁻¹ lactate in children may be used in a similar manner to the 4.0 mmol L⁻¹ value in adults.

Direct comparisons of VO₂ and heart rate responses corresponding to the 2.5 whole blood lactate values between children and adults do not appear to be available. Studies designed to compare children and adult physiological responses at the 4.0 mmol L⁻¹ level have only measured heart rate, and not VO₂. Inter-study comparison between children and adults at submaximal exercise intensities are difficult due to differences in protocol, lactate sampling and assay techniques.

Only one study to date has examined lactate responses to exercise with progression through the five stages of maturation identified by Tanner; Williams and Armstrong failed to identify any significant differences in lactate response across the maturity groups. Conversely, using Tanner’s indices, Wirth et al. classified 25 boys into prepubertal, pubertal, and postpubertal groups. They reported that blood lactate concentrations following cycling exercise at 70% peak VO₂ increased significantly with increasing maturity. The influence of maturation on children’s blood lactate response to exercise is equivocal.

The aim of this study was to evaluate whether fixed whole blood lactate reference values of 2.5 and 4.0 mmol L⁻¹ correspond to the equivalent level of exercise in children and adults; and also to see if differences existed between groups of boys classified as prepubertal and teenage. This information could be important when recommending optimal training intensities and evaluating children’s exercise endurance capacity.

Methods

Subjects

Twenty six prepubertal boys, mean(sd) age 11.1(0.4) years, and 26 teenage boys, mean(sd) age 14.1(0.3) years, were pair matched on the basis of mass related peak VO₂. An adult group of 23 men, mean(sd) age 22.4(2.7) years, with similar mass related peak VO₂ scores were drawn from an active group of university students. Institutional ethics approval for the project and written informed consent from all subjects, including parental/guardian consent for the boys, was obtained. Mean physical characteristics for each subgroup are shown in Table 1. Subjects were familiar with all test procedures before actual testing.

Measurement of peak VO₂

Peak VO₂ was determined via a discontinuous, incremental treadmill test, comprising 3 min stages of increasing intensity. Following a 5 min warm up the groups of subjects began the tests at the following belt.

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Child-adult differences in lactate response to exercise: K Tolfrey, N Armstrong

Table 1. Physical and physiological characteristics of the subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Prepubertal (n = 26)</th>
<th>Teenage (n = 26)</th>
<th>Adult (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>11.10(4)*</td>
<td>14.10(3)*</td>
<td>22.4(2.7)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.440(0.04)*</td>
<td>1.630(0.09)*</td>
<td>1.770(0.06)</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>34.3(3.9)*</td>
<td>49.58(9)*</td>
<td>76.7(9.4)</td>
</tr>
<tr>
<td>Peak VO$_2$ (1·min$^{-1}$)</td>
<td>1.820(0.22)*</td>
<td>2.600(0.47)*</td>
<td>4.180(0.30)</td>
</tr>
<tr>
<td>Peak VO$_2$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>5.36(0)</td>
<td>5.35(0)</td>
<td>5.5(4)</td>
</tr>
<tr>
<td>HR at peak VO$_2$ (beats·min$^{-1}$)</td>
<td>200(7)*</td>
<td>201(10)</td>
<td>195(8)</td>
</tr>
<tr>
<td>Lactate at peak VO$_2$ (mmol·l$^{-1}$)</td>
<td>4.5(1.5)*</td>
<td>5.6(2.1)*</td>
<td>8.7(1.9)</td>
</tr>
</tbody>
</table>

All values are means±d.).

*Significantly different from teenage and adult groups (P < 0.05);
*Significantly different from pre-pubertal and adult groups (P < 0.05);
*Significantly different from adult group (P < 0.05).

speeds with no incline: prepubertal 1.94 m·s$^{-1}$ (7 km·h$^{-1}$), teenage 2.22 m·s$^{-1}$ (8 km·h$^{-1}$), adult 2.78 m·s$^{-1}$ (10 km·h$^{-1}$). The speed was held constant and the gradient raised for each stage subsequent to a belt speed of 2.78 m·s$^{-1}$ (10 km·h$^{-1}$). Exercise stages were separated by a maximum of 90 s for blood sampling. Expired air was monitored continuously throughout each stage using a computerized on-line system (Oxyconsigma, Mijnhardt B.V., The Netherlands) which was automatically calibrated according to the manufacturers' instructions before each test. End of stage VO$_2$ and peak VO$_2$ values were determined from the last 30 s of each stage. Heart rate was monitored using a Rigel (Morden, UK) electrocardiogram and recorded at the end of each stage. A measure of peak VO$_2$ was accepted if: (1) the heart rate levelled off before the final exercise intensity or was ≥ 95% of age predicted maximum; and/or (2) if the respiratory exchange ratio was equal to or above unity.10 Peak VO$_2$ is highly reproducible in children,10 and as reliable as VO$_2$ max in adults, if the objective criteria given above are satisfied.1011

Blood sampling

Following each exercise stage, and upon termination of the test, capillary blood was taken from a thumb tip which had been wiped with alcohol. The first drop of blood was discarded and 25 µl of free flow whole blood were collected in heparinized microvets (Sarstedt CB300). These samples were immediately assayed in duplicate for lactic acid concentration using a YSI 2300 STAT lactate analyser (Yellow Springs Instruments, Yellow Springs, USA). The analyser self calibrated every 5 min using a 5 mmol·l$^{-1}$ standard; the maximum acceptable calibration error was < 2%. The coefficient of variation of whole blood lactate measurement for the laboratory quality control was 2.85%. It is acknowledged that the substantial changes in packed cell volume that can occur during a maximal test can influence lactate values determined from whole blood, but that the use of whole blood analysers in exercise science laboratories is widespread.

Oxygen consumption and heart rate responses were plotted against lactate concentration for each subject. Oxygen consumption and heart rate at 2.5 and 4.0 mmol·l$^{-1}$ were determined by visual interpolation for each subject.9

Analysis

The data were analysed using the statistical package for the social sciences (SPSS Inc, USA). Significant differences between group means were ascertained using one way analysis of variance (ANOVA) with subsequent Scheffé follow up where homogeneity of variance was established. Where homogeneity of variance was not established, Kruskal-Wallis ANOVA was used.12 Significance was assumed at $P < 0.05$. Relationships among measures were examined using Pearson product moment correlation analyses. All data are presented as mean±sd.

Results

Oxygen consumption and heart rate responses at 2.5 and 4.0 mmol·l$^{-1}$ lactate concentrations are presented in Tables 2 and 3 respectively. For some subjects interpolation at one, or both, of the lactate values was not possible. Consequently, 2.5 mmol·l$^{-1}$ lactate analysis was based upon 21 prepubertal boys, 22 teenage boys, 22

Table 2. Oxygen consumption response at exercise intensities corresponding to 2.5 and 4.0 mmol·l$^{-1}$ blood lactate concentration in the three groups

<table>
<thead>
<tr>
<th>Lactate variable</th>
<th>Prepubertal</th>
<th>Teenage</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>% peak VO$_2$ at 2.5 mmol·l$^{-1}$</td>
<td>85(6)</td>
<td>85(9)</td>
<td>80(9)</td>
</tr>
<tr>
<td>VO$_2$ (1·min$^{-1}$)</td>
<td>1.590(15)</td>
<td>2.250(47)</td>
<td>3.430(62)</td>
</tr>
<tr>
<td>VO$_2$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>46(5)</td>
<td>45(5)</td>
<td>45(6)</td>
</tr>
<tr>
<td>% peak VO$_2$ at 4.0 mmol·l$^{-1}$</td>
<td>94(0)</td>
<td>92(6)</td>
<td>87(9)</td>
</tr>
<tr>
<td>VO$_2$ (1·min$^{-1}$)</td>
<td>1.770(16)</td>
<td>2.430(53)</td>
<td>3.680(59)</td>
</tr>
<tr>
<td>VO$_2$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>51(5)</td>
<td>49(6)</td>
<td>48(6)</td>
</tr>
</tbody>
</table>

All values are means±d.).

*Significantly different from teenage and adult groups (P < 0.05);
*Significantly different from pre-pubertal and adult groups (P < 0.05);
*Significantly different from adult group (P < 0.05).

Table 3. Heart rate response at exercise intensities corresponding to 2.5 and 4.0 mmol·l$^{-1}$ blood lactate concentration in the three groups

<table>
<thead>
<tr>
<th>Lactate variable</th>
<th>Prepubertal</th>
<th>Teenage</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>% peak HR at 2.5 mmol·l$^{-1}$</td>
<td>95(3)</td>
<td>94(4)</td>
<td>87(4)</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)</td>
<td>191(7)</td>
<td>188(9)</td>
<td>169(11)</td>
</tr>
<tr>
<td>% peak HR at 4.0 mmol·l$^{-1}$</td>
<td>98(2)</td>
<td>98(2)</td>
<td>92(4)</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)</td>
<td>199(5)</td>
<td>199(9)</td>
<td>179(10)</td>
</tr>
</tbody>
</table>

All values are means±d.).

*Significantly different from teenage and adult groups (P < 0.05).
and 18 men. At 4.0 mmol·l⁻¹ lactate, analysis was based upon 17 prepubertal boys, 21 teenage boys, and 21 men.

Mean percent VO₂ at the 2.5 mmol·l⁻¹ level (Table 2) was not significantly different across the three age groups (P > 0.05). Both groups of boys had significantly lower (P < 0.05) absolute VO₂ (l·min⁻¹) values than the men; the prepubertal group values were also significantly lower (P < 0.05) than the teenage group at the 2.5 mmol·l⁻¹ level. Body mass related VO₂ (ml·kg⁻¹·min⁻¹) at 2.5 mmol·l⁻¹ lactate was not significantly different between the three groups (P > 0.05).

For both groups of boys 4.0 mmol·l⁻¹ lactate occurred at a significantly (P < 0.05) higher mean % peak VO₂ than in the men. The prepubertal and teenage values were not significantly different (P > 0.05). The prepubertal and teenage boys had lower absolute VO₂ values (P < 0.05) than the men at the 4.0 mmol·l⁻¹ level; the prepubertal group values were also significantly lower (P < 0.05) than the teenage group. Body mass related VO₂ between age groups at 4.0 mmol·l⁻¹ lactate was not significantly different (P > 0.05).

Both groups of boys showed higher mean absolute heart rate and % peak heart rate (P < 0.05) than the men at the 2.5 and 4.0 mmol·l⁻¹ lactate levels. The prepubertal and teenage values were not significantly different (P > 0.05). Correlation coefficients between peak VO₂ (ml·kg⁻¹·min⁻¹) and % peak VO₂ at the 2.5 and 4.0 mmol·l⁻¹ levels were not significantly different from zero (P > 0.05).

Discussion

Mean percent peak VO₂ values at both the 2.5 and 4.0 mmol·l⁻¹ fixed blood lactate reference values in the prepubertal and teenage groups concur with the findings from the sparse number of studies conducted to date. It is clear that boys are able to exercise at intensities close to those which elicit peak VO₂ without accumulating high levels of blood lactate. Submaximal lactate indices can be reliably reproduced given subjects who have had a period of familiarization with all test procedures. For some subjects interpolation at one, or both, of the lactate values was not possible. Lactate values for 13 subjects (nine prepubertal and four teenage) were lower than 4.0 mmol·l⁻¹ throughout the test. Furthermore, four prepubertal boys failed to reach a 2.5 mmol·l⁻¹ lactate value. An explanation for these low lactate responses is not clear. All subjects satisfied the criteria for maximum effort, so less than maximal effort is not a plausible explanation. Large between-subject variance in blood lactate responses to incremental exercise may partially explain this finding. One teenage boy and two men had lactate values that were above 4.0 mmol·l⁻¹ throughout the test. In addition, two prepubertal boys, two teenage boys and three men had lactate values above 2.5 mmol·l⁻¹ throughout the test. The most plausible explanation for these responses is an exaggerated, anxiety provoked catecholamine response at the onset of exercise. The relationship between adrenaline and glycolysis, with subsequent lactate accumulation, is well documented in both children and adults. These results suggest that the 4.0 mmol·l⁻¹ value may represent an inappropriate submaximal reference point for monitoring and assessing endurance performance in prepubertal and teenage boys, at least where a whole blood lactate assay is used. It is unlikely that prepubertal and teenage boys would be able to sustain exercise that corresponds to a 4.0 mmol·l⁻¹ fixed lactate for a period of time common to endurance events. Such a high lactate concentration would soon lead to exercise cessation in these groups.

The results of the limited number of child based studies indicate that the blood lactate response to submaximal exercise may be age or maturity related. As no significant differences between the prepubertal and teenage boys were found in % peak VO₂, body mass related peak VO₂ (ml·kg⁻¹·min⁻¹), heart rate, and % peak heart rate at either the 2.5 or the 4.0 mmol·l⁻¹ lactate level this hypothesis is not supported. When expressed in terms of absolute peak VO₂ (l·min⁻¹) the significant differences between the two groups of boys were to be expected due to the well documented increases in peak VO₂ with increasing body mass. Although the 4.0 mmol·l⁻¹ blood lactate level corresponds to a significantly lower % peak VO₂, % peak heart rate, and absolute heart rate in the men as compared to both groups of boys reaffirms previous findings. When considering the heart rate responses that correspond to fixed blood lactate concentrations, children's hearts respond in a dissimilar manner to adults during submaximal exercise. This difference could be attributed to higher heart rate and lower stroke volume at each level of cardiac output compared to adults. Thus, lactate responses expressed in relation to heart rate at submaximal exercise must be viewed with caution when comparing children and adults. Possible explanations for the child-adult differences remain at present speculative. As differences at both the 2.5 and 4.0 mmol·l⁻¹ reference values between the two groups of boys were not demonstrated in this study, variations may not simply be ascribed to maturation. Child-adult differences in exercising blood lactate may be due to a lower muscle lactic acid production although, more recently, studies using radioactive isotope tracers to measure lactate turnover have indicated that this may only partly explain the differences. As these studies involved adult subjects and not children, it may not be possible simply to extrapolate these results to paediatric populations. Research has shown that children may have an enhanced ability to derive energy aerobically as compared to adults. It has also been suggested that the mediating role of catecholamine concentration on lactate response to exercise is different in children compared to adults. These differences may lead to preferential use of aerobic energy systems and a blunted catecholamine response resulting in a lower lactate production.

The finding that the 2.5 mmol·l⁻¹ lactate level represented a similar mean percent peak VO₂ for each group is in contrast to previous reports. Although further statistical analyses revealed strong trends when comparing the prepubertal and the adult groups (effect size = 0.66), and the teenage and adult groups (effect size = 0.71)
size = 0.56) at the 2.5 mmol·L⁻¹ level; the non-significant findings at P > 0.05 were accepted due to the strong power of the study.²⁴

In conclusion, unlike men, prepubertal and teenage boys are able to exercise at intensities close to those which elicit peak VO₂ without accumulating high levels of blood lactate. Conversely, the exercise intensity corresponding to the 2.5 mmol·L⁻¹ lactate concentration results in a similar % peak VO₂ in prepubertal boys, teenage boys and men. Factors other than maturation appear to influence whole blood lactate responses to exercise. Why physiological responses corresponding to blood lactate were different when comparing the boys and men at the higher exercise intensity and not the lower intensity is unknown. These results should be taken into account when considering physiological variables corresponding to fixed blood lactate concentrations to monitor and assess prepubertal and teenage boys’ endurance performance.

References

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