Blood Lactate Responses to Exercise in Children: Part 1. Peak Lactate Concentration

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Part 1 reviews the literature concerning peak blood lactate responses to exercise in children. After a brief overview of lactate metabolism, an analysis is presented comparing children to adults regarding peak blood lactate concentration. Possible factors accounting for lower blood lactate concentrations during maximal exercise in children are considered.

Pyruvic acid produced during glycolysis is either oxidized to form acetyl coenzyme A or reduced to produce lactic acid. Lactic acid, therefore, is the metabolic by-product of incomplete oxidation of carbohydrate via the glycolytic pathway. Accumulation of lactate, the salt of lactic acid, in the muscles is one of several factors that has been related to fatigue during and following strenuous exercise. Lactic acid accumulation in the muscles inhibits glycolytic enzymes and thereby limits glycolytic capacity.

In the early 1970s, a series of papers by Eriksson and various coauthors (8, 9, 10, 11) found lower peak lactate values in children than in adults, and substantially reduced phosphofructokinase (PFK) activity in children compared to adults. These findings led to the hypotheses that children have lower glycolytic capacity than adults, and that this reduced capacity is related to reduced glycolytic enzyme activity. Over the past 25 years, a number of studies have reported reduced lactate concentrations in children following maximal and submaximal exercise. These studies typically explain that these reduced lactate concentrations in children are due to reduced PFK activity, as originally described by Eriksson. Subsequent studies of enzyme profiles in skeletal muscle in children, however, have not fully supported Eriksson’s findings. Thus, several other plausible causal factors have been proposed to explain the lower observed exercise induced lactate levels in children.

Direct comparison of the results of different studies is complicated by the variability in terminology used between studies to describe peak lactate concentration. The following definitions are provided for clarification, and will be used throughout this review:
• **Peak lactate** describes the highest lactate concentration achieved during or shortly after an exercise protocol.

• **Maximal lactate** describes the highest lactate concentration observed when the full capacity of the glycolytic pathway is used, such as immediately following a maximal effort of 1 to 6 min duration.

• **VO\textsubscript{max} lactate** describes the blood lactate concentration measured at completion of a VO\textsubscript{2}\text{max} test.

This paper considers blood lactate responses to exercise in children, and possible explanations for lower observed lactate concentrations in children than in adults. After a brief overview of lactate metabolism, this paper presents the evidence that supports the hypothesis that peak blood lactate concentration is lower in children than adults. The associations between peak lactate concentration versus age/sexual maturation and other variables are also examined. Finally, this paper considers possible factors accounting for lower peak blood lactate concentrations during maximal exercise in children.

**Overview of Lactate Metabolism**

Lactic acid \((\text{C}_3\text{H}_6\text{O}_3)\) is a strong organic acid with a pK of 3.7 to 3.8. At pH ranges normally found in skeletal muscle at rest or during exercise of 6.4 to 7.4, over 99% of lactic acid is dissociated to an anion \((\text{C}_3\text{H}_5\text{O}_4^-)\), and a proton \((\text{H}^+)\) (26). Lactic acid, therefore, is formed in the muscles and is quickly converted to lactate, a salt of lactic acid, and the two terms are often used interchangeably.

Lactate concentration, generally measured in the blood, is used to represent muscle lactate levels. There is a time lag, however, between production of lactic acid in skeletal muscle and the appearance of lactate in the blood due to the time required for (a) diffusion of lactate across the sarcolemma and capillary wall, and (b) transport to the sampling site. During the first several minutes after a change in exercise intensity, therefore, blood lactate concentration may not accurately reflect muscle lactate levels. Blood lactate also may not accurately represent muscle lactate due to the dilution of lactate as it enters the blood.

Lactate is continuously produced by skeletal muscle, even at rest (26). Skeletal muscle can simultaneously produce and consume lactate, therefore net lactate output does not reflect total lactate production by the muscle (4). During rest and moderate exercise, blood lactate levels remain relatively constant because the rate of diffusion into the blood is equal to the rate of removal. At the onset of exercise or during non-steady-state exercise, the rates of lactate production and diffusion exceed the rate of removal and blood lactate levels rise, whereas during recovery, the rate of removal exceeds the rate of production and blood lactate decreases. The amount of change in blood lactate levels depends on the size of the lactate pool and the differential between the rates of production and removal (26).

Lactic acid is produced as a by-product of glycolysis, so increases in the rate of glycolysis will result in a greater production of lactate. The rate of glycolysis is increased through stimulation of the rate limiting enzyme PFK, which is activated by increases in the ADP/ATP and AMP/ATP ratios (16). The rate of lactic acid production from pyruvate depends on the rate of pyruvate production, the rate of use of pyruvate by oxidative phosphorylation in the mitochondria, and the activity of lactate dehydrogenase (LDH), the enzyme that catalyzes the reduction of pyruvate to lactate.
Under conditions in which the mitochondria cannot use all of the pyruvate produced by glycolysis, the excess pyruvate forms lactate through the action of LDH (4). Lactate in skeletal muscle can either diffuse into the blood or be converted to pyruvate. The rate of formation of pyruvate from lactate will be influenced by the rate of use of pyruvate and the rate of formation of pyruvate from other substrates. Pyruvate formed from lactate can follow four pathways: oxidation by the mitochondria, conversion to glycogen, alanine formation, or diffusion from muscle (16). Lactate that diffuses into the bloodstream is removed by oxidation in the skeletal muscles or heart, or is converted to glucose through the process of gluconeogenesis in the liver and kidney (26).

Lactate metabolism, therefore, is a complex cycle in which lactate is produced and consumed in various tissues. Lower blood lactate concentrations have been found in children both during submaximal and maximal exercise and at the lactate threshold, but it is unclear whether these lower lactate concentrations reflect reduced production, increased removal, increased dilution, or some combination of these factors.

**Peak Blood Lactate Concentration in Children**

The ability to generate high blood lactate concentrations reflects the subject's glycolytic capacity. It has been hypothesized that glycolytic capacity may not be fully developed in children. To determine children's glycolytic capacity, a large number of studies have investigated the ability of children to produce peak concentrations of lactate during exercise. These studies have generally measured blood lactate immediately postexercise, or several minutes postexercise (maximal or peak VO₂ tests on a treadmill or cycle ergometer, or on Wingate tests). A variety of studies have investigated the associations between the ability to generate peak lactate concentrations and various exercise protocols, age/sexual maturation, and exercise training. The results of several studies investigating peak blood lactate concentrations in children are summarized in Table 1.

Motivational considerations may also play a role in the lower lactate concentrations observed in children. For example, Cumming et al. (5) observed that their laboratory found higher peak lactate concentrations in children as the investigators gained greater experience in testing children, and attributed the difference to greater experience in getting maximal efforts from the children tested. The lower values observed in children, therefore, could be due, at least in part, to reduced motivation in the children to give a maximal effort, less comfort with testing protocols that may have been designed for adults, or both.

**Peak Lactate and Exercise Test Protocol**

The specific exercise protocol used is likely to have an effect on the ability to accumulate high levels of blood lactate because protocols of differing length, intensity, and exercise mode will stimulate glycolysis to different degrees. The following studies by Mero (19), Fellmann et al. (12), and Nazar et al. (21) illustrate these relationships.

In a 1988 study, Mero (19) investigated the ability of boys to generate peak lactate levels using three different exercise protocols: a 15-s modified Wingate test, a 60-s modified Wingate test, and a VO₂max test on the treadmill with a mean
duration of 23 min. Twenty-five boys participated, including 19 trained athletes (both power and endurance athletes), and 6 controls (age $M \pm SD = 12.6 \pm 0.8$ years). Mero found that the VO$_{\text{max}}$ protocol and 15-s Wingate test elicited peak lactate concentrations that were only 60.6% and 68.7% as high, respectively, as the 60-s Wingate test. No significant differences were reported between the athletes and the controls, but the athletes included a mix of endurance and power athletes. It is therefore possible that differences were obscured by combining both types of athletes into one group. The 60-s maximal effort on the cycle ergometer utilized more of the subjects' glycolytic capacity than either the shorter duration maximal test, or the longer duration VO$_{\text{max}}$ test. This finding challenges the practice of measuring lactate concentrations at the end of a VO$_{\text{max}}$ test and considering those results as the highest achievable lactate concentrations.

Consistent with these results, Fellmann et al. (12) found similar peak lactate concentrations were elicited by a conventional VO$_{\text{max}}$ test and a 30-s Wingate test. This suggests that children are capable of achieving higher lactate concentrations than those achieved during either test, because a 30-s test is likely too brief to maximally utilize glycolytic capacity. It is possible that different trends would be found between children of different ages, and between children and adults if exercise protocols of close to optimal duration were consistently used to elicit peak lactate concentrations.

Nazar et al. (21) investigated the relationship between plasma ammonia and blood lactate concentration in 22 boys and 20 girls ages 13.2 to 13.7 years. Measurements were obtained at completion of a VO$_{\text{max}}$ test on a treadmill, and higher blood lactate concentrations were seen in girls participating in general sporting activities than in elite swimmers. The relatively low peak lactate concentrations found in the swimmers may be related to the exercise mode used in that the treadmill may not have taken advantage of training-specific adaptations to swimming.

The results of the above studies suggest that VO$_{\text{max}}$ lactate does not necessarily represent the maximal lactate concentration that a subject can achieve. Maximal lactate is generally seen in events lasting 1–6 minutes, whereas a VO$_{\text{max}}$ test typically lasts 10–20 minutes, depending on the specific protocol employed. The criteria typically used to signify achievement of VO$_{\text{max}}$ (leveling off of VO$_2$, $R > 1.0$ or 1.1, achievement of close to predicted maximum heart rate) do not necessarily signify that the subject's glycolytic capacity has been fully utilized. It seems reasonable to assume that subjects who terminate a VO$_{\text{max}}$ test by volitional exhaustion, and who meet the criteria for achievement of VO$_{\text{max}}$ may differ markedly in the amount of anaerobic work performed. It is difficult, therefore, to assign specific physiological significance to VO$_{\text{max}}$ lactate values. A maximal effort lasting 1 to 6 min would elicit lactate values more representative of the glycolytic capacity of the muscle. A comparison of peak blood lactate concentrations achieved under these conditions appears to be a more valid indication of glycolytic capacity than does a conventional VO$_{\text{max}}$ test.

**Reliability of Peak Lactate in Children**

The reliability of a variable upon repeated measurements has implications for the ability to compare results on that variable across subjects, over time, or consequent to some treatment. Cunningham et al. (7) investigated the reliability of VO$_{\text{max}}$ and VO$_{\text{max}}$ lactates in children by measuring these variables twice in sixty-six 10-year-old boys, 4 to 5 months apart. The correlation coefficients for
Table 1  Peak Blood Lactate Concentration in Children

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Peak lactate concentration</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eriksson et al. (10)</td>
<td>8</td>
<td>VO₂ max protocol elicited 11.3 mmol · kg⁻¹ wet muscle—only study not to present blood lactate</td>
<td>Peak lactate levels may be related to sexual maturity in 13-year-old boys.</td>
</tr>
<tr>
<td>Eriksson (8)</td>
<td>12</td>
<td>16 weeks training</td>
<td>Increased peak lactate found with training in 11- to 13-year-old boys. Some portion of increase may be related to increased age/maturity.</td>
</tr>
<tr>
<td>Cumming et al. (7)</td>
<td>33</td>
<td>Age 11.6 years = 7.9 ± .5 mmol</td>
<td>Max lactate increased with age in boys. Oldest group had values close to adults.</td>
</tr>
<tr>
<td>Rusko et al. (25)</td>
<td>15</td>
<td>Pretraining 8.0 ± .5 mmol</td>
<td>Reliability weak (r = .24–.60) across 2 tests 4-5 months apart in athletic 10 year old boys.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Posttraining 9.5 ± .7 mmol</td>
<td>Max lactate not high in endurance-trained girls, age 15 to 20 years. Duration of VO₂ protocol (20–25 min) may lead to lower peak lactates.</td>
</tr>
<tr>
<td>Cumming et al. (6)</td>
<td></td>
<td>Age Males Females</td>
<td>Boys, but not girls, exhibited increased peak lactate with age. Higher than typical-peak lactates for ages 4-12, but not ages 13–20.</td>
</tr>
<tr>
<td>Cumming et al. (5)</td>
<td></td>
<td>Age Males Females</td>
<td>Max lactate increased with age in both genders. Larger increase for boys after onset of puberty.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>4-5</th>
<th>6-7</th>
<th>8-9</th>
<th>10-12</th>
<th>13-15</th>
<th>16-20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>9.5 mmol</td>
<td>9.1</td>
<td>9.9</td>
<td>10.2</td>
<td>11.3</td>
<td>12.1</td>
</tr>
<tr>
<td>Males</td>
<td>10.4</td>
<td>9.5</td>
<td>10.2</td>
<td>10.2</td>
<td>11.6</td>
<td>10.4</td>
</tr>
<tr>
<td>Females</td>
<td>10.4</td>
<td>9.5</td>
<td>10.2</td>
<td>10.2</td>
<td>11.6</td>
<td>10.4</td>
</tr>
<tr>
<td></td>
<td>4-5</td>
<td>6-7</td>
<td>8-9</td>
<td>10-12</td>
<td>13-15</td>
<td>16-20</td>
</tr>
<tr>
<td>Age</td>
<td>8.1 mmol</td>
<td>8.9</td>
<td>9.0</td>
<td>9.5</td>
<td>10.8</td>
<td>13.7</td>
</tr>
<tr>
<td>Males</td>
<td>8.4</td>
<td>9.4</td>
<td>10.5</td>
<td>10.7</td>
<td>11.5</td>
<td>11.5</td>
</tr>
<tr>
<td>Females</td>
<td>8.4</td>
<td>9.4</td>
<td>10.5</td>
<td>10.7</td>
<td>11.5</td>
<td>11.5</td>
</tr>
</tbody>
</table>
Atomi et al. (2)

Paterson et al. (23)

Mero (19)

Williams et al. (28)

Williams & Armstrong (27)

Mocellin et al. (20)

Nazar et al. (21)

Fellman et al. (12)

Fernhall et al. (13)

<table>
<thead>
<tr>
<th>Age</th>
<th>Boys 6.2 ± 0.4 mmol</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>7.0 ± 1.4 mmol</td>
</tr>
<tr>
<td>12</td>
<td>7.7 ± 1.7</td>
</tr>
<tr>
<td>13</td>
<td>9.0 ± 1.9</td>
</tr>
<tr>
<td>14</td>
<td>9.0 ± 1.7</td>
</tr>
<tr>
<td>15</td>
<td>10.1 ± 1.5</td>
</tr>
</tbody>
</table>

Peak lactate concentration increased 44.2% with age in 11- to 15-year-old athletic boys. Subjects may have become more highly trained with increasing age.

<table>
<thead>
<tr>
<th>Tanner Stage</th>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.3</td>
<td>NA</td>
</tr>
<tr>
<td>2</td>
<td>5.0</td>
<td>5.8</td>
</tr>
<tr>
<td>3</td>
<td>4.7</td>
<td>5.7</td>
</tr>
<tr>
<td>4</td>
<td>5.8</td>
<td>6.6</td>
</tr>
<tr>
<td>5</td>
<td>5.8</td>
<td>5.7</td>
</tr>
</tbody>
</table>

Relatively low V0,max lactates could be related to exercise protocol, blood sampling, and assay.

Boys 6.8 ± 1.5 mmol

<table>
<thead>
<tr>
<th>Age</th>
<th>15-s Wingate</th>
<th>60-s</th>
<th>23-min V0,max</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>9.3 ± 2.0</td>
<td>13.1 ± 2.6</td>
<td>7.9 ± 2.5</td>
</tr>
<tr>
<td>12</td>
<td>8.5 ± 1.5</td>
<td>12.8 ± 2.3</td>
<td>7.8 ± 1.5</td>
</tr>
</tbody>
</table>

Girls higher lactate at peak VO2 than boys. Lactate at peak VO2 was not significantly related to Tanner stage of sexual maturity or chronological age in either girls or boys.

Boys elite swimmers
Girls elite swimmers
Girls general athletes
Boys elite swimmers
Boys general athletes
Boys untrained

| High altitude, high SES | 5.1 mmol |
| High altitude, low SES  | 4.8 mmol |
| Low altitude, high SES  | 7.4 mmol |
| Low altitude, low SES   | 6.3 mmol |

Highest lactate concentration found in girl athletes participating in general sports activities. Lowest peak lactates found in untrained boys. Relatively low peak lactate in swimmers could be due to use of treadmill for V0,max testing.

Boys 7.8 ± 2.4 mmol
Girls 8.5 ± 1.7 mmol

Peak Lactate for endurance-trained adolescents similar to endurance-trained adults.
peak blood lactate concentrations between the two tests were $r = .28$ for those subjects who achieved a plateau in VO$_2$ on both tests, $r = .60$ for those subjects who achieved a plateau in VO$_2$ on one test only, and $r = .24$ for those subjects who did not achieve a plateau in VO$_2$ on either test. Although the means were similar across the two tests, these correlations indicate that VO$_{max}$ lactate is not a reliable measure. This relatively poor reliability may be related to the concerns discussed above in that VO$_{2,max}$ can be achieved without full or consistent utilization of glycolytic capacity.

**Peak Lactate and Chronological Age/Sexual Maturation**

The relationship between chronological age or stage of sexual maturation and peak lactate concentrations has been investigated in studies by Eriksson et al. (10), Eriksson and Saltin (11), Cumming et al. (5, 6), Mero (19), Paterson et al. (23), Paterson and Cunningham (22), Williams and Armstrong (27), and Fellmann et al. (12). Changes in peak lactate concentration with age or stage of sexual maturation reported in these studies provide an indication of how children's glycolytic capacity changes with growth and development. The findings of these studies have been inconclusive.

Eriksson et al. (10) found an "almost significant" positive relationship between testicular volume and VO$_{max}$ muscle lactate in eight 13-year-old boys. In addition, muscle lactate concentration in boys was lower than for adult males throughout the range of relative workloads. Eriksson and Saltin (11) reported VO$_{max}$ lactate concentrations in four groups of boys with mean ages of 11.6, 12.6, 13.5, and 15.5 years, of 7.9, 9.6, 9.2, and 10.5 mmol · L$^{-1}$, respectively. The authors conclude that peak lactate concentration increases with age; however, the effects of training and diet may affect the interpretation. ATP and creatine phosphate (CP) stores were similar in children and adults. Glycogen stores for the four age groups were 54, 70, 69, and 87 mmol · kg$^{-1}$ wet weight, respectively, which suggests that glycogen stores increase with age in children. In addition, younger boys utilized less glycogen during exercise than the older boys, which is consistent with the hypothesis that glycolytic enzyme activity increases with age/sexual maturation in children.

Two studies by Cumming et al. (5, 6) using the Bruce protocol found higher VO$_{max}$ lactate concentrations in young children than found in other studies. The authors speculate that the relatively high lactate values found in these two studies may be due, at least in part, to the ability to motivate children to achieve maximal efforts. These lactate values may have been higher than those found in other studies because Cumming et al. analyzed serum, whereas the majority of studies have analyzed whole blood. The serum fraction of the blood has a higher lactate concentration than the erythrocytes, so serum has a higher lactate concentration than whole blood, which contains both fractions (15). Interstudy results can only be validly compared, therefore, when the same blood fraction is analyzed between studies.

The first study (6) reported mean VO$_{max}$ lactate concentrations for boys of 9.5 ± 2.3 mmol for ages 4–5 years, 9.1 ± 2.2 mmol for ages 6–7 years, 9.9 ± 2.1 mmol for ages 8–9 years, 10.2 ± 2.1 mmol for ages 10–12 years, 11.3 ± 2.5 mmol for ages 13–15 years, and 12.1 ± 3.2 mmol for ages 16–20. For the girls, mean VO$_{max}$ lactate concentrations for the above age groups were 10.4, 9.5, 10.2, 10.2, 11.6, and 10.4 mmol, respectively. The boys exhibited a small increase in maximal lactate concentrations with increasing age, with mean maximal lactate concentrations increasing by 2.6 mmol from ages 4–5 years to ages 16–20 years. The girls, however, did not exhibit increased maximal lactate concentrations with age.
Cumming et al. (6) explain that the lactate concentrations elicited in their study for ages 13 to 20 were similar to or only slightly higher than results found in other studies with this age group. For ages 4–12, however, lactate concentration was substantially higher than found in other studies.

In a subsequent study, Cumming et al. (5) found VO₂max lactate concentration to increase with age in both genders, with a greater increase observed in boys. For the boys, VO₂max lactate values increased from 8.1 ± 2.6 mmol in 4–5 year olds to 13.7 ± 3.4 mmol in 16–20 year olds. For the girls, VO₂max lactates increased from 8.4 ± 2.3 mmol in 4–5 year olds to 11.5 ± 2.8 mmol in 16–20 year olds. The lactate values found in this study are similar in the boys and girls until puberty. Clear increases in VO₂max lactate concentrations were found with increasing age, with larger increases in the boys emerging after the onset of puberty.

Similarly, Mero (19) observed significant correlations between peak blood lactate concentration and both serum testosterone and type II muscle fiber area. These data support the hypothesis that the ability to produce high lactate levels depends on the level of circulating testosterone. The influence of type II muscle fiber area may be related to the higher content of muscle lactate dehydrogenase enzyme in type II fibers that regulates the reduction of pyruvate to lactate.

The only longitudinal study to investigate peak lactate and age/sexual maturation was conducted by Paterson et al. (23). In this study, VO₂max and VO₂max lactate were measured over 5 years in 11- to 15-year-old boys. VO₂max lactate increased by 44% from the first test at age 11 to the last test at age 15. These subjects were highly athletic, and many of them increased their training over the 5 years of the study. These boys may have become more highly trained over time, so the effects of training, age, and maturation cannot be separated. VO₂max (ml·kg⁻¹·min⁻¹) has been reported to remain relatively stable over this age span in boys (1). For the subjects in this study, relative VO₂max increased by 11.8% between the first and last tests, which suggests that the boys' level of aerobic fitness increased modestly during this time period.

Analyzing data from subjects in the same 5 year longitudinal investigation, Paterson and Cunningham (22) observed a relationship between peak lactate and chronological age, but no relationship between peak lactate and stage of maturation in 11- to 15-year-old boys. Using a treadmill protocol with a 20% grade designed to maximally stress the capacity of the anaerobic energy system, this study found a 38% increase in peak lactate between the ages of 11 and 15, but no difference in peak lactate concentration between early and late maturing boys of the same age.

In a 1991 study, Williams and Armstrong (27) investigated the influences of age and sexual maturation on lactate concentration at peak VO₂ and the lactate threshold in 100 boys and 91 girls, aged 11 to 16 years. VO₂max lactate was significantly higher for the girls (6.1 ± 1.7 mmol) than boys (5.4 ± 1.7 mmol). For the boys, mean VO₂max lactates were 5.3, 5.0, 4.7, 5.8, and 5.8 mmol·L⁻¹, at Tanner Stage 1 through 5, respectively. For the girls, lactate at peak VO₂ averaged 5.8, 5.7, 6.6, and 5.7 mmol·L⁻¹ at Tanner Stages 2 through 5, respectively. The relatively low VO₂max lactates found in this study could be related to the use of the whole-blood method, which measures only the lactate in the blood plasma and not in the erythrocytes. VO₂max lactate was not significantly related to Tanner stage of sexual maturity or chronological age in either girls or boys.

Fellmann et al. (12) investigated blood lactate after maximal and supramaximal exercise in 10- to 12-year-old boys from Bolivia. The authors reported lower lactate
concentrations in children from high altitude than in children from low altitude, and lower lactate concentrations in children from low socioeconomic status than in children from high socioeconomic status following both maximal exercise and a 30-s Wingate test. The authors attribute these differences to lower gonadal maturation measured as lower testosterone levels in the children from high altitude, low socioeconomic status, or both.

The results of the above studies suggest that the ability to generate high peak blood lactate concentrations during exercise may be related to age, and that the relationship with age may be due, at least in part, to differences in stage of sexual maturation. There is less evidence of these trends in girls than boys, but evidence suggests that girls and boys have similar peak lactate concentrations until sometime during puberty, at which point peak lactate concentrations increase more in boys than in girls. It is possible that circulating testosterone levels may be a factor contributing to this pubertal increase in peak lactate in boys. Analysis of the findings of Williams and Armstrong (27) and Paterson and Cunningham (22), however, shows no evidence of increased peak lactate with stage of sexual maturation. In light of these data, it has been suggested that glycolytic capacity may be related to chronological age rather than biological age (24). Further investigation is warranted to investigate the relationship between peak lactate concentration and sexual maturation.

**Peak Lactate and Exercise Training**

Strength, power, and interval training have been found to be related to increased glycolytic capacity in adults, whereas endurance training has been shown to have only a minimal effect. Investigation of the relationship between exercise training and the ability to generate high peak lactate concentrations provides insight into the effects of training on glycolytic capacity. Eriksson (8) reported the results of a training study with twelve 11- to 13-year-old boys. The subjects trained three times per week for 16 weeks, including a combination of endurance training and interval training. VO$_2$max lactate concentrations averaged 8.0 ± 0.5 mmol pretraining, and 9.5 ± 0.7 mmol posttraining. There was no control group in this study, however, so the portion of the increase in maximal lactate that may be attributable to increases in age and maturation cannot be separated from the training effect.

In a 1980 study, Rusko et al. (25) investigated peak lactate concentrations and anaerobic threshold in 15 elite female cross country skiers, aged 15 to 20 years. VO$_2$max lactate concentrations on a cycle ergometer averaged 8.2 ± 1.4 mmol · L$^{-1}$. These values were similar to those that would be expected for untrained girls of this age, which suggests that endurance training may not be a strong stimulus to increase the ability to achieve peak lactate concentrations. The relatively lengthy VO$_2$max protocol used in this study (20–25 min duration) may have led to lower VO$_2$max lactates than would have been elicited by a shorter protocol. Similarly, Fernhall et al. (13) investigated VO$_2$max lactate, lactate threshold, and cross-country run performance in boys and girls aged 15.0–18.3 years. The investigators found VO$_2$max lactate concentrations of 7.8 ± 2.4 mmol in the boys and 8.5 ± 1.7 mmol in the girls. Fernhall et al. report that these VO$_2$max lactate concentrations are similar to those seen in endurance-trained adults, which supports Rusko et al.'s (25) observation that endurance training does not increase the capacity to achieve peak lactate concentrations.
Limited evidence is available on the relationship between different forms of training and peak lactate concentration in children. The results of the above studies suggest that endurance training may not increase the ability to accumulate high levels of lactate. This hypothesis appears reasonable given that endurance training has more of a tendency to stimulate oxidative metabolism than glycolytic metabolism. The Eriksson (8) study provides the only training data, however, so the results of the Rusko et al. (25) and Fernhall et al. (13) investigations, which studied endurance athletes at one point in time, should be interpreted with caution. The results of the Eriksson study suggest that a combination of endurance and interval training may increase VO\textsubscript{2}\text{max} lactate concentrations in children.

### Possible Factors Accounting for Lower Peak Lactate Concentration in Children

A possible explanation for lower peak lactate concentrations in children compared to adults is altered enzyme profiles in skeletal muscles in children. In particular, there is evidence that children have reduced activity of glycolytic enzymes, and lower ratios of glycolytic to oxidative enzymes compared to adults. The results of four studies investigating enzyme profiles in children are discussed below (3, 9, 14, 17).

The earliest of these studies, by Eriksson et al. (9) investigated changes in PFK (which is a rate-limiting enzyme in glycolysis) and succinate dehydrogenase (SDH; a Kreb’s cycle enzyme), with training in five 11-year-old boys. Mean PFK activity in the vastus lateralis was 8.4 IU pretraining, which is only about 30% of typical PFK activity in adults. Pretraining SDH activity (5.4 IU), however, was slightly higher in the children than typical levels in sedentary adults. The results of Eriksson et al. have been widely cited in studies that find lower peak lactates in children compared to adults.

A 1982 study by Haralambie (17) measured the activity of a wide range of glycolytic and oxidative enzymes in the vastus lateralis of 14 children ages 13–15 years and 14 adults. In contrast to Eriksson et al. (9), Haralambie found no statistically significant differences in glycolytic enzymes, including PFK (38.6 ± 4.4 vs. 45.5 ± 10.9 IU wet weight), between the children and the adults. In discussing the differences in the findings of these two studies, Haralambie explains that Eriksson et al.’s study was based on a small number of subjects, used nonoptimal procedures, and was conducted at nonphysiologic temperatures. Haralambie also points out, however, that Eriksson’s subjects were younger, and likely at an earlier stage of sexual maturation, and that sexual maturation may be related to PFK activity.

This study also found significantly higher activity of the Kreb’s cycle enzymes ICDH, fumarase, MDH, and NADH-dehydrogenase in the children, and a lower PFK-to-ICDH ratio in the children (0.84) than in the adults (1.63). These results suggest that children in the 13- to 15-year-age group have a similar or slightly reduced glycolytic capacity compared to adults, but are capable of oxidizing pyruvate at a higher rate than adults (16).

Fournier et al. (14) investigated PFK and SDH activity before and after training in 12 boys, ages 16 to 17 years. These investigators found pretraining PFK activity of 28.1 and 29.6 IU in the two groups studied, which is lower than the results of Haralambie (17), but substantially higher than those of Eriksson et al. (9). The findings for pretraining SDH activity in the two groups (6.4 and 8.2 IU) were similar to those found by Eriksson et al. These results suggest that children as old
as 17 years may have moderately reduced PFK activity, and moderately greater SDH activity compared to adults.

To determine changes in skeletal muscle enzyme profiles in children with age and sexual maturation, Berg et al. (3) compared enzyme activities in three groups of children: prepubertal (aged 6.4 ± 2.1 years); circumpubertal (aged 13.5 ± 1.3 years); and postpubertal (aged 17.1 ± 0.8 years). The investigators measured the activity of several glycolytic enzymes, several Kreb’s cycle enzymes, and creatine kinase. Significant increases were found in the glycolytic enzymes aldolase and pyruvate kinase, and a decrease was found in the activity of the oxidative enzyme fumarase, between the prepubertal and postpubertal groups. In addition, LDH activity was significantly higher in the middle age group than the other two groups, and was higher in the postpubertal group than in the prepubertal group. These findings generally agree with the findings of the studies discussed above in that the activity of the glycolytic enzymes tended to increase, while the activity of the oxidative enzymes tended to decrease, with age.

The results of these studies suggest that children have somewhat lower levels of glycolytic enzymes, including PFK, which is consistent with the lower peak lactate concentrations observed in children compared to adults. The evidence suggests that there is a lower ratio of glycolytic to oxidative enzyme activity in children, which could be related to reduced production of lactic acid, increased oxidation of lactate, or both.

Despite the evidence that children have altered skeletal muscle enzyme profiles compared to adults, there is no indication whether these differences are the primary cause of the lower observed lactate concentrations in children during maximal exercise, or whether other factors may also contribute. A number of alternative explanations have been proposed to explain why children may have a reduced ability to accumulate high levels of lactate during exercise compared to adults, including (a) lower glycogen concentration and lower rate of glycogen utilization, particularly in prepubescent children (18, 30); and (b) lower sympathetic activity during exercise in children (30). Reduced sympathetic activity may result in reduced vasoconstriction to the liver and increased lactate removal by that organ. This explanation supports the hypothesis that lower lactate levels in children are due to a differential in lactate removal between children and adults, rather than differences in lactate production or oxidation. An additional result of reduced sympathetic activity in children may be a reduced insulin concentration. Wirth et al. (29) found an increase in lactate concentration with an age-dependent increase in insulin concentration. The authors hypothesize that insulin level may influence lactate level and that insulin secretion may be determined, at least in part, by catecholamines.

As the literature on lactate metabolism evolves, additional mechanisms for reduced lactate concentrations in children are suggested. Given the complex nature of lactate metabolism, perhaps lower lactate concentrations in children are due to the interaction of several factors and not just one primary factor. In particular, since lactate is both produced and oxidized within skeletal muscles, and children have been found to have lower ratios of glycolytic to oxidative enzymes (16), it seems plausible that children produce less lactate than adults, but that they may also oxidize lactate more quickly than adults and, therefore, do not accumulate high lactate concentrations. Their altered ratio of glycolytic to oxidative enzymes may explain, at least in part, the lower peak blood lactate concentrations observed in children compared to adults, because there would be a tendency for more pyru-
vate to be oxidized, and less to be reduced to lactate. In addition, children may have greater dilution of lactate as it diffuses from skeletal muscles into the blood due to their higher total water content compared to adults.

**Conclusions**

The balance of evidence on peak lactate concentrations and skeletal muscle enzyme activity suggests that glycolytic capacity may not be fully developed in children. Peak lactate concentrations have consistently been lower in children than adults. The precise relationship between age and the ability to generate peak lactate concentrations is unclear, due to a variety of factors: (a) methodological differences in blood assay procedures between studies, (b) use of inappropriate or variable exercise protocols to elicit peak lactate concentrations, and (c) possible differences in the motivation to exercise maximally. Although it appears that the ability to generate peak lactate concentrations increases with age, this increase may occur throughout childhood or may only be related to sexual maturation. Further investigation is warranted (a) to determine the magnitude of any difference in the ability to achieve high peak lactate concentrations using an exercise protocol specifically developed to maximally utilize glycolytic capacity, and (b) to elucidate the causal factors contributing to that difference.

**References**


