

TITLE

Muscle metabolism changes with age and maturation: How do they relate to youth sport performance?

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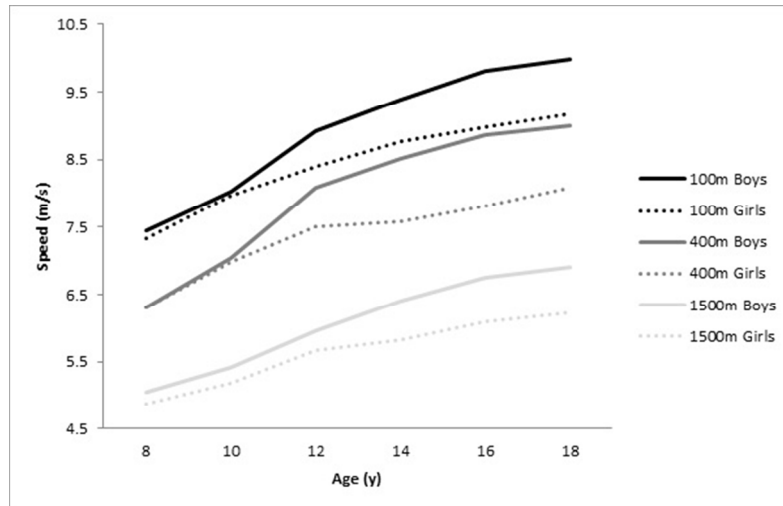
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British Journal of
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**MUSCLE METABOLISM CHANGES WITH AGE AND
MATURATION WITH REFERENCE TO YOUTH SPORT
PERFORMANCE**

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The 100m sprint is primarily supported by the catabolism of phosphocreatine and anaerobic glycolysis with ~10% of the energy being provided by aerobic metabolism. During youth, a 400m sprint is ~60-70% supported by anaerobic metabolism, predominantly glycolysis, with minor support from aerobic sources. The 1500m is a ~80% aerobic event although increases in pace (e.g. final sprint) have high anaerobic components

203x162mm (96 x 96 DPI)

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3 **MUSCLE METABOLISM CHANGES WITH AGE AND MATURATION WITH**
4 **REFERENCE TO YOUTH SPORT PERFORMANCE**
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29 **Narrative review designated for the IPHP edition (injury prevention + health promotion**
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Abstract

Aim: To provide an evidence-based review of muscle metabolism changes with sex- age- and maturation with reference to the development of youth sport performance.

Methods: A narrative review of data from both invasive and non-invasive studies, from 1970-2015, founded on personal databases supported with computer searches of PubMed and Google Scholar.

Results: Youth sport performance is underpinned by sex- age- and maturation-related changes in muscle metabolism. Investigations of muscle size, structure and metabolism; substrate utilization; pulmonary oxygen uptake kinetics; muscle phosphocreatine kinetics; peak anaerobic and aerobic performance; and fatigue resistance; determined using a range of conventional and emerging techniques present a consistent picture. Age-related changes have been consistently documented but specific and independent maturation-related effects on muscle metabolism during exercise have proved elusive to establish. Children are better equipped for exercise supported primarily by oxidative metabolism than by anaerobic metabolism. Sexual dimorphism is apparent in several physiological variables underpinning youth sport performance. As young people mature there is a progressive but asynchronous transition into an adult metabolic profile.

Conclusion: The application of recent developments in technology to the laboratory study of the exercising child and adolescent has both supplemented existing knowledge and provided novel insights into developmental exercise physiology. A sound foundation of laboratory-based knowledge has been established but the lack of rigorously designed child- and sport-specific testing environments has clouded the interpretation of the data in real life situations. The primary challenge remains the translation of laboratory research into the optimisation of youth sports participation and performance.

Abstract: 249 words

Text: 3997 words

What are the new findings?

Physiological data collected over the last 45 years using techniques ranging from invasive muscle biopsy studies to non-invasive ^{31}P magnetic resonance spectroscopy studies present a remarkably consistent picture:

- Children are better equipped for exercise supported by oxidative metabolism than by anaerobic metabolism.
- As children mature there is a progressive but asynchronous transition into an adult metabolic profile with a greater increase in performances supported by anaerobic metabolism than in performances supported by oxidative metabolism.
- Resistance to fatigue and the ability to recover from bouts of high-intensity exercise undergo a gradual decline from childhood into young adulthood in males whereas in females an adult profile is established by mid-puberty.

Laboratory data are consistent but may lack ecological validity:

- The primary challenge remains the translation of what is discovered in the laboratory to optimising youth sport participation and performance

How might it impact on clinical practice in the near future?

This review provides clinicians with current knowledge of **muscle metabolism** during exercise in youth. It provides an evidence-based foundation to inform decision-making by clinicians and others involved in promoting optimal athlete development during youth.

Introduction

To inform the IOC Consensus Statement on Youth Athlete Development this paper provides a narrative review of age- and maturation-related changes in muscle metabolism which influence youth sport performance. The review is founded on personal databases supported with computer searches of PubMed and Google Scholar and covers the period 1970-2015. The contribution of other physiological variables to youth sport performance is acknowledged but they are addressed elsewhere in this Special Issue and integrated in the Consensus Statement. To meet the challenge of synthesising this vast topic area the paper assembles studies into coherent sections related to methodology. It critically reviews data within each section and compares findings across methodologies to integrate current knowledge.

The ethics of involving minors who volunteer to participate in research are discussed elsewhere in the Consensus Statement. Methodological limitations are discussed in the section introductions and, unless individually flagged, only rigorous studies are cited. Muscle biopsy studies with young people are sparse. Some biopsy studies with methodological flaws have therefore been included if they have significantly influenced paediatric exercise physiology over several decades but have not been replicated, usually for ethical reasons. In this case, explicit caveats about relevant aspects of the methodology are emphasised and where relevant sample sizes are noted. Each section summary reports a consensus of what is known in relation to study limitations.

Muscle biopsy studies

Muscle biopsies are carried out almost routinely in adult investigations but ethical considerations have restricted the use of the technique with young people. Sparse studies of healthy youth have generally involved biopsies of the vastus lateralis of small samples of boys and focused on resting and post-exercise measures. **Participants have been classified by age and independent effects of maturation have not been assessed even though several studies refer to 'adolescents'**. The interpretation of data from muscle biopsy studies is further confounded by large inter-individual variations in fibre profiles and comparisons being made with adult data from previously published studies. Data should therefore be interpreted cautiously although some consistent patterns have emerged.

Muscle fibre size and type

Autopsy data have indicated that muscle fibre size increases in an almost linear manner with a ~20 fold increase in the cross-sectional area of both type I and type II fibres from birth to young adulthood. The % of type I fibres in males has been noted to decrease with age from 10-19 years. Trends in females are less consistent, possibly due to methodological artefacts as data from girls are sparse¹⁻³. Statistically significant sex differences in % of type I fibres during youth have not been reported but despite underpowered experimental designs there is a consistent trend with 15-24 year-old males presenting 4-15% more type I fibres than similarly aged females biopsied in the same study⁴⁻⁷.

Muscle energy stores

In the 1970s, Eriksson et al.⁸⁻¹² published a series of influential studies of 11.6-15.5 year-old boys (n=8 or 9 per year). Resting adenosine triphosphate (ATP) stores were observed to be invariant with age but phosphocreatine (PCr) and glycogen stores increased by ~60% over the age range studied^{8,12}. Recent work using magnetic resonance spectroscopy (³¹PMRS) and modelling equations to estimate and compare high energy phosphates confirmed Eriksson's data on the age invariance of ATP concentration but no differences in PCr concentrations between 10 year-old boys and adults were found¹³.

Muscle enzymes activity

Eriksson et al.¹¹ reported resting phosphofructokinase (PFK) and succinic dehydrogenase activities in 11 year-olds to be ~33% and ~125% respectively of values they had previously reported for adults¹⁴. Subsequent studies confirmed the activities of oxidative enzymes to be higher in 13-15 year-olds (n=14) than in young adults (n=14)¹⁵, higher in 6 year-olds (n=8) than in 13 (n=12) and 17 year-olds (n=13)¹⁶, and the activities of anaerobic enzymes to be lower in 3-11 year-olds (n=20) compared to adults (n=12)¹⁷. In contrast the activities of anaerobic enzymes were noted to be similar in 13-15 year-olds and adults although a lower ratio of glycolytic to oxidative enzyme activities was reported in the teens, with the ratio of PFK/isocitric dehydrogenase activity 93% higher in adults¹⁵.

Muscle and blood lactate

Eriksson et al.¹² observed muscle PCr and glycogen stores to gradually decline following exercise of increasing intensity with glycogen depletion being three times greater in 15 year-

olds than in 11 year-olds. The depletion of glycogen was reflected by a corresponding increase in muscle lactate production which was higher in older boys. In his thesis Eriksson⁹ hypothesised a maturation effect on muscle lactate production as he observed it to be 'almost significantly' correlated with testicular volume in eight 13 year-old boys. He postulated that boys' blood lactate accumulation (BLa) would reflect their muscle lactate production but subsequent studies have found the interpretation of BLa as a surrogate of muscle lactate to be clouded by methodological issues including mode of exercise, exercise protocol, time of sampling, site of sampling and assay technique¹⁸⁻²⁰.

During and immediately following exercise the blood lactate/pyruvate ratio rises in an age-related manner from 7-17 years²¹. Studies have consistently demonstrated that the lactate threshold (TLAC) expressed as % peak oxygen uptake (peak VO₂) is negatively correlated with age^{22,23}. A positive relationship between age and peak BLa is generally^{24,25} but not always²² observed. A compelling theoretical argument can be made for a maturational effect on the production of muscle lactate and BLa^{19,25} but empirical studies have consistently failed to detect an independent effect of maturation on BLa during exercise^{22,26,28}. An investigation using multiple regressions to examine the effect of salivary testosterone upon the blood lactate responses to exercise of 50 12-16 year old boys observed no significant independent effect of testosterone on BLa²⁶. An analysis of 200 (100 girls) 12 year-olds classified into the maturity stages described by Tanner²⁷ found no relationship between stage of maturation and post- Wingate anaerobic test (WAnT) BLa²⁸. A similar study of 119 11-16 year-old boys and girls reported no relationship between submaximal or post-peak VO₂ BLa and maturation²².

Summary: Taken together muscle biopsy and blood lactate studies strongly suggest that children have a well-developed capacity for oxidative metabolism during exercise but may be disadvantaged in activities predominantly supported by anaerobic metabolism when compared to adults. Muscle biopsy studies reveal little about potential maturational effects on performance and although BLa during exercise is related to age an independent relationship with maturation remains to be proven.

Substrate Utilization

Conventionally, the respiratory exchange ratio (RER) is used to estimate substrate utilization. The RER is, however, unable to quantify the contribution of protein, or to clarify the various

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3 lipid (intramuscular triglyceride vs blood fatty acids in the blood) and carbohydrate (CHO)
4 (muscle glycogen vs blood glucose) sources. It is also influenced by prior exercise and
5 nutritional intake before and during exercise. When comparing children with adults
6 confounding factors include children's relative hyperventilation, reduced capacity for carbon
7 dioxide storage, faster pulmonary $\dot{V}O_2$ ($p\dot{V}O_2$) kinetics, earlier $p\dot{V}O_2$ steady state attainment
8 during moderate intensity exercise and smaller $p\dot{V}O_2$ slow component (SC), which results in
9 a time-dependent increase in $\dot{V}O_2$, in exercise, above the T_{LAC} ²⁹⁻³¹.

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Despite the limitations of the technique, there is a consensus from numerous studies that
young people oxidise a higher % of lipids and a lower % of CHOs for energy at a given
relative exercise intensity than adults although age-related differences in substrate use are
more evident in males than in females^{29,30,32,33}. High rates of lipid oxidation during exercise
decline during maturation and there is evidence that the development of an adult fuel-
utilization profile occurs during the transition from mid- to late- puberty, at least in males^{34,35}.

Further insights have recently emerged from a series of innovative studies using RER and ¹³C
stable isotope methodology to investigate the effects of exogenous CHO (¹³C-enriched 6%
CHO in the form of a drink, CHO_{exo}) on substrate use during sub-maximal exercise. In boys,
CHO_{exo} oxidation rate expressed as a % of total energy expenditure was found to be inversely
related to serum testosterone levels. The utilization of CHO_{exo} as an energy source was
strongly related to pubertal status with the highest oxidation rates observed in pre- and early
pubertal boys and the lowest in mid- to late- pubertal boys regardless of chronological age. In
contrast the CHO_{exo} oxidation rate in girls was not related to age or pubertal status despite
large differences in circulating oestradiol levels³⁶⁻³⁸.

Summary: Data derived from the RER during submaximal exercise infer that with their
enhanced ability to oxidise lipids and spare glycogen children are well-equipped for long-
term moderate intensity exercise. The optimal CHO feeding regime to sustain endurance
performance is unknown, but evidently is related to maturation in boys.

Pulmonary oxygen uptake kinetics and muscle phosphocreatine kinetics

A high peak $\dot{V}O_2$ and/or peak power are prerequisites of elite performance in many sports but
in others it is the ability to engage in rapid changes in exercise intensity which is paramount.

Under these circumstances, it is the transient kinetics of pVO₂ and PCr which best reflect the integrated response of the pulmonary, circulatory and muscle metabolic systems.

In adults the measurement of muscle VO₂ using the Fick technique has been shown to agree with pVO₂ within ~10%³⁹. This relationship has been confirmed by simultaneously determining adults' pVO₂ kinetics and intramuscular PCr kinetics in a MR scanner⁴⁰. The work has not been replicated with children but a close relationship between children's intramuscular PCr kinetics during prone quadriceps exercise in a MR scanner and pVO₂ kinetics during upright cycling at both the onset and offset of exercise has been demonstrated⁴¹. These studies demonstrate that pVO₂ kinetics has the potential to provide a non-invasive window into muscle metabolism during exercise.

Pulmonary oxygen uptake kinetics

Resolution of pVO₂ kinetics in children has proved challenging. As the range of potential exercise intensities is lower in children than in young adults the scope of the metabolic transitions to exercise possible within each exercise domain is reduced⁴². In addition, children's inherently erratic breathing pattern, low signal-to-noise ratio and large inter-breath fluctuations reduce confidence in resolving parameters of the pVO₂ kinetics response, in particular the primary time constant (τ)⁴³. The issue is further compounded by several studies not adhering to strict definitions of exercise domains, using sub-optimal numbers of repeated transitions, not reporting confidence intervals and applying a confusing array of analytical models with limited physiological rationales⁴⁴⁻⁴⁷.

The advent of on-line breath-by-breath analysis systems and appropriate mathematical modelling procedures has allowed rigorous investigation of the kinetics of pVO₂ during youth⁴⁴⁻⁴⁷. Simultaneous analysis of pVO₂ kinetics, beat-by-beat heart rate kinetics, cardiac output (using thoracic impedance), blood deoxygenation kinetics (using near infra-red spectroscopy) and the introduction of experimental models such as priming exercise, manipulation of pedal rates and control of respiratory gases (e.g. hypoxic/hyperoxic stimuli) have provided intriguing insights into paediatric exercise metabolism^{31,48-51}.

Slow pVO₂ kinetics is associated with a greater depletion of intramuscular high energy phosphates and accumulation of lactate and hydrogen ions. The mechanisms underlying the SC in exercise above TLAC remain speculative but appear to be a function of muscle fibre

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3 distribution, motor unit recruitment and the matching of oxygen delivery to active muscle
4 fibres. In adults it has been demonstrated that the τ of the exponential rise of the pVO_2
5 kinetics response to exercise corresponds with indices of aerobic fitness and that the
6 amplitude of the SC is closely associated with the fatigue process and related to indices of
7 anaerobic fitness^{52,53}. In contrast, during youth no relationships between peak VO_2 and τ or
8 the SC and fatigue have been reported⁵⁴⁻⁵⁶.

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14 Rigorous investigations have demonstrated unequivocally that the SC and the τ response to
15 exercise above the $TLAC$ increase with age from childhood through to young adulthood
16 although independent maturational effects remain to be demonstrated⁵⁵⁻⁵⁷. These data provide
17 compelling evidence that at the onset of exercise young people have a higher potential for
18 oxidative metabolism than adults. The attenuated SC is in agreement with young people
19 being fatigue resistant. During exercise above but not below $TLAC$ boys display a faster τ and
20 a smaller SC than girls which is also in accord with muscle biopsy data^{54,55,58}.

27 28 *Muscle phosphocreatine kinetics*

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30 ³¹PMRS studies are constrained by exercising in a small bore tube and the need to
31 synchronise data acquisition with the rate of muscle contraction which can be challenging for
32 children. Interpretation of existing paediatric ³¹PMRS data is clouded through inter-study
33 differences in body position (e.g. supine; prone), exercise protocols (e.g. intermittent
34 exercise; incremental exercise; constant intensity exercise), muscle(s) interrogated (e.g.
35 forearm; calf; thigh), types of muscle contractions (e.g. isometric; isotonic), classification of
36 participants (e.g. teens grouped by chronological age without reference to maturity; pooling
37 of data from mixed sex groups), and data normalization. Moreover, the technique is
38 expensive and sample sizes are generally small. Sparse data from ³¹PMRS studies should be
39 interpreted in the context of these limitations.

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48 Incremental exercise studies to exhaustion using ³¹PMRS have revealed age- and sex--related
49 modulation of muscle metabolism during high intensity exercise with children relying less on
50 anaerobic metabolism than adults^{59,60} and boys less than girls, possibly because of girls' more
51 advanced level of maturation in relation to chronological age⁶⁰. The age-related data have
52 been replicated during high intensity intermittent exercise although no sexual dimorphism has
53 been reported⁶¹.

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3 The interpretation of some ^{31}P MRS studies^{62,63} comparing the recovery from exercise of
4 children and adults is confounded by significant age differences in pH at exhaustion^{64,65}.
5 However, rigorous investigations have consistently observed young people's faster re-
6 synthesis of PCr during recovery from exhaustive exercise and concluded that young people
7 have a greater mitochondrial oxidative capacity than adults^{66,67}. PCr recovery from fatiguing
8 isometric exercise has been shown to be ~33% faster in boys than men with no difference in
9 PCr recovery time between girls and women⁶². Notably, the rapid recovery of skeletal muscle
10 PCr concentration has been reported to be negatively related to linear growth velocity⁶⁸.

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18 Two studies of responses to high-intensity constant work rate exercise have not identified
19 statistically significant age⁶⁹ or maturational⁷⁰ differences in metabolic responses but large
20 standard deviations, small sample sizes, and differences of ~42-66% between groups in end-
21 exercise PCr or PCr kinetics during exercise infer possible biological significance.

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26 **Summary:** Rigorously designed and executed ^{31}P MRS and pVO₂ kinetics studies with young
27 people are sparse but taken together the data support an age and/or maturation influence on
28 muscle energetics, with children relying more on oxidative metabolism during high-intensity
29 exercise than adults. Young people's pVO₂ and PCr kinetics data are consistent with an
30 enhanced oxidative enzymatic profile and sex- and age-related differences in both % of type I
31 muscle fibres and recruitment of higher threshold (type II) motor units

32 33 34 35 36 37 38 **Maximal (or peak) aerobic and anaerobic performance**

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Young people's aerobic and anaerobic performance has been assessed in laboratories for
several decades with techniques becoming more refined over time^{20,71}. Data from thousands
of young people are remarkably consistent and the relationship of aerobic and anaerobic
performance with age is well-established. The (mis)interpretation of data in relation to body
size has, however, masked physiological understanding during growth and maturation.

51 52 53 54 55 56 57 58 59 60 **Peak oxygen uptake**

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Peak VO₂ is widely recognised as the best single measure of young people's aerobic fitness
and values collated from studies of ~5000 children show boys' and girls' peak VO₂ to
increase, in an almost linear manner, by ~150% and ~80% respectively from 8-16 years⁷².
Sparse longitudinal studies generally concur with cross-sectional data⁷³. A longitudinal

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3 study, founded on 388 data points analysed using multilevel modelling, reported boys' peak
4 VO₂ to almost double from 11-17 years. Girls' values increased by ~50% and the sex
5 difference increased from ~10-35%⁷⁴.
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9 Boys' progressive rise in muscle mass accounts for much of the increasing sex difference in
10 peak VO₂ as it not only facilitates oxygen utilization but also augments venous return. Boys'
11 peak VO₂ may be further supplemented by an increase in blood haemoglobin concentration
12 during late teens⁷³⁻⁷⁵. The pre-pubertal sex difference in peak VO₂ has been attributed to
13 boys' greater stroke index⁷⁶⁻⁷⁸, boys' higher maximal arterial-venous oxygen difference⁷⁹ and
14 differences in the balance between oxygen delivery to and utilization in the muscles⁸⁰.
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21 Although the fallacy of expressing peak VO₂ in ratio with body mass (mL.kg⁻¹.min⁻¹) has
22 been documented for over 65 years⁸¹ it is still widely used. Using ratios a different picture
23 emerges with girls' peak VO₂ declining, from ~8-18 years, from ~45-35 mL.kg⁻¹.min⁻¹ and
24 boys' peak VO₂ remaining essentially unchanged at ~48-50 mL.kg⁻¹.min⁻¹^{72,73}. This
25 methodology is informative in relation to the performance of youth athletes who carry their
26 body mass (e.g. track athletes)⁸². Elite youth athletes have values ~50% higher than their
27 untrained peers when peak VO₂ is expressed in this manner⁸³. However, during growth body
28 mass increases at a greater rate than peak VO₂ and comparative studies expressing peak VO₂
29 in ratio with body mass favour children and have confused our understanding of aerobic
30 fitness during growth and maturation⁸⁴⁻⁸⁶. When body mass is controlled for appropriately
31 using allometry or multilevel modelling boys' values have been shown to progressively
32 increase from childhood into young adulthood. Girls' peak VO₂ increases from childhood to
33 mid-teens and then shows no observable decline into young adulthood⁸⁷.
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45 *Short-term power output*

46 **Laboratory research on** anaerobic performance has focused on the assessment of external
47 power output using variants of the WAnT in which cycling peak power (CPP) is determined
48 over a 1s or 5s period and cycling mean power (CMP) over the 30s test period. CMP,
49 although primarily supported by anaerobic energy sources, includes an unquantified
50 contribution from aerobic metabolism which has been estimated to vary between ~10-44%
51 and is higher in young people than in adults probably due to their faster pVO₂ kinetics^{88,89}.
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3 There is an almost linear increase in CPP from ~7-12 years, with girls often outscoring
4 similar aged boys due to their more advanced stage of maturation. From ~13 years boys
5 experience a marked increase in CPP through to young adulthood resulting in a ~50% sex
6 difference by age 17 years⁹⁰⁻⁹². Using the force-velocity test to determine CPP (or optimal
7 peak power, OPP) girls' and boys' values have been reported to increase by 295% and 375%,
8 respectively, from 7-17 years⁹².

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15 CPP increases at a greater rate than body mass through adolescence. The determinants of
16 enhanced CPP during maturation include changes in muscle fibre size, muscle fibre type and
17 muscle metabolism. A series of longitudinal studies has demonstrated MRI-determined thigh
18 muscle volume to be prominently and significantly related to CPP⁹³ and OPP⁹⁴. However,
19 neuromuscular factors, particularly the ability of young adults to better recruit and more fully
20 use higher threshold (type II) motor units than children also play a crucial role in optimising
21 OPP and/or CPP⁹⁵.

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28 Independent of age there is an asynchronous contribution of maturation to peak anaerobic and
29 aerobic performance, which was clearly demonstrated in a study of 200 (100 girls) 12 year-
30 olds. Children were classified according to the stages of maturation described by Tanner²⁷.
31 Boys and girls in maturity stage 4 for pubic hair were reported to have, respectively, 32% and
32 25% higher peak VO₂ and 66% and 51% higher CPP scores than those in stage 1. With body
33 mass appropriately controlled for using allometry, the differences between stages 4 and 1 in
34 peak VO₂ and CPP were 14% and 31%, respectively, in boys and 12% and 20%,
35 respectively, in girls^{28,96}.

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43 **Summary:** Both peak anaerobic and aerobic performances increase with age and maturation
44 but at different rates. Longitudinal data on the same children show that from 12-17 years CPP
45 increases by ~65% in girls and ~120% in boys. Relative increases in peak VO₂ are somewhat
46 smaller at ~50% in girls and ~70% in boys^{74,93}.

47 48 49 50 51 52 53 **Recovery from short-term maximal- or high-intensity exercise**

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56 The rate of recovery from maximal- or high-intensity exercise has been studied using a range
57 of methodologies, including intermittent 'all-out' cycling or running tests or maximal
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3 isokinetic contractions on a variety of ergometers and dynamometers. Young people have
4 consistently been reported to recover more rapidly than adults from intermittent bouts of
5 maximal or high-intensity running^{97,98} and cycling exercise^{99,100} and maximal isokinetic
6 contractions^{101,102}. However, analysing the relative fatigue and recovery rate of children and
7 adults is complex as their maximal short-term performance or power output is not
8 comparable. Subjective exertion may be identical but maximal power output is lower in
9 children and this applies to any given percentage of maximal power. It can therefore be
10 argued that as children generate lower power their faster recovery from high-intensity
11 exercise is not directly comparable to adults because they have less to recover from¹⁰³.
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20 Nevertheless, the consensus from numerous investigations is that the ability to recover from
21 bouts of high-intensity exercise undergoes a gradual decline from childhood to adulthood in
22 males whereas in females an adult profile is established by 14-15 years of age¹⁰⁴⁻¹⁰⁶. This
23 resistance to fatigue has been attributed to young people having enhanced oxidative capacity,
24 faster recovery kinetics of cardiorespiratory variables, more rapid PCr re-synthesis,
25 differential motor unit recruitment and usage, better acid-base regulation and lower
26 production and/or more efficient removal of metabolic by-products than adults^{103,105,106}.
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33 **Summary:** Children recover from short-term maximal- and high-intensity exercise faster than
34 adults. There are persuasive physiological hypotheses for their resistance to fatigue in this
35 context but the exercise models used cannot refute the view that adults' slower recovery is
36 simply a direct consequence of their ability to generate more power.
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41 **Sport performance across maturation**

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44 It is not unusual to see pre-school children in sport induction programmes and participating in
45 organised competitive sport as young as 6-8 years of age. Youth athletes therefore experience
46 several years of training and competition across maturation. Success in youth sport is
47 underpinned by a range of physiological variables which operate in a sport-specific manner
48 and are dependent on the progress of individual biological clocks. Their influence can be
49 illustrated by perusing the asynchronous development of world best performances in relation
50 to age and sex in track events primarily supported by different energy systems (Fig 1).
51 Changes in body size and composition and increases in muscle strength significantly affect
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3 performance but track world best performances illustrate quite nicely the changes in muscle
4 **metabolism** described herein.
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10 The value of applying laboratory findings to youth athlete development is readily apparent.
11 However, experimental limitations such as exercising within a constrained space (e.g.
12 ³¹PMRS), exercising on laboratory rather than sport-specific ergometers (e.g. running on
13 elevated treadmills or cycling against high resistances), extrapolating resting and recovery
14 measures to exercise situations (e.g. muscle biopsies), and the general lack of rigorously
15 designed, ecological, child- and sport-specific testing environments have confounded the
16 practical application of laboratory data to real life situations.
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22 The specific effect and magnitude of changes in physiological variables measured in the
23 laboratory on subsequent athletic performance during youth are still to be elucidated. Will a
24 change in a single physiological variable impact on subsequent performance? Should coaches
25 focus on maintaining one physiological variable (e.g. peak VO₂) in parallel with improving a
26 related variable (e.g. running economy)? It may take several years of training for some
27 factors related to performance to improve and this is compounded in youth by asynchronous
28 development of aspects of **muscle metabolism**. In a context in which extremely small changes
29 in performance can be the difference between winning and losing should the laboratory
30 scientist be more concerned with statistical significance or the smallest worthwhile effect?
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38 We still have much to learn about the application of paediatric physiology to athletic
39 performance but the development of a comprehensive scientific foundation and an
40 understanding of both the benefits and limitations of data generated in the laboratory provide
41 a platform to inform decision-making by those involved in promoting optimal athlete
42 development during youth.
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47 **Conclusion**

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50 The introduction of recent developments in technology has provided new insights into
51 developmental muscle metabolism. Data from a number of sources are remarkably consistent
52 but lack ecological validity. Nevertheless, coaches and exercise scientists working in youth
53 sport need to understand the changes (or plateaus) in performance which might be more
54 related to individual biological clocks than training if they are to provide optimum support to
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young athletes. Paediatric sport science is rapidly evolving and a sound foundation of knowledge has been established. The primary challenge remains the translation and utility of what is discovered in the laboratory to optimising youth sports participation and performance.

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Figure

Title: Average speed of World best performances in 100m, 400m and 1500m track events in relation to age and sex

Legend: The 100m sprint is primarily supported by the catabolism of phosphocreatine and anaerobic glycolysis with ~10% of the energy being provided by aerobic metabolism. During youth, a 400m sprint is ~60-70% supported by anaerobic metabolism, predominantly glycolysis, with minor support from aerobic sources. The 1500m is a ~80% aerobic event although increases in pace (e.g. final sprint) have high anaerobic components.