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INVITED PAPER
SPECIAL ISSUE: 'GAPS IN OUR KNOWLEDGE'

Aerobic fitness and trainability in healthy youth: Gaps in our knowledge

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Abstract

Peak oxygen uptake ($\dot{V}O_2$) is widely recognised as the criterion measure of young people's aerobic fitness. Peak $\dot{V}O_2$ in youth has been assessed and documented for over 75 years but the interpretation of peak $\dot{V}O_2$ and its trainability are still shrouded in controversy. Causal mechanisms and their modulation by chronological age, biological maturation and sex remain to be resolved. Furthermore, exercise of the intensity and duration required to determine peak $\dot{V}O_2$ is rarely experienced by most children and adolescents. In sport and in everyday life young people are characterized by intermittent bouts of exercise and rapid changes in exercise intensity. In this context it is the transient kinetics of pulmonary (p) $\dot{V}O_2$ which best describe aerobic fitness. There are few rigorously determined and appropriately analysed data from young people's p $\dot{V}O_2$ kinetics responses to step changes in exercise intensity. Understanding of the trainability of p $\dot{V}O_2$ kinetics is principally founded on comparative studies of trained and untrained youth and much remains to be elucidated. This paper reviews peak $\dot{V}O_2$, p $\dot{V}O_2$ kinetics and their trainability in youth. It summarizes 'what we know', identifies significant gaps in our knowledge, raises relevant questions, and indicates avenues for future research.

Introduction

Aerobic fitness can be defined as the ability to deliver oxygen to the muscles and to utilize it to generate energy to support muscle activity during exercise. In one paper it is not possible to do justice to all indicators of aerobic fitness (e.g. blood lactate thresholds, ventilatory threshold (T_{VENT}), exercise economy, exercise recovery kinetics, etc) and the focus herein is on arguably the two most important markers of aerobic fitness, peak oxygen uptake (peak $\dot{V}O_2$) and pulmonary oxygen uptake (p$\dot{V}O_2$) kinetics responses at the onset of exercise. We recognize that aerobic fitness has a genetic component with both the heritability and trainability of peak $\dot{V}O_2$ estimated to be ~50%, but genetic influences on youth aerobic fitness are outside the scope of this paper.

Peak $\dot{V}O_2$, determined in a laboratory as the highest rate at which oxygen can be consumed by the muscles during an exercise test to exhaustion, is widely recognized as the best single measure of aerobic fitness in youth. However, in everyday life young people's spontaneous play and participation in organised sport consist largely of intermittent exercise bouts of varying duration and include rapid changes in exercise intensity. Under these conditions peak $\dot{V}O_2$ can be conceived as a laboratory-based variable of investigative convenience and it is the transient kinetics of p$\dot{V}O_2$ which best describe aerobic fitness.

The development of peak $\dot{V}O_2$ during growth and maturation has been comprehensively documented and the trainability of peak $\dot{V}O_2$ extensively examined but the literature is not without controversy. In contrast, there are few rigorously controlled and appropriately analysed studies of young people's p$\dot{V}O_2$ kinetics responses to step changes in exercise intensity. Studies of the trainability of p$\dot{V}O_2$ kinetics in youth are even sparser with an emphasis on comparisons between trained and untrained swimmers and footballers. The

balance of evidence and supposition is therefore somewhat different across the two variables.

In this invited paper we were challenged to consider 'what we know and what we don't know' about aerobic fitness and trainability in healthy youth. The paper brief included a restriction on reference citations (10-20) so throughout we have referred readers to recent reviews to access source material and cited few individual studies. Our objectives are to outline current understanding of peak $\dot{V}O_2$ and $p\dot{V}O_2$ kinetics, identify significant gaps in knowledge, reveal controversies in interpretation of data, raise relevant research questions, and indicate avenues for future research.

Peak oxygen uptake

Peak $\dot{V}O_2$ is the most researched variable in paediatric exercise science and the extant literature has been critically analysed in numerous reviews. Herein we will outline 'what we know' and focus on key questions which still cloud our understanding of peak $\dot{V}O_2$ and its trainability during childhood and adolescence.

Chronological age, biological maturation and sexual dimorphism

Cross-sectional studies reveal that boys' and girls' peak $\dot{V}O_2$ (in $L \cdot min^{-1}$) increases, in a near-linear manner, by ~150% and ~80% respectively from 8-16 years. Longitudinal studies indicate that the largest annual increase in boys' peak $\dot{V}O_2$ occurs between 13-15 years whereas data from girls suggest that following a progressive rise from 8-13 years there is a tendency for peak $\dot{V}O_2$ to plateau in the mid-teens. The sex difference increases from ~10% to ~35% from 10-16 years (5, 6). But,

    i)   *why are there sex differences in young people's peak $\dot{V}O_2$?*

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There is a consensus in the literature that in puberty the dominant influence on peak $\dot{V}O_2$ is muscle mass. Boys' greater muscle mass not only facilitates the utilization of oxygen during exercise but through the peripheral muscle pump also supplements the venous return to the heart, boosting stroke volume (SV). Boys' peak $\dot{V}O_2$ may be augmented further by a greater increase in blood haemoglobin concentration [Hb] during the late teens. In addition, biological maturation has an undefined but positive effect on peak $\dot{V}O_2$ which is independent of chronological age, body size, muscle mass and [Hb] in both boys and girls (5, 6).

Explanations of pre-pubertal sexual dimorphism in peak $\dot{V}O_2$ are contentious but the recent application of new technologies to the study of paediatric exercise physiology has enabled new avenues of research (4, 5). Sex differences in peak $\dot{V}O_2$ prior to puberty have been commonly attributed to boys' greater cardiac size and stroke index. Studies using Doppler echocardiography have reported SV max to be the sex distinguishing variable at peak $\dot{V}O_2$ but have also offered conflicting views on the relative contribution of cardiac size and cardiac function (e.g. ref 17 vs ref 18). In contrast, a study using thoracic bioimpedance to estimate cardiac output ($\dot{Q}$) reported that at peak $\dot{V}O_2$ pre-pubertal boys had a higher arteriovenous oxygen difference (a-vO_2 diff) than girls, with no sex difference in $\dot{Q}$ or SV (20). In the same study, magnetic resonance imaging (MRI) at rest revealed no significant sex differences in left ventricular muscle mass, left ventricular muscle volume, posterior wall thickness, septal wall thickness, left ventricular end diastolic chamber volume, or left ventricular end systolic chamber volume. A recent publication using near-infra red spectroscopy (NIRS) to estimate changes in deoxyhaemoglobin [HHb] during exercise reported that sex-specific differences in the balance between oxygen delivery and oxygen utilization might contribute to pre-pubertal sex differences and potentially to age-related changes in peak $\dot{V}O_2$ (13).

Clearly more research using recent non-invasive technological advances is required to explain pre-pubertal sex differences in peak $\dot{V}O_2$.

Growth and biological maturation

Peak $\dot{V}O_2$ is strongly correlated with body mass and paediatric exercise scientists have traditionally attempted to control for this by simply dividing peak $\dot{V}O_2$ by body mass and expressing it as a ratio ($mL \cdot kg^{-1} \cdot min^{-1}$). When peak $\dot{V}O_2$ is expressed in this manner a different picture emerges from that apparent when absolute values ($L \cdot min^{-1}$) are used. Boys' mass-related peak $\dot{V}O_2$ remains essentially unchanged, from 8-18 years, whilst a progressive decline is apparent in girls (5, 6). But,

> ii) *how meaningful is the interpretation of peak $\dot{V}O_2$ using ratio scaling during growth and maturation?*

In puberty body mass increases at a greater rate than peak $\dot{V}O_2$ and studies expressing peak $\dot{V}O_2$ in ratio with body mass penalise more mature (heavier) individuals and have confounded our understanding of aerobic fitness during childhood and adolescence. For example, studies using ratio scaling have reported no effect of biological maturation on peak $\dot{V}O_2$ whereas allometric analyses have clearly demonstrated biological maturation to have an independent effect on peak $\dot{V}O_2$ over and above the effects of chronological age and body size. In direct conflict with ratio scaling, when body mass is controlled using allometry or multi-level modelling boys' values of peak $\dot{V}O_2$ progressively increase from childhood into young adulthood. Girls' peak $\dot{V}O_2$ increases from childhood to mid-teens and then shows no observable decline into young adulthood (5, 19).

Ratio scaling of peak $\dot{V}O_2$ can be informative, for example, in relation to the performance of young athletes who carry their body mass. On the other hand, it is futile to compare and contrast the aerobic fitness of young athletes from different sports (e.g. artistic gymnasts with rugby players) in $mL \cdot kg^{-1} \cdot min^{-1}$. The fallacy of per-body mass scaling has been empirically documented for over 65 years. Yet, papers using ratio scaling to describe peak $\dot{V}O_2$ during growth and maturation are still published uncritically in the paediatric literature.

Informed discussion of contextual reporting of peak $\dot{V}O_2$ and appropriate action by journal reviewers and editors are required to clarify interpretation of peak $\dot{V}O_2$ during growth and maturation.

Trainability of peak $\dot{V}O_2$

Optimal training programmes

The vast majority of training intervention studies that have reported increases in children's peak $\dot{V}O_2$ have used sustained periods of constant intensity exercise training (CIET) (e.g. duration ~20 min; intensity ~85% of maximum heart rate (HR)). But,

    iii)   *is CIET the optimal training programme for enhancing peak $\dot{V}O_2$ in youth?*

High-intensity interval training (HIIT), which consists of repeated sessions of brief, intense bouts of exercise interspersed by short periods of rest or low intensity exercise, has been shown to be an effective and time-efficient approach to training in adults (9). As pre-pubertal children recover more rapidly from high-intensity exercise than adults (4) it is surprising that HIIT protocols have not been prioritized in paediatric training studies. It was demonstrated over 20 years ago that pre-pubertal girls could improve their peak $\dot{V}O_2$ through either CIET or HIIT (11) but it is only recently that a concerted research effort has focused on HIIT as a

means of enhancing aerobic fitness in youth. A recent systematic and meta-analytic review of the literature identified eight studies which met its eligibility criteria and concluded that HIIT is an effective means of enhancing peak $\dot{V}O_2$ in adolescence (7). However, it is readily apparent from the reviewed literature (only eight studies met the eligibility criteria for the meta-analysis) that more rigorous research procedures are required to establish optimal and sustainable HIIT protocols with adolescents. Studies with pre-pubertal children are few and contradictory (3).

Further investigations with well-defined participant groups and both CIET and HIIT protocols are required to tease out the relative efficacy of CIET and HIIT in increasing peak $\dot{V}O_2$ in youth.

Biological maturation

Following reports that training of pre-pubertal children did not induce significant changes in peak $\dot{V}O_2$ the existence of a 'maturational threshold' was hypothesized. It was proposed that there is a 'trigger point', influenced by the modulating effects of the hormones which initiate puberty, below which the effects of training will be minimal or will not occur. This notion of pre-pubertal children having no response or a blunted response to endurance training has become embedded in the paediatric literature (3, 12). But,

    iv)   *is there really a 'maturational threshold' or 'trigger point' which governs responses to training?*

A persuasive theoretical argument can be made for a maturational effect on the response of peak $\dot{V}O_2$ to training but there is no compelling empirical evidence to support the case. A systematic review of the literature identified the intensity of the training programme as the

critical component and noted that several studies of pre-pubertal children did not employ an intense enough training program to induce changes in peak $\dot{V}O_2$. It was concluded that in studies which used a sufficiently intense training stimulus the magnitude of increase in peak $\dot{V}O_2$ was not related to chronological age, stage of maturation or sex (3).

A recent series of investigations specifically addressed the maturational threshold hypothesis. In cross-sectional studies a similar influence of training status on the physiological responses to exercise of pre-pubertal and pubertal girls was demonstrated. In a longitudinal study of 10-12 year-old trained swimmers and untrained peers, observed over three annual measurement points, it was noted that increases in peak $\dot{V}O_2$ were not associated with changes in maturation status (12, 14).

Present data support the view that the maturational threshold hypothesis remains to be proven. But, longitudinal data from both sexes with measurement occasions covering a time period which includes pre-puberty, puberty and post-puberty are required to elucidate further the possible influence of biological maturation on aerobic trainability.

Physiological mechanisms

Understanding the mechanisms underlying training-induced changes in young people's peak $\dot{V}O_2$ has been clouded by ethical and methodological issues related to the determination of $\dot{Q}$ and a-vO$_2$ diff during maximal exercise. As scant research has indicated that the maximal a-vO$_2$diff and HR max of trained young athletes are not different from those of untrained young people differences in peak $\dot{V}O_2$ have been conventionally attributed to increases in SV max. But,

v)   *do similar mechanisms underpin CIET- and HIIT-induced changes in peak $\dot{V}O_2$ in youth?*

Both morphological and functional adaptations of the myocardium have been proposed as reasons for enhanced SV following CIET. Explanatory postulates include increases in ventricular dimensions, wall thickness and mass, shortening fraction, and ejection fraction but empirical evidence is sparse and conflicting (3, 12). There are no published studies which specifically explore the mechanisms underpinning HIIT-induced changes in peak $\dot{V}O_2$ in youth. Extrapolation of findings in adults to children and adolescents must be done cautiously but data from training intervention studies with adults suggest that peripheral (oxygen utilization) rather than central (oxygen delivery) adaptations might be primarily responsible for HIIT-induced increases in peak $\dot{V}O_2$ (9).

The causal mechanisms of CIET-, HIIT- and combined CIET and HIIT-induced increases in peak $\dot{V}O_2$ in childhood and adolescence remain to be elucidated. But,

vi)   *if HIIT produces peripheral adaptations and CIET causes central adaptations in youth, would a combination of the two protocols maximize training-induced changes in young people's peak $\dot{V}O_2$?*

if so,

vii)   *which mechanisms would primarily support increases in peak $\dot{V}O_2$?*

and,

viii)   *would both CIET- and HIIT-induced responses be similarly modulated by chronological age, biological maturation and/or sex?*

Training intervention studies, including CIET, HIIT and combined CIET/HIIT programmes, in relation to chronological age, biological maturation and sex, need to be designed and implemented. The utilization of non-invasive technologies such as NIRS, MRI, MR spectroscopy (MRS), Doppler echocardiography and thoracic bioimpedance will facilitate exploration of causal mechanisms.

Responses to incremental exercise following training

A different SV response pattern during incremental exercise has been observed in trained compared to untrained children. In untrained children SV has been assumed to reach its peak at ~40-50% of peak $\dot{V}O_2$ and then plateau but in trained children SV has been noted to increase progressively to exhaustion, perhaps through elevated diastolic filling. There is no compelling empirical evidence to suggest that resting or maximal a-vO$_2$ diff in youth is affected by training but training status has been related to a rightward shift in the response pattern between these two points, with a sigmoidal oxygen extraction curve relative to % exercise intensity *(12, 14)*. But,

ix) *what is the physiological explanation for a rightward shift of a-vO$_2$ diff during incremental exercise in trained youth?*

The non-invasive estimation of $\dot{Q}$ and HR coupled with the emergence of NIRS and the ability to monitor [HHb], offers the potential to reveal new insights into the balance between SV and a-vO$_2$ diff during incremental exercise following appropriate training interventions.

Pulmonary oxygen uptake kinetics

Exploration of the p$\dot{V}O_2$ kinetics response at the onset of exercise depends upon the ability to evaluate the rate and magnitude of the respiratory gas exchange response to a given metabolic

demand. The rigorous resolution of young people's $p\dot{V}O_2$ kinetics is challenging and data from early studies are inconsistent. Recent research, using appropriate methodologies, sophisticated mathematical modelling techniques and emerging technologies, has begun to map out young people's transient responses to the onset of a step change in exercise intensity. Pulmonary $\dot{V}O_2$ kinetics responses at the onset of exercise have been critically analysed elsewhere (2, 5) and readers are referred to these reviews for detailed discussion of the extant literature and source references. Herein we will summarize current understanding (with the proviso that in several cases confirmatory evidence is required), identify significant gaps in our knowledge and raise potential research questions.

Exercise phases, exercise domains, chronological age, biological maturation and sexual dimorphism

Phase I

The initial rise in $p\dot{V}O_2$ at the onset of exercise (phase I or cardiodynamic phase) is dissociated from $\dot{V}O_2$ at the muscle due to the almost instantaneous increase in $\dot{Q}$ which is initiated by vagal withdrawal and the mechanical pumping action of the contracting muscles. But,

x)   *is the duration of phase I related to chronological age, biological maturation and/or sex?*

and,

xi)   *is the duration of young people's phase I specific to exercise domains?*

Investigations of the length of phase I and the mechanisms underlying changes with chronological age have been constrained by methodological challenges. Men have been reported to have a longer phase I than boys in response to the onset of a transition to 50% of

peak $\dot{V}O_2$, perhaps due to the shorter distance between exercising muscles and the lungs in boys. In contrast to men, boys' phase I duration does not appear to decrease at higher metabolic rates. Chronological age-related data from females are restricted to a longitudinal study of 10-13 year-olds where it was noted that over three annual measurement occasions the duration of phase I at the onset of heavy intensity exercise increased in both girls and boys (8). Pre-pubertal boys present a shorter duration cardiodynamic phase than pre-pubertal girls but why there is sexual dimorphism is unclear. It might be indicative of a more rapid increase in SV at the onset of exercise in boys than in girls, an observation noted in a study using Doppler echocardiography (17).

Little is known about chronological age- and sex-related differences (and their mechanisms) in the duration of phase I across exercise domains. Independent effects of biological maturation on phase I have not been studied. Further investigation is warranted.

Phase II

Children are characterized by a faster primary (phase II) time constant (τ) at the onset of moderate intensity exercise ($<$ T$_{VENT}$), heavy intensity exercise ($>$ T$_{VENT}$ $<$ critical power [CP]) and very heavy intensity exercise ($>$ CP $<$ peak $\dot{V}O_2$) compared with adults. Secure data from the onset of severe intensity exercise ($>$ peak $\dot{V}O_2$) are sparse. Despite significant differences in peak $\dot{V}O_2$, there is no sexual dimorphism in the primary τ at the onset of exercise below T$_{VENT}$. Intriguingly, during the transition from rest to exercise intensities above T$_{VENT}$ boys present a significantly faster primary τ than girls. Longitudinal studies have noted that at the onset of heavy intensity exercise the primary τ increases with age in both children and adolescents (2, 5). But,

xii) *why are there chronological age differences in the speed of the primary τ at the onset of exercise?*

xiii) *is there a biological maturation influence, independent of chronological age, on the speed of the primary τ at the onset of exercise?*

and,

xiv) *why is there sexual dimorphism in the speed of young people's primary τ at the onset of exercise above but not below T_{VENT}?*

Children's faster τ and therefore greater aerobic contribution to ATP re-synthesis at the onset of exercise indicates that they have an enhanced oxidative capacity. This is likely due to greater oxygen delivery and/or better oxygen utilization in the muscles. In conflict with data from adults, young people's peak $\dot{V}O_2$, which is primarily dependent on oxygen delivery, is not significantly related to the primary τ across all exercise domains. Furthermore, there is no convincing theoretical hypothesis to suggest that increased delivery of oxygen would speed the rate of p $\dot{V}O_2$ kinetics in healthy children or adolescents. However, an observational study using breath-by-breath technology, HR kinetics and NIRS reported that, compared with men, pre-pubertal boys demonstrated a faster primary τ at the onset of moderate intensity exercise which was supported by both a faster adjustment in [HHb] kinetics and faster local blood flow. Thus, suggesting that both oxygen delivery and oxygen utilization have a role to play in children's faster p $\dot{V}O_2$ phase II kinetics below T_{VENT} (10). In contrast, investigations of the relative contribution of oxygen delivery and oxygen utilization to boys' p $\dot{V}O_2$ kinetics have used priming exercise to raise $\dot{Q}$ and muscle oxygenation prior to the onset of subsequent very heavy intensity exercise and noted the primary τ to be unchanged. This suggests that in exercise above T_{VENT} the phase II τ is principally dependent on intrinsic muscle metabolic factors rather than oxygen delivery (2, 5).

A reasonable hypothesis is that the faster phase II τ in children is due to an age-dependent effect on mitochondrial oxidative phosphorylation which is in accord with children's enhanced aerobic enzymes activities and higher % of type I muscle fibres compared to adults. Similarly, the reported sexual dimorphism is consistent with girls having a lower % of type I fibres than similarly aged boys (4, 5). Rigorously determined and appropriately analysed data on young people's phase II τ response at the onset of exercise are accumulating but knowledge of independent effects of biological maturation on the primary τ is almost non-existent (2, 5).

Targeted research using non-invasive technologies is required to tease out the mechanisms of chronological age- biological maturation- and sex-related differences in $p\dot{V}O_2$ kinetics across exercise domains

Phase III

During exercise above T_{VENT} phase III is characterized by the oxygen cost increasing over time as a slow component of $p\dot{V}O_2$ (SC) is superimposed. In the heavy exercise domain pre-pubertal children present a SC which contributes ~10% of the end-exercise $p\dot{V}O_2$ after 10 min of exercise and increases in magnitude with chronological age. A longitudinal study identified significant sex differences in the relative magnitude of pre-pubertal children's SC and also noted that, in both sexes, despite an increase in the size of the SC the overall oxygen cost at the end of exercise did not change on test occasions 2 years apart. This implies that the phosphate turnover needed to sustain exercise is independent of chronological age and that younger children achieve a larger portion of their end-exercise $p\dot{V}O_2$ during phase II (5, 8).

There is a phase III SC in both children and adolescents during very heavy intensity exercise but, to date, sexual dimorphism has not been addressed in this exercise domain. Intriguingly, in the very heavy exercise intensity domain young people's SC has been observed to stabilize at ~85-90% of peak $\dot{V}O_2$ rather than project to peak $\dot{V}O_2$ as in adults. This may be due to an early termination of exercise through exhaustion or lack of motivation but it requires further investigation. In the severe intensity exercise domain a SC is not discernible from the primary component. Whether this is due to the prominence of the primary component of $p\dot{V}O_2$ or insufficient time for a SC to be expressed before attainment of peak $\dot{V}O_2$ is unclear. However, as there is a large SC in recovery from exercise in this domain it is likely that a SC also exists during the onset of exercise (2, 5). But,

xv)   *what are the mechanisms underpinning young people's SC in each exercise domain?*

xvi)   *why does young people's SC not project to peak $\dot{V}O_2$ in the very heavy exercise domain?*

xvii)   *why are there chronological age and sex differences in the magnitude of the SC? and,*

xviii) *does biological maturation influence the SC?*

Data from adults provide compelling evidence that > 80% of the SC originates from the exercising muscles (16). If this is the case in youth, the increase in magnitude of the SC with age might be due to progressive changes in muscle fibre recruitment. A rational hypothesis is that enhanced glycogen depletion of type I fibres and greater recruitment of type II fibres with chronological age promote an increased SC. Reported chronological age-related differences are in accord with children presenting higher oxidative enzymes activities and lower glycolytic enzymes activities, and a higher % of type I fibres than adults. There are no

data on whether, independent of chronological age, biological maturation influences the SC. Sex-related differences in the SC are consistent with boys having a higher % of type I fibres than similarly aged girls but, for ethical reasons, muscle biopsy data from healthy children and adolescents are sparse (2, 5). Evidently much remains to be learned about the development of the SC in different exercise domains.

The introduction of non-invasive experimental models such as priming exercise and manipulation of pedal rates supplemented by electromyography, MRI, MRS and NIRS provide a framework for intervention studies of the magnitude of the SC during childhood and adolescence.

Trainability of p$\dot{V}O_2$ kinetics

Training interventions that either speed the primary τ (reducing the oxygen deficit) or attenuate the SC (reducing the oxygen cost) can improve exercise tolerance. In adults the primary τ and the SC have been shown to respond positively and rapidly to both CIET and HIIT but the optimal training programme remains to be determined. Adults' primary τ is generally correlated with peak $\dot{V}O_2$ but the relationship can be dissociated with training suggesting that the two variables are reliant on different mechanisms. The balance of evidence from studies with adults indicates that the speeding of τ is restricted to exercise engaging the trained musculature and is associated with increased oxidative enzymes activities within those muscles. This might be supplemented by improved matching of oxygen delivery to oxygen utilization. Enhanced muscle oxygen delivery and homogeneity of its distribution, as well as improved muscle fibre oxidative capacity linked to changes in muscle fibre recruitment patterns have been postulated as potential mechanisms responsible for the training-induced reduction of the SC (16). But,

xix) *what do we know about p $\dot{V}O_2$ kinetics responses to training in youth?*

Data are sparse and current understanding of the trainability of p $\dot{V}O_2$ kinetics in youth is largely founded on cross-sectional studies of swimmers and footballers. Studies of the responses of swim trained and untrained pre-pubertal and pubertal girls at the onset of heavy intensity exercise revealed a faster τ in both trained groups than in their untrained peers during arm exercise and a faster τ during leg exercise in the trained pubertal group. No significant difference between untrained and trained girls in the magnitude of the SC was observed. As the trained pubertal girls exhibited faster [HHb] kinetics and HR kinetics than untrained pubertal girls the authors suggested that the faster τ could be attributed to both enhanced oxygen delivery and oxygen utilization (12, 14).

Two comparative studies of 15 year-old male and female footballers with untrained peers observed a faster primary τ at the onset of moderate intensity exercise in the trained footballers. It was hypothesized that faster HR kinetics and capillary blood flow kinetics but no difference in [HHb] kinetics between trained and untrained male footballers indicated that the faster primary τ was due to increased oxygen delivery and oxygen utilization. In contrast, faster [HHb] kinetics were observed in trained compared with untrained girls. Despite not having estimates of oxygen delivery, the authors speculated that the faster primary τ emanated from enhanced oxygen utilization in the muscles, implying sexual dimorphism in the response of p $\dot{V}O_2$ kinetics to training (1, 12).

Comparative studies are informative but cross-sectional designs preclude distinguishing between genetic selections and training effects on the observed differences. In the only published training intervention study to date, a 6 week high-intensity games intervention

programme significantly speeded the primary τ of pre-pubertal obese children but had no significant effect on the primary τ of normal weight pre-pubertal children. In general agreement with cross-sectional studies, peak $\dot{V}O_2$ in the obese group did not change and was therefore dissociated from the speeding of the primary τ. In accord with comparative data, the magnitude of the SC was unaffected by training in both groups (15).

Research questions

It is readily apparent that knowledge of p$\dot{V}O_2$ kinetics responses to training during childhood and adolescence is meagre. Numerous intriguing research questions arise and a non-exhaustive list includes:

xx)   *is CIET, HIIT, or a combination of CIET and HIIT the most effective programme to speed the primary τ?*

xxi)   *why are training-induced changes in the primary τ dissociated from changes in peak $\dot{V}O_2$?*

xxii)   *what mechanisms underpin training-induced speeding of the primary τ?*

xxiii)   *are p$\dot{V}O_2$ kinetics responses to training interventions specific to exercise domains?*

xxiv)   *are there chronological age-, biological maturation- and/or sex-related influences on p$\dot{V}O_2$ kinetics responses to training?*

xxv)   *does exercise training in youth reduce the magnitude of the SC?*

if so,

xxvi)   *is CIET, HIIT, or a combination of CIET and HIIT the most effective programme to reduce the magnitude of the SC?*

and,

xxvii) *what mechanisms underpin training-induced reductions in the magnitude of the SC?*

Well-controlled, rigorous intervention studies are needed to identify effective training programmes, elucidate the magnitude of training-induced changes in the primary τ and SC, and reveal the mechanisms underlying the changes during growth and maturation.

Conclusions

Peak $\dot{V}O_2$ has long been recognized as the best single indicator of aerobic fitness. As such it is well-documented but the causal mechanisms of pre-pubertal sexual dimorphism require clarification. The appropriate interpretation of peak $\dot{V}O_2$ in relation to body size during growth and maturation is shrouded in controversy and will remain so until journal editors insist on contextual reporting of the variable. Unique insights into aerobic fitness during youth rest in the transient response to a forcing exercise regimen. But, in comparison with peak $\dot{V}O_2$ the rigorous study of p$\dot{V}O_2$ kinetics at the onset of exercise is in its infancy and characteristics of the primary τ and SC during childhood and adolescence are still being explored.

The aerobic trainability of young people is no longer in dispute but the optimal training programme(s) to enhance improvements in peak $\dot{V}O_2$, the speed of the primary τ and the reduction of the magnitude of the SC remain to be established. The mechanistic bases for training-induced changes and the modulating effects of chronological age, biological maturation and sex are yet to be resolved.

Recent advances in experimental techniques initially pioneered with adults and new non-invasive technologies have been successfully developed and modified for use with children. New avenues of research into aerobic fitness and training in youth have been unlocked and now need to be pursued with vigour.

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