ABSTRACT
Peak oxygen uptake (VO₂ max) is internationally recognized as the criterion measure of youth aerobic fitness, but despite pediatric data being available for almost 80 years, its measurement and interpretation in relation to growth, maturation, and health remain controversial. The trainability of youth aerobic fitness continues to be hotly debated, and causal mechanisms of training-induced changes and their modulation by chronological age, biological maturation, and sex are still to be resolved. The daily physical activity of youth is characterized by intermittent bouts and rapid changes in intensity, but physical activity of the intensity and duration required to determine peak VO₂ is rarely (if ever) experienced by most youth. In this context, it may therefore be the transient kinetics of pulmonary VO₂ that best reflect youth aerobic fitness. There are remarkably few rigorous studies of youth pulmonary VO₂ kinetics at the onset of exercise in different intensity domains, and the influence of chronological age, biological maturation, and sex during step changes in exercise intensity are not confidently documented. Understanding the trainability of the parameters of youth pulmonary VO₂ kinetics is primarily based on a few comparative studies of athletes and nonathletes. The underlying mechanisms of changes due to training require further exploration. The aims of the present article are therefore to provide a brief overview of aerobic fitness during growth and maturation, increase awareness of current controversies in its assessment and interpretation, identify gaps in knowledge, raise 10 relevant research questions, and indicate potential areas for future research.

Aerobic fitness may be defined as the ability to deliver oxygen to the muscles and to utilize it to generate energy to support muscle activity during exercise. Maximal oxygen uptake (VO₂ max), 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 and has been the criterion measure of youth aerobic fitness for about 80 years. VO₂ max (or later peak VO₂) is the most comprehensively documented laboratory-determined variable in pediatric exercise physiology, but its assessment, interpretation, trainability, and relationship with other health-related variables during growth and maturation remain shrouded in controversy. VO₂ max limits the capacity to perform aerobic exercise, but it does not define all aspects of aerobic fitness. In everyday life, young people's spontaneous play and participation in sport are more concerned with short duration, intermittent exercise, and rapid changes in exercise intensity. Under these conditions, VO₂ max might be considered a variable of investigatory convenience rather than the principal physiological variable underpinning exercise behavior, and it is the kinetics of pulmonary VO₂ (pVO₂) that best describe aerobic fitness. In contrast with studies of VO₂ max, there are remarkably few investigations of young people's pVO₂ kinetic responses to step changes in exercise intensity. Studies of the trainability of youth pVO₂ kinetic responses are even rarer, and current comprehension is primarily based on comparative analyses of youth athletes and nonathletes.

The present discussion recognizes that there are several other indicators of aerobic fitness, including blood lactate and ventilatory thresholds, exercise economy, and recovery pVO₂ kinetics, but the focus herein is on VO₂ max (or peak VO₂) and pVO₂ kinetics at the onset of exercise, arguably the two most pertinent markers of youth aerobic fitness. The objectives of this article are to outline current understanding, reveal controversies in assessment and interpretation, identify gaps in knowledge, raise 10 relevant research questions (see Table 1), and indicate avenues for future research in youth aerobic fitness. The focus is primarily on the aerobic fitness of normal, healthy youth, and comparisons with athletes and nonathletes are not considered.
Table 1. Top 10 research questions related to youth aerobic fitness.

1. Is peak VO₂ a maximal index of youth aerobic fitness? Can it be confidently compared across studies?
2. Does peak VO₂ vary with chronological age? Is there sexual dimorphism in youth peak VO₂?
3. Should ratio scaling be used to interpret peak VO₂ in relation to body mass during growth and maturation?
4. Are today's youth aerobically fit? Has youth peak VO₂ deteriorated during the last 80 years?
5. Is youth peak VO₂ trainable? Does a maturation threshold exist?
6. Is youth peak VO₂ related to habitual physical activity?
7. Is Phase I of the youth pVO₂ kinetics response at the onset of exercise related to chronological age, biological maturity, or sex?
8. Is the youth pVO₂ kinetics response at the onset of exercise below the gas exchange threshold related to chronological age, biological maturity, or sex?
9. Is the youth pVO₂ kinetics response at the onset of exercise above the gas exchange threshold related to chronological age, biological maturity, or sex?
10. Is youth pVO₂ kinetics trainable?

children, and readers interested in other populations (e.g., athletic, clinical) are referred elsewhere for discussion (e.g., Armstrong & McManus, 2011; Armstrong & Van Mechelen, 2017; Bar-Or & Rowland, 2008).

Maximal (or peak) oxygen uptake

The seminal work of Hill and Lupton (1923) in England gave rise to the concept of VO₂ max in humans. They determined the rate of pVO₂ during running at a variety of speeds around a grass track carrying a Douglas bag and observed a near-linear relationship between running speed and pVO₂ with an eventual leveling-off (plateauing) of pVO₂ at exhaustion. These observations evolved into laboratory treadmill protocols to examine the pVO₂ response to incremental exercise founded on the concept of VO₂ max being attained when a plateau in pVO₂ occurred.

By the late 1930s, boys were participating in laboratory determinations of VO₂ max.

The first laboratory studies of 6- to 18-year-old boys’ VO₂ max were carried out at Harvard (Robinson, 1938) and at the University of Chicago (Morse, Schultz, & Cassels, 1949) using a treadmill protocol involving a 15-min walk at 3.5 miles·h⁻¹ up an 8.6% gradient, followed by a 10-min rest and a run to exhaustion at a speed of 6 miles·h⁻¹ or 7 miles·h⁻¹ up an 8.6% gradient. Åstrand’s (1952) doctoral dissertation, published in Scandinavia in 1952, was the first to include both boys and girls, aged 4 to 18 years. The three studies reported VO₂ max in ratio with body mass (mL·kg⁻¹·min⁻¹) but Åstrand insightfully expressed reservations about whether this approach was appropriate with children (see Research Question 3).

Åstrand (1952) criticized the treadmill protocol used by Robinson (1938) and Morse et al. (1949) as “certainly practical from the investigator’s point of view but hardly so from that of the subject, especially if he is 6–10 years old” (p. 96). He commented that on the basis of the exercise protocol and postexercise blood lactate accumulation, “the work in several cases must have been submaximal” (p. 110), a point conceded by Morse et al. (1949), who reported, “[U]ndoubtedly all of the boys did not push themselves to the same state of exhaustion, and some had not reached the limit of their capacity in 5 minutes of running at 7 mph . . . This limitation must be kept in mind when maximal values are discussed” (p. 699). Åstrand observed in his own studies that at exhaustion, a plateau in pVO₂ was attained in only “70 of 140 running experiments with schoolchildren” (p. 23). The methodological experiences of these pioneers lead nicely into the first research question and allow a foundation for subsequent research questions to emerge.

1. Is peak VO₂ a maximal index of youth aerobic fitness? Can it be confidently compared across studies?

Although the issue was seldom explicitly addressed in the extant literature for about 30 years, confirmation of Åstrand’s (1952) observation of about 50% of youth not exhibiting a pVO₂ plateau was evident in numerous subsequent studies (Armstrong & Welsman, 1994). Several authors argued that the failure of some children to attain a pVO₂ plateau at voluntary exhaustion was related to low motivation or low anaerobic capacity (Krahenbuhl, Skinner, & Kohrt, 1985). Armstrong, Welsman, and Winsley (1996) addressed the problem experimentally by determining the peak VO₂ of 40 9-year-olds (20 girls) on three occasions 1 week apart. The initial determination of peak VO₂, using an incremental treadmill exercise test to voluntary exhaustion, was followed by two “supramaximal” tests with participants running at the same speed at which the initial test was terminated but at gradients 2.5% and 5.0% greater than the highest gradient achieved on the first test. Only about 33% of the participants exhibited a pVO₂ plateau in the initial test, but no significant differences in either girls’ or boys’ mean peak VO₂ were observed across the three tests. The data implied that maximal values can be achieved in a single, incremental exercise
test to voluntary exhaustion. Nevertheless, as the term $\dot{V}O_2\text{max}$ conventionally assumes that a p$\dot{V}O_2$ plateau has been exhibited, it has become common practice in pediatric exercise science to refer to the highest p$\dot{V}O_2$ observed during a progressive exercise test to voluntary exhaustion as peak $\dot{V}O_2$, and this terminology will be adopted herein.

The question of whether reported peak $\dot{V}O_2$ can be considered a maximal index of an individual’s aerobic fitness has been frequently raised, but there is no easy way to confirm, in the single tests typical of most studies, whether an individual child or adolescent has delivered a maximal effort. A number of “secondary criteria” such as preset values of heart rate (HR; e.g., $\geq 85\%$–90% of predicted HR max), respiratory exchange ratio (R; e.g., $\geq 1.00$), and blood lactate accumulation (e.g., $\geq 6.0\text{ mL.L}^{-1}$) at the termination of exercise have been proposed to verify efforts as maximal, but they all have questionable theoretical justification, exhibit wide individual variations, and are exercise protocol- and ergometer dependent. For example, the peak HR of 8- to 16 year-olds in incremental treadmill and cycle ergometer tests has a mean ± standard deviation of $\sim 200 \pm 7$ and $\sim 195 \pm 7\text{ beats.min}^{-1}$, respectively, and as HR normally peaks prior to and not simultaneous with or after p$\dot{V}O_2$, a spot HR value of $\geq 85\%$ to 90% of predicted HR max does not confirm whether an individual’s effort was maximal. It has been demonstrated that terminating a test in accord with “secondary criteria” (HR, R, and blood lactate accumulation) underestimates a child’s $\dot{V}O_2\text{max}$ by about 10% to 22% on average, but dismissal of a test performance for not meeting “secondary criteria” can result in falsely rejecting a true maximal value (Barker, Williams, Jones, & Armstrong, 2011).

There is no international consensus on the appropriate methodology for the determination of peak $\dot{V}O_2$ as a true maximal index, which has clouded understanding of youth aerobic fitness and its interpretation in relation with, for example, health and well-being. To be confident of obtaining secure data in a single session, it appears necessary to follow an initial exercise test with a “supramaximal” test to confirm a maximal value of peak $\dot{V}O_2$. An appropriate example is a short-duration ramp test (8–10 min) to exhaustion followed about 15 min later by a “supramaximal” test such as that described by Barker et al. (2011). Typical error of the peak $\dot{V}O_2$ determined in a ramp test is, with children, about 4% across three ramp tests each a week apart (Welsman, Bywater, Farr, Welford, & Armstrong, 2005). Barker et al.’s (2011) proposed “supramaximal” test consists of a 2-min warm-up at 10 W followed by a step change to 105% of the peak power elicited at the end of the initial ramp test. With prepubertal children, the time to exhaustion is about 90 s (i.e., $> 4$ time constants [$\tau$]; see the section on p$\dot{V}O_2$ kinetics for clarification of $\tau$). On the few occasions (in our hands, ~5%) that the peak $\dot{V}O_2$ is higher than in the initial ramp test, the “supramaximal” test can be repeated at 110% of peak power following full recovery. This methodology is facilitated by the ability of youth to recover rapidly from exhaustive exercise (Ratel & Williams, 2017) and has been utilized across a broad range of aerobic fitness (e.g., Robben, Poole, & Harms, 2013).

In addition to an appropriate exercise protocol, the determination of peak $\dot{V}O_2$ requires knowledge of environmental conditions and accurate measurement of inspired and/or expired air per unit time and the fraction of oxygen and carbon dioxide therein. Respiratory gas analysis systems with appropriate calibration facilities are commonplace in research laboratories, but pediatric physiologists need to be cautious when measuring children’s respiratory responses to exercise with apparatuses primarily designed for use with adults (e.g., magnitude of dead space of a mouth-piece/facemask and breathing valve and volume of a mixing chamber in relation to size of the child). Breath-by-breath analysis systems are increasing in popularity, but they are challenged by the large interbreath variations of exercising children in relation to their p$\dot{V}O_2$ response amplitude (i.e., high noise-to-signal ratio; Potter, Childs, Houghton, & Armstrong, 1998). The breath-by-breath sampling interval can have a significant impact on the reported peak $\dot{V}O_2$. Short sampling intervals increase the variability in measuring p$\dot{V}O_2$, and with their smaller peak $\dot{V}O_2$, this is more significant in youth than in adults. In contrast, large sampling intervals may “oversmooth” the data and artificially reduce the true p$\dot{V}O_2$ response. A sampling interval in the range of about 15 s to 30 s appears optimal for youth, but whatever the chosen interval, it should be recorded and reported (see McManus & Armstrong, in press, for review).

It is readily apparent from the foregoing discussion that despite almost 80 years of reporting the peak $\dot{V}O_2$ of youth, there is still no consensus on appropriate experimental protocols, data acquisition, or confirmation of maximal values. There is an urgent need to critically review, design, validate, and harmonize methodology to enable meaningful cross-study comparisons of youth aerobic fitness and scrutiny of its implications.
for sport, health, and well-being. The Pediatric Exercise
Network Working Group based at University of
California–Irvine is addressing the problem (Ashish
et al., 2015; Pianosi et al., 2017), but international cooperation of researchers and journal editors is
required for progress.

2. Does peak VO₂ vary with chronological age? Is there sexual dimorphism in youth peak VO₂?

The peak VO₂ of youth is extensively documented but should be interpreted in relation to the previous discussion of methodology. The vast majority of data have been derived from cycle ergometry or treadmill running. Pearson product-moment correlations between data from the two modalities are ~0.90, but peak VO₂ determined on a treadmill is typically about 8% to 10% higher than that determined on a cycle ergometer (Boileau, Bonen, Heyward, & Massey, 1977). Data from the two ergometers should therefore not be merged for analysis, although data trends across studies are consistent. Similarly, the validity of peak VO₂ determinations of children younger than 8 years old has been questioned since the original studies of Robinson (1938), who commented, “[T]he youngest boys were unwilling to work after it ceased to be fun, whereas all of the boys 8 years and older could be encouraged to carry on for some time after the first signs of fatigue” (p. 281). The present discussion will therefore emphasize trends in peak VO₂ rather than cite absolute values (in L-min⁻¹), and the focus throughout this article will be on the more secure database of youth aged 8 years and older.

Data collated from studies including about 5,000 cycle ergometer and about 5,000 treadmill determinations and analyzed independently show boys’ peak VO₂ increases in a near-linear manner by about 150% from 8 to 16 years. Girls’ data display a similar trend with peak VO₂ increasing by about 80% during the same age range although there is a tendency for girls’ values to level-off at about 14 years of age (Armstrong & Welsman, 1994).

Longitudinal studies provide more granular analyses, but few longitudinal studies have reported data from a wide age range and coupled rigorous determination of peak VO₂ with substantial sample sizes. Armstrong and McManus (2017) collated data from longitudinal studies of treadmill-determined peak VO₂ that included 1,818 boys and 707 girls (Armstrong & van Mechelen, 1998; Armstrong & Welsman, 2001; Armstrong, Welsman, Nevill, & Kirby, 1999; Mirwald & Bailey, 1986; Sprynarova, Parizkova, & Bunc, 1987), and they cautiously summarized trends. Boys’ data are consistent and increase in a similar manner to that shown in cross-sectional studies. The pooled longitudinal data show an increase in boys’ peak VO₂ of about 150% from 8 years to 18 years with the greatest increases occurring from 13 years to 15 years. Girls’ data indicate an increase in peak VO₂ of about 88% from 8 years to 17 years with a progressive rise from 8 years to 13 years and a leveling-off from about 14 years. Mirwald and Bailey (1986) recorded evidence of a spurt in peak VO₂ aligned with peak height velocity in both sexes—an observation also noted by Geithner et al. (2004).

Cross-sectional data indicate that, on average, boys’ peak VO₂ values are about 10% higher than those of girls at 10 years of age, and the sex difference increases to about 25% at 12 years, 30% at 14 years, and about 35% by age 16 years. Longitudinal data show a similar trend, but there is some variation in the size of sex differences across studies particularly around 12 to 14 years of age, which is likely due to individual differences in the timing and tempo of biological maturation. Sexual dimorphism has been attributed to a combination of physiological factors including differences in body size, body composition, and blood hemoglobin concentration. Differences in habitual physical activity (HPA) have been proposed as a contributory factor, but there is no compelling evidence to suggest a meaningful relationship between peak VO₂ and HPA (Armstrong & McManus, 2017, and see Research Question 6).

In adolescence, the dominant influence on peak VO₂ is muscle mass. Boys’ greater muscle mass not only enhances total muscle VO₂ (mVO₂) during exercise, but through the peripheral muscle pump, it also augments the venous return to the heart, thereby boosting stroke volume (SV). Boys’ oxygen delivery to the muscles may be further supplemented by an increase in blood hemoglobin concentration during the late teens. However, in childhood, there are only small sex differences in muscle mass and blood hemoglobin concentration, but even with body size appropriately controlled (see Research Question 3), prepubertal boys have been shown to have significantly higher peak VO₂ than prepubertal girls (see Armstrong & McManus, 2017, for review of individual studies).

Explanations of prepubertal sexual dimorphism in peak VO₂ are contentious. The recent application of noninvasive technologies to developmental physiology has generated interesting but, to date, generally conflicting data. There is no compelling evidence to suggest sex differences in HR max, and two studies using Doppler echocardiography have attributed prepubertal sexual dimorphism in peak VO₂ to boys’ greater maximal cardiac and stroke indices, although they offered contradictory views on the relative contribution of cardiac size and...
function. Vinet et al. (2003) concluded that sexual dimorphism was due to differences in cardiac size, whereas Rowland, Goff, Martel, and Ferrone (2000) suggested that cardiac functional factors (skeletal muscle pump function, systemic vascular resistance, and adrenergic responses) rather than intrinsic left ventricular size are responsible for boys’ greater SV in childhood.

In contrast, a study using thoracic bioimpedance to estimate cardiac output (Q) at peak VO\(_2\) revealed prepubertal boys have larger arteriovenous oxygen differences (a-vO\(_2\) diffs) than girls with no dissimilarities in Q max or SV max (Winsley, Fulford, Roberts, Welsman, & Armstrong, 2009). The same study noted magnetic resonance imaging at rest revealed no significant sex differences in posterior wall thickness, septal wall thickness, left ventricular muscle mass, left ventricular muscle volume, left ventricular end-systolic chamber volume, or left ventricular end-diastolic chamber volume.

McNarry et al. (2015) used near-infrared spectroscopy to monitor changes in deoxygenated hemoglobin and myoglobin during a ramp exercise test to voluntary exhaustion and noted sex-specific differences in the balance between oxygen delivery and oxygen utilization, which might contribute to prepubertal sex differences and potentially to age-related changes in peak VO\(_2\).

It is evident that more longitudinal studies that explore the physiological factors influencing aerobic fitness through childhood and adolescence into young adulthood are required. Much remains to be learned about the mechanisms underpinning chronological age-related increases and sexual dimorphism in peak VO\(_2\), and recent technological advances in noninvasive methodology are opening up promising new avenues of research that need to be rigorously pursued.

3. Should ratio scaling be used to interpret peak VO\(_2\) in relation to body mass during growth and maturation?

Youth peak VO\(_2\) is highly correlated with body mass, and researchers have typically “controlled” for body mass differences by simply dividing peak VO\(_2\) (mL·min\(^{-1}\)) by total body mass (actually body weight) in kg and expressing it as a ratio (mL·kg\(^{-1}\)·min\(^{-1}\)). Ratio-scaled peak VO\(_2\) is, however, heavily influenced by largely metabolically inert fat mass, and when peak VO\(_2\) is analyzed in mL·kg\(^{-1}\)·min\(^{-1}\), a different picture emerges from that apparent when absolute values (in L·min\(^{-1}\)) are examined. Boys’ peak VO\(_2\) decreases slightly or remains unchanged from 8 years to 18 years while girls’ values progressively decline through adolescence (Armstrong & McManus, 2017).

Ratio scaling can be informative, for example, in monitoring the performance of youth athletes who transport their body mass although it is futile to compare the peak VO\(_2\) of youth athletes from different sports (e.g., artistic gymnasts vs. American footballers) in mL·kg\(^{-1}\)·min\(^{-1}\). Ratio scaling has, however, confused the physiological understanding of peak VO\(_2\) during growth and maturation and generated spurious relationships between aerobic fitness and other health-related variables.

Tanner (1949) described the fallacy of ratio scaling in 1949, and as noted earlier, Åstrand (1952) commented on its limitations in relation to expressing children’s peak VO\(_2\) in 1952, but despite regular critical reviews over decades (e.g., Katch & Katch, 1974; Loftin, Sothern, Abe, & Bonis, 2016; Welsman & Armstrong, 2000; Winter, 1992), researchers persist in reporting and academic journals continue to publish ratio-scaled peak VO\(_2\) as the primary (or often the only) descriptor of youth aerobic fitness regardless of context. Exploration of the theoretical bases of scaling exercise data in relation to body dimensions (e.g., body mass, lean body mass, leg muscle mass, stature, limb length) is beyond the scope of the present discussion, but interested readers are referred to Welsman and Armstrong (2008) where the theoretical principles of allometry and multilevel modeling are explained and applied to sets of pediatric data. The inadequacy of ratio scaling of body mass can, however, be easily demonstrated, and examples can be provided of how it has not only clouded understanding of youth aerobic fitness but also misrepresented its relationship with biological processes and other health-related variables.

To create a size-free variable in the present context requires a product–moment correlation coefficient between peak VO\(_2\) (in mL·kg\(^{-1}\)·min\(^{-1}\)) and body mass (in kg) that is not significantly different from zero. It has been frequently demonstrated, using large data sets, that ratio-scaled peak VO\(_2\) is negatively and significantly correlated with body mass, thus showing the inability of ratio scaling to remove the influence of body mass. On the other hand, allometric scaling of the same data sets has revealed correlations with body mass not significantly different from zero showing that a body mass-free variable had been produced (see Armstrong & McManus, 2017, for worked examples).

Ratio scaling “over scales” favors lighter individuals and penalizes heavier (more mature or overweight) youth. Both cross-sectional studies using allometric analyses (e.g.,...
Welsman, Armstrong, Kirby, Nevill, & Winter, 1996) and longitudinal studies using multilevel modeling (e.g., Armstrong et al., 1999) to control for body mass have challenged the conventional (ratio-scaled) interpretation of peak VO₂ during growth and unequivocally demonstrated that there is a progressive increase in peak VO₂ with chronological age in both sexes independent of body mass. Furthermore, ratio-scaled peak VO₂ has persistently been reported as unrelated to biological maturation (e.g., Fahey, Del Valle-Zuris, Oehlsen, Trieb, & Seymour, 1979), but it has concealed the true relationship between aerobic fitness and biological maturation. Several studies have shown that with chronological age and body mass appropriately controlled using allometry (e.g., Armstrong, Welsman, & Kirby, 1998a) or multilevel modeling (e.g., Armstrong & Welsman, 2001), there are, in both sexes, significant, additional, incremental effects of biological maturation on peak VO₂.

In an insightful review, Loftin et al. (2016) evaluated several investigations where ratio scaling of peak VO₂ with body mass has confounded understanding of the aerobic fitness of overweight/obese young people. The reviewers used their own data to demonstrate the unsuitability of using ratio scaling to control body mass with an obese population by calculating a significant negative correlation (r = −.47) between peak VO₂ in mL·kg⁻¹·min⁻¹ and body mass in kg (Loftin et al., 2001). They then presented empirical examples where overweight/obese youth had higher peak VO₂ (in L·min⁻¹) than normal-weight peers and typically presented lower peak VO₂ in ratio with body mass (mL·kg⁻¹·min⁻¹), but when peak VO₂ was allometrically scaled for body mass, differences were greatly reduced or absent. Loftin and colleagues (2016) concluded that expressing peak VO₂ in ratio with body mass may reflect overweight or obesity status in addition to aerobic fitness. They analyzed studies relating the peak VO₂ of obese youth to cardiovascular risk factors and resolved that putative relationships between aerobic fitness and cardiovascular risk factors in overweight youth may have been misrepresented as statistical relationships with ratio-scaled peak VO₂ and reflect obesity status to a greater extent than aerobic fitness.

Much of the chronological age-related and biological maturity-related increase in peak VO₂ reflects increases in muscle mass, and peak VO₂ is therefore strongly correlated with muscle mass. Some investigators have argued compellingly that as leg muscles are the prime movers in most activities, peak VO₂ should preferably be allometrically scaled to leg muscle mass or muscle volume rather than body mass (e.g., Tolfrey et al., 2006). Graves et al. (2013) empirically demonstrated that scaling of peak VO₂ to the lean muscle mass (LMM) of both legs or to lean body mass (LBM) is superior to scaling by body mass in the interpretation of children’s peak VO₂. However, as determination of LMM and LBM in youth is complex, time-consuming, and relatively expensive, body mass will probably continue as the primary scaling variable, particularly in large studies. As techniques for assessing LMM and LBM become more readily available, they will in some contexts likely complement body mass as allometric scaling variables in analyses of youth peak VO₂.

Convincing evidence of the fallacy of ratio-scaling peak VO₂ in childhood and adolescence is well documented but frequently ignored in the extant literature. More research-led evaluation of potential scaling variables is required to inform discussion. Evidence-based implementation of appropriate scaling methodology in the context of the problem being addressed is essential to progress physiological understanding of youth peak VO₂, clarify its relationship with health-related variables during growth and maturation, and explore current levels of youth aerobic fitness.

**4. Are today’s youth aerobically fit? Has youth peak VO₂ deteriorated during the last 80 years?**

Credible international norms for youth aerobic fitness (peak VO₂) are not available (Pianosi et al., 2017; Rowland, 2007). Comprehensive studies of the peak VO₂ of healthy volunteers have shown typical coefficients of variation of about 15%, and youth athletes frequently present values about 40% to 50% higher than healthy, untrained peers, but there is no agreement as to what constitutes a low, acceptable, or high peak VO₂ in healthy youth.

The laboratory determination of peak VO₂ is expensive, time-consuming, dependent on sophisticated apparatuses, and reliant on skilled technical assistance. This has encouraged some researchers to predict youth peak VO₂ from field tests of performance. Numerous field tests are available with the 20-m shuttle run test (20mSRT), currently the most popular international test of youth maximal performance (Tomkinson & Olds, 2008). International age- and sex-specific norms for 20mSRT performance, based on more than 1 million tests in 50 countries, have been recently published (Tomkinson et al., 2016), but the number of completed shuttle runs is not a physiological measure of aerobic fitness. Shuttle run performance is an indicator of maximal voluntary performance influenced by a network of
psychosocial, physical, and biomechanical factors as well as physiological variables (Tomkinson & Olds, 2007a).

A positive relationship between youth aerobic fitness and health has been well documented (e.g., Shearer & Cumming, 2017), and for at least 30 years, youth aerobic fitness “health thresholds” have been periodically advocated based on, for example, expert opinion (Bell, Macek, Rutenfranz, & Saris, 1986), extrapolations from adult data (The Cooper Institute, 2004), or statistical links with cardiometabolic risk factors (Adegboye et al., 2011). There are, however, no extant data to verify the existence of a peak VO\(_2\) value that represents a specific threshold associated with youth health and well-being. The proposed “health thresholds” are generally comparable and supported by similar arguments, but none are based on direct determinations of youth peak VO\(_2\). They are all compromised by ignoring the highly significant effects of the timing and tempo of biological maturation and, in particular, by expressing peak VO\(_2\) recommendations in ratio with body mass. It is not usually possible with large published studies, which have directly determined peak VO\(_2\), to accurately calculate the percentage of participants who meet recommended “health threshold” values. But, a reanalysis of treadmill-determined peak VO\(_2\) data from two UK investigations of untrained, volunteer participants revealed that the “health thresholds” proposed by Bell et al. (1986) were met by more than 97% of 220 (107 girls) 12- to 15-year-olds (Armstrong, Williams, Balding, Gentle, & Kirby, 1991) and 100% of 164 (53 girls) 11-year-old prepubertal children (Armstrong, Kirby, McManus & Welsman, 1995).

Despite frequent claims to the contrary, there is no compelling empirical evidence to show that the current generation of youth have low levels of peak VO\(_2\) (Mountjoy et al., 2011). Research into and dissemination of the current state of youth aerobic fitness and its putative relationship with health and well-being need to be reappraised in the light of a clear definition of aerobic fitness and elucidation of its assessment and interpretation during growth and maturation.

Low levels of youth peak VO\(_2\) remain to be proven, but has aerobic fitness declined over time? Studies of youth aerobic fitness must be interpreted in the context of the elucidated limitations of methodology and dissemination outlined in Research Questions 1 through 3, but taken collectively, VO\(_2\) compilations of data over time indicate a general consistency in youth peak VO\(_2\) values over several decades, particularly in boys (Armstrong, Tomkinson, & Ekelund, 2011; J. C. Eisenmann & Malina, 2002; Freedson & Goodman, 1993). Comparisons of data from different laboratories, using various equipment and exercise protocols, over large time frames are interesting, but they provide only partial insights into temporal trends of youth peak VO\(_2\). Literature reviews are not a valid substitute for epidemiological studies but collections of discrete investigations providing local snapshots of the peak VO\(_2\) of volunteer participants who do not necessarily reflect the populations from which they are drawn. Nevertheless, there is little empirical evidence to support the view that youth peak VO\(_2\) has deteriorated over time. In the Exeter Children’s Health and Exercise Research Centre, about 3,000 8- to 18-year-olds from the same catchment area and schools have had their peak VO\(_2\) determined with no discernible change in values noted during a 30-year period (N. Armstrong, unpublished data).

In contrast with peak VO\(_2\), youth 20mSRT performance has declined during the last 40 years (Armstrong et al., 2011; Tomkinson, Leger, Olds, & Carzola, 2003; Tomkinson & Olds, 2007b) and has frequently been misrepresented in both the popular and scientific literature as a deterioration in youth aerobic fitness. There is convincing evidence of an increase in youth body fatness in recent decades, and temporal increases in body fatness explain about 40% to 60% of the deterioration in 20mSRT performance (Olds & Dolman, 2004; Olds, Ridley, & Tomkinson, 2007). Changes in other nonphysiological factors contribute further to a decline in 20mSRT performance (Tomkinson & Olds, 2007a). As most activity involves transporting body mass, a secular decrease in 20mSRT performance is a cause for concern and the underlying causes are worthy of further investigation, but the use of 20mSRT performance as a proxy for peak VO\(_2\) is untenable.

There are at least 17 different published equations to predict youth peak VO\(_2\) from 20mSRT performance, equations that result in markedly different estimates of peak VO\(_2\) (Tomkinson et al., 2016). A recent meta-analysis of youth 20mSRT performance in relation to peak VO\(_2\) revealed that the criterion validity of the 20mSRT for estimating peak VO\(_2\) is considerably less with children and adolescents than with adults; furthermore, it showed that more than 50% of correlation coefficients explained less than 50% of the variance in peak VO\(_2\) (Mayorga-Vega, Aguiler-Soto, & Viciana, 2015). In addition, shuttle run performance can be improved by practice and various training methods without an associated increase in peak VO\(_2\) (Harrison, Gill, Kinugasa, & Kilding, 2015). It is therefore evident that the use of 20mSRT scores as surrogates for or predictors of peak VO\(_2\) not only seriously misrepresents...
youth aerobic fitness but also has the potential to confound putative relationships with health-related variables.

To enhance knowledge, researchers need to unambiguously explain, differentiate, and justify the assessment and interpretation of measures of youth aerobic fitness. This is crucial if the data are to be used in subsequent statistical analyses with other health-related morphological, cardiometabolic, or behavioral variables or to be fed into recommendations or policy statements relating to youth health and well-being. More research into the relationship between clearly defined, directly determined, and appropriately interpreted youth aerobic fitness and current and future health and well-being is urgently required.

5. Is youth peak VO\textsubscript{2} trainable? Does a maturation threshold exist?

Early studies investigating the influence of training on youth aerobic fitness frequently concluded that there were minimal or no effects on peak VO\textsubscript{2}, but these observations need to be interpreted with caution due to logistical, experimental design, methodological, and analytical limitations, which have been detailed elsewhere (Armstrong & Barker, 2011; McNarry & Armstrong, 2017; Pfeiffer, Lobelo, Ward, & Pate, 2008). It is, however, worthy of note at the onset of discussion that the heritability of youth trainability has been estimated at about 50% (Wackerhage, Smith, & Wisneiwski, 2017). Although it is likely that some individuals are genetically high responders to training whereas others are almost nonresponders, with a range of response phenotypes between these two extremes, it is rarely considered in training studies with youth. Baseline peak VO\textsubscript{2} is negatively correlated with the percentage change in peak VO\textsubscript{2} following training (Mahon, 2008), but the view that high youth HPA might pretrain study participants has been largely discounted (Rowland, 2005, and see Research Question 6).

The most recently published systematic literature review revealed 69 published training studies and 21 investigations that had rigorously examined the effect of exercise training on youth peak VO\textsubscript{2} (see Armstrong & Barker, 2011, for a tabulated analysis). Almost all studies consisted of constant-intensity exercise training (CIET) programs with large variations in the frequency, duration, and, particularly, relative intensity of sessions. The majority of studies that elicited a significant training-induced increase in peak VO\textsubscript{2} utilized a training stimulus of about 85% to 90% of HR max, whereas most studies in which no significant changes had been observed employed lower relative exercise intensities (~70%–80% of HR max). The review concluded that the intensity of exercise was crucial and suggested that the popular assertion that youth have a “blunted” response to training compared with adults (Mahon, 2008) might be explained by adults being able to enhance their peak VO\textsubscript{2} with a lower relative training intensity than youth. In what appear to be the only studies in which boys (Savage et al., 1986) and girls (P. A. Eisenmann & Golding, 1975) were subjected to the same relative intensity training programs as adults, the increases in peak VO\textsubscript{2} were not significantly different between boys and men and between girls and women, respectively. In agreement with an earlier review (Pfeiffer et al., 2008), no credible evidence to support sexual dimorphism in peak VO\textsubscript{2} responses to CIET in youth was identified.

The concept of children being unable to benefit from training prior to a “maturation threshold” or “trigger point” has been embedded in the pediatric exercise science literature for about 35 years. It originated with the work of Gilliam and Freedson (1980), who introduced an enhanced physical education program into the lifestyles of 8-year-olds during a period of 12 weeks. When no significant changes in peak VO\textsubscript{2} (or other physiological variables) were observed, they proposed the existence of “a maturational threshold whereby pre-pubescent children are unable to elicit physiological changes in response to exercise training” (p. 76). In 1983, Katch developed further the concept and hypothesized, “[T]here is one critical time period in a child’s life (termed the ‘trigger point’) which coincides with puberty in most children, but may occur earlier in some, below which the effects of physical conditioning will be minimal, or will not occur at all” (p. 241). It was suggested that the hypothesized phenomenon occurs through the modulating effects of hormones that initiate puberty and influence functional development and consequent organic adaptations. Periodically, others have rolled out persuasive theoretical arguments for a maturational effect on the peak VO\textsubscript{2} response to training (see Rowland, 1997), but there is no compelling empirical evidence to support the case. Although acknowledging that most studies are based on chronological age rather than maturity status, comprehensive reviews of the literature have consistently concluded that there is no convincing empirical evidence to support a maturation threshold (Armstrong & Barker, 2011; McNarry & Jones, 2014; Pfeiffer et al., 2008). The definitive investigation is yet to be published, but a series of recent observational studies by
McNarry and her colleagues (summarized in McNarry & Armstrong, 2017) has challenged the maturation threshold hypothesis. In their latest contribution, McNarry, Mackintosh, and Stoedefalke (2014) monitored the peak VO\textsubscript{2} of boys and girls in swim training and untrained peers from age 10 to 12 years with three annual measurement points. The trained swimmers presented a higher peak VO\textsubscript{2} than their untrained peers at each measurement, and the difference between the two groups increased on each occasion. The data were analyzed using multilevel modeling, and the researchers noted that the increase in peak VO\textsubscript{2} was not associated with changes in maturity status and did not therefore support the maturation threshold hypothesis.

Present data indicate that the trainability of youth peak VO\textsubscript{2} is independent of chronological age, biological maturity, and sex and that the maturation threshold hypothesis remains to be proven. However, as Rowland (1997) argued, there are some tantalizing clues that hormonal responses at puberty might modulate aerobic trainability. To elucidate the possible influence of biological maturation on the trainability of peak VO\textsubscript{2} and to rigorously test the maturation threshold hypothesis, longitudinal data from well-controlled, interventional training studies, including both sexes, with measurement occasions covering a time period that includes prepuberty, puberty, and postpuberty are necessary.

High-intensity interval training (HIIT) has been shown to be an effective and time-efficient approach to enhancing peak VO\textsubscript{2} in adults (Gist, Fedewa, Dishman, & Cureton, 2014). As HIIT better reflects youth HPA patterns and youth recover more rapidly from high-intensity exercise than adults (Ratel & Williams, 2017), it is surprising that there are so few HIIT protocols in pediatric training studies. It was demonstrated more than 20 years ago that prepubertal girls could improve their peak VO\textsubscript{2} through both CIET and HIIT (McManus, Armstrong, & Williams, 1997), but it is only recently that a concerted research effort has focused on HIIT as a means of increasing youth peak VO\textsubscript{2} (e.g., Tolfrey & Smallwood, 2017). In their systematic review and meta-analysis, Costigan, Eather, Plotnikoff, Taaffe, and Lubans (2015) concluded that HIIT is an effective means of enhancing peak VO\textsubscript{2} in adolescence, but as only eight published investigations met their eligibility criteria, the paucity of extant HIIT studies is readily apparent.

Studies of the mechanisms underpinning training-induced changes in youth peak VO\textsubscript{2} have been limited by ethical and methodological issues related to the determination of VO\textsubscript{2} max and hence calculation of SV max and maximal a-vO\textsubscript{2}diff. Sparse research has indicated that the maximal a-vO\textsubscript{2}diffs of youth athletes and nonathletes are similar, and training-induced changes in peak VO\textsubscript{2} have therefore been associated with increases in oxygen delivery. As HR max does not increase with training, enhanced peak VO\textsubscript{2} has been generally attributed to an enlarged SV max. Both morphological and functional adaptations of the myocardium have been hypothesized as explanations for an augmented SV max following CIET. Increases in blood volume, left ventricular dimensions and mass, intraventricular and posterior wall thickness, shortening fraction, and ejection fraction have been postulated, but empirical evidence is sparse and conflicting (see Armstrong & Barker, 2011, and McNarry & Armstrong, 2017, for analyses of individual studies).

No published studies have specifically investigated the mechanisms of HIIT-induced changes in youth peak VO\textsubscript{2}, and extrapolation of adult data to youth must be done with extreme caution. However, training interventions with adults have indicated that peripheral (oxygen utilization) rather than central (oxygen delivery) adaptations might be primarily responsible for HIIT-induced increases in peak VO\textsubscript{2} (Gibala & McGhee, 2008). The intriguing question therefore arises that if CIET primarily induces central adaptations and HIIT stimulates peripheral changes, would a combination of the two protocols optimize training-induced increases in youth peak VO\textsubscript{2}?

Appropriate 8- to 12-week CIET programs have been shown to induce, on average, increases of about 8% to 9% in youth peak VO\textsubscript{2} (Armstrong & Barker, 2011; Pfeiffer et al., 2008), but the quantitative potential of HIIT programs to enhance youth peak VO\textsubscript{2} is still to be realized. Greater increases in peak VO\textsubscript{2} are likely with more sustained periods of CIET, HIIT, and/or combined CIET and HIIT, but no well-controlled studies have been published. Much remains to be learned about optimizing training programs in relation to growth and maturation and elucidating the mechanisms underpinning training-induced changes in youth peak VO\textsubscript{2}.

6. Is youth peak VO\textsubscript{2} related to habitual physical activity?

In youth health-related studies, HPA, defined as "usual physical activity carried out in normal daily life in every domain and any dimension" (Hildebrand & Ekelund, 2017, p. 303), and aerobic fitness (peak VO\textsubscript{2}) are often erroneously used interchangeably. HPA is a behavior
and peak $\dot{V}O_2$ is a physiological variable. They are not synonymous, but do they share a meaningful relationship?

It is recognized that peak $\dot{V}O_2$ has a genetic component, with the heritability of peak $\dot{V}O_2$ estimated to be about 50%. Data from studies of the heritability of voluntary activity behavior are more variable with heritability estimates in childhood being low to moderate and in adolescence being moderate to high. Further comment on genetics is beyond the scope of the present article, but interested readers are referred to a recent review of the topic (Schutte, Bartels, & de Gues, 2017). Herein the focus is on investigations that have directly determined youth peak $\dot{V}O_2$ in a laboratory and objectively estimated HPA in the same study.

The first team to estimate HPA objectively and compare the data with directly determined peak $\dot{V}O_2$ was Seliger, Trefny, Bartenkova, and Pauer (1974), who estimated the HPA of 11 12-year-old boys from 1 day of HR monitoring and found no significant relationship with peak $\dot{V}O_2$. Subsequent investigations from several mainly European countries confirmed these findings and consistently reported either no relationship or a very weak relationship between peak $\dot{V}O_2$ and HPA.

We have tabulated in chronological order summaries of all located studies of objectively estimated HPA and directly determined peak $\dot{V}O_2$ elsewhere (Armstrong & Fawkner, 2007), but as it could be argued that pre-1990 studies did not monitor physical activity (PA) long enough for it to be representative of HPA, Table 2 only describes studies that monitored PA for at least 3 days. Based on the extant evidence, periodic reviews of the literature have consistently concluded that, at best, there is only a very weak relationship between peak $\dot{V}O_2$ and HPA (Armstrong, 1998, 2012; Morrow & Freedson, 1994).

Longitudinal investigations are sparse but strongly reinforce cross-sectional data. A study of 202 (98 girls) children, aged 11 years at onset, used multilevel modeling to examine the influence of chronological age, biological maturity, and body mass on HPA on three annual occasions. With the primary variables, controlled peak $\dot{V}O_2$ was introduced to the model as an additional variable, and a nonsignificant parameter estimate demonstrated no significant relationship between peak $\dot{V}O_2$ and HPA. A subsequent analysis of accumulated time spent in at least moderate-intensity PA (equivalent to brisk walking) for three 10-hr periods of HR monitoring in relation to peak

### Table 2. Habitual physical activity and peak oxygen uptake in youth.

<table>
<thead>
<tr>
<th>Citations</th>
<th>Participants</th>
<th>Means of physical activity estimation</th>
<th>Mode of exercise in peak $\dot{V}O_2$ determination</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Armstrong, Balding, Gentle, Williams, &amp; Kirby (1990)</td>
<td>111 girls, 85 boys; aged 11–16 years</td>
<td>3-day HR monitoring Cycle ergometer or treadmill</td>
<td>No significant relationships.</td>
<td>Nonsignificant correlation coefficients ranged from $r = .01$ to $r = .26$.</td>
</tr>
<tr>
<td>Armstrong, McManus, Welsman, &amp; Kirby (1996)</td>
<td>63 girls, 60 boys; aged 12.2 years</td>
<td>3-day HR monitoring</td>
<td>No significant relationships.</td>
<td>Nonsignificant correlation coefficients ranged from $r = .13$ to $r = .16$ in boys and from $r = -.02$ to $r = .04$ in girls.</td>
</tr>
<tr>
<td>Armstrong, Welsman, &amp; Kirby (1998b)</td>
<td>43 girls, 86 boys; aged 10–11 years</td>
<td>3-day HR monitoring Treadmill</td>
<td>No significant relationships.</td>
<td>Nonsignificant correlation coefficients ranged from $r = -.15$ to $r = .09$.</td>
</tr>
<tr>
<td>Armstrong et al. (2000)</td>
<td>98/70/79 girls, 104/73/81 boys; longitudinal study from 11–13 years</td>
<td>3-day HR monitoring Treadmill</td>
<td>In a multilevel regression model, peak $\dot{V}O_2$ was a nonsignificant parameter estimate of PA.</td>
<td></td>
</tr>
<tr>
<td>Ekelund et al. (2001)</td>
<td>40 girls, 42 boys; aged 14–15 years</td>
<td>3-day HR monitoring Treadmill</td>
<td>No significant relationship between MVPA and peak $\dot{V}O_2$ ($r = -.04$); AEE explained 14% of the variance in peak $\dot{V}O_2$.</td>
<td></td>
</tr>
<tr>
<td>Eiberg et al. (2005)</td>
<td>309 boys, 283 girls; aged 6–7 years</td>
<td>3-day accelerometer Treadmill</td>
<td>Sustained periods of PA explained 9% of the variance in peak $\dot{V}O_2$.</td>
<td></td>
</tr>
<tr>
<td>Dencker et al. (2006)</td>
<td>101 girls, 127 boys; aged 8–11 years</td>
<td>3- to 4-day accelerometer Cycle ergometer</td>
<td>In a multiple forward regression analysis, VPA and MDPA together explained 10% of the variance in peak $\dot{V}O_2$ (VPA 9%, and MDPA 1%).</td>
<td></td>
</tr>
<tr>
<td>Butte, Puyau, Adolph, Vohra, &amp; Zakeri (2007)</td>
<td>424 nonoverweight and 473 overweight 4- to 19-year-olds</td>
<td>3-day accelerometer Treadmill</td>
<td>PA explained 1% to 3% of the variance in peak $\dot{V}O_2$.</td>
<td></td>
</tr>
<tr>
<td>Dencker, Bugge, Hermansen, &amp; Andersen (2010)</td>
<td>222 girls, 246 boys; aged 6–7 years</td>
<td>4-day accelerometer Treadmill</td>
<td>PA explained 0% to 8% and 0% to 2% of the variance in peak $\dot{V}O_2$ in boys and girls, respectively.</td>
<td></td>
</tr>
</tbody>
</table>

Note. PA = physical activity; TDEE = total daily energy expenditure; MPA = moderate physical activity; MVPA = moderate-to-vigorous physical activity; VPA = vigorous physical activity; MDPA = mean daily physical activity; AEE = activity-related energy expenditure; HR = heart rate. Table adapted and updated from Armstrong & Fawkner (2007).
VO₂ clearly showed that not only was there no relationship between HPA and peak VO₂, but PA of at least moderate intensity significantly decreased with age, whereas peak VO₂ in both L-min⁻¹ and appropriately normalized for body mass significantly increased from 11 to 13 years in both girls and boys (Armstrong, Welsman, & Kirby, 2000; Armstrong et al., 1999).

The Amsterdam Growth and Health Longitudinal Study is probably the most comprehensive longitudinal study of youth health and fitness to date, and following analysis of HPA and peak VO₂ data, Kemper and Koppes (2004) concluded that “if we take into account that the relationship calculated with autoregression over 23 years resulted in non-significant relationships, we must admit that in this observational study no clear association could be proved between PA and VO₂ max in free-living males and females” (p. 163).

The extant data clearly show that HPA is not (or at best, is only very weakly) related to peak VO₂ in youth. This finding is not unexpected as HPA typically lacks the duration and intensity associated with the enhancement of peak VO₂ in youth (Ekelund, Tomkinson, & Armstrong, 2011). Further development and refinement of methods with which to objectively estimate HPA are required, and as there appears to be no meaningful relationship with peak VO₂, researchers should explore other measures of aerobic fitness to clarify the association (if any) between HPA and aerobic fitness in youth. pVO₂ kinetics better reflect the pattern of youth HPA and may provide a fruitful avenue for research.

Pulmonary oxygen uptake kinetics

The introduction of breath-by-breath respiratory gas exchange technology enabled a group of innovative physiologists to chart the kinetic responses of pVO₂ at the onset of exercise (Whipp, Ward, Lamarra, Davis, & Wasserman, 1982), to show that pVO₂ kinetics have the potential to provide a window into the metabolic activity of the muscle (Grassi et al., 1996), and through a combination of breath-by-breath technology and magnetic resonance spectroscopy, to deliver a noninvasive, relatively inexpensive means of investigating youth muscle metabolism during exercise (Rossiter et al., 1999).

Macek and Vavra (1980) were the first to investigate the half-time of children’s transient responses at the onset of exercise, but the initial application of breath-by-breath technology to youth pVO₂ kinetics was done by Cooper and his colleagues (e.g., Cooper, Berry, Lamarra, & Wassermann, 1985). Data from early studies are inconsistent, but recent research using more rigorous methodology, sophisticated mathematical modeling techniques, and evolving technologies has begun to elucidate youth pVO₂ kinetics.

The application of pVO₂ kinetics and muscle phosphocreatine kinetics to youth muscle metabolism and the related research opportunities have been discussed elsewhere (Armstrong, Barker, & McManus, 2017; Barker & Armstrong, 2010). Periodic reviews of the literature on youth pVO₂ kinetics have been published as it evolved (Armstrong & Barker, 2009; Barstow & Scheuermann, 2005; Fawkner & Armstrong, 2003), and the potential mechanisms explaining pVO₂ kinetics in youth have been recently explored (Barker & Armstrong, 2017). But, herein, the discussion is focused on research questions emerging from pVO₂ kinetics in the context of youth aerobic fitness, and with space restrictions, only essential background will be outlined.

Determination of the pVO₂ kinetic response is achieved by imposing a predetermined square wave exercise stress and then using nonlinear regression and iterative fitting procedures with the response data to fit a specified model to return the rate of the exponential rise in pVO₂ and the amplitude of the response. However, the use of a range of models, several with limited physiological rationales, has confused understanding of youth pVO₂ kinetics. Furthermore, the inherently erratic breathing pattern in youth reduces the signal-to-noise ratio of the pulmonary gas exchange kinetics. Large interbreath fluctuations reduce the confidence with which pVO₂ kinetic responses can be estimated, and confidence intervals are likely to be beyond acceptable limits unless sufficient identical transitions are time-aligned and averaged to improve the signal-to-noise ratio. Few pediatric studies have reported acceptable confidence intervals for the primary component time constant (τ). Interested readers are referred to Fawkner and Armstrong (2007) for a critique of the methodology that has been employed in the determination of youth pVO₂ kinetics.

The pVO₂ response to a step change from rest (or low-intensity exercise) to higher-intensity exercise is characterized by three phases. Phase I (the cardiodynamic phase), which lasts 15 s to 20 s in youth, is associated with an increase in VO₂, which occurs prior to the arrival at the lungs of venous blood from the exercising muscles and is therefore independent of mVO₂. Phase I is followed by an exponential rise in pVO₂ (Phase II) that, with moderate-intensity exercise (i.e., exercise intensity below the gas exchange threshold [GET]), drives pVO₂ to a steady state (Phase III) within 90 s. Phase II is described by its time constant (τ), which is the time taken to achieve 63% of the change in pVO₂...
(98% of the total change in $p\dot{V}O_2$ therefore occurs in $4\tau$). A step change to heavy-intensity exercise (i.e., exercise intensity higher than the GET but below critical power [CP]) also shows an exponential Phase II $p\dot{V}O_2$ response following Phase I but stimulates a Phase III where the oxygen cost increases with time as a slow component (SC) of $p\dot{V}O_2$ is superimposed and the achievement of a steady state is delayed in youth by about 10 min. Gaesser and Poole (1994) have argued compellingly that based on adult data, about 85% of the $p\dot{V}O_2$ SC originates from the exercising muscles, perhaps largely due to a change in muscle fiber recruitment as exercise progresses.

Phase III of a step change to very heavy-intensity exercise (i.e., exercise intensities lying between CP and peak $\dot{V}O_2$) is not characterized by an eventual steady state, and at least in adults, the SC rises with time and projects to peak $\dot{V}O_2$. Step changes to higher exercise intensities result in exhaustion with a single exponential $p\dot{V}O_2$ response and the absence of a discernible SC and are classified as being in the severe exercise domain. Under these circumstances, peak $\dot{V}O_2$ is achieved in youth within about 90 s.

### 7. Is Phase I of the youth $p\dot{V}O_2$ kinetics response at the onset of exercise related to chronological age, biological maturity, or sex?

Following the onset of exercise, $p\dot{V}O_2$ is dissociated temporarily from $m\dot{V}O_2$ by the muscle–lung transit delay. The speed of the response is reliant on the almost instantaneous increase in $Q$, which is initiated by vagal withdrawal. If the muscle–lung transit delay is a function of growth, the shorter distance between exercising muscles and the lung in children might suggest an age-related increase in the length of Phase I. Data on Phase I are, however, ambiguous and frequently confounded by methodological challenges. For the purposes of modeling the primary component, the duration of Phase I is not usually measured but is assumed to be constant at about 15 s to 20 s and removed from the data set to model Phases II and III. However, as Phase I is linked to $Q$ kinetics (i.e., pulmonary blood flow) at the onset of exercise and when compared with adults, children require a greater increase in $Q$ to achieve a given $p\dot{V}O_2$ (Turley & Wilmore, 1997), it seems reasonable to suggest that the characteristics of Phase I might be altered during growth and maturation.

Men have been reported to have a longer-duration Phase I than boys in response to the onset of a transition to 50% of peak $\dot{V}O_2$ (Hebestreit, Kriemler, Hughson, & Bar-Or, 1998). A longitudinal study of 10- to 13-year-olds revealed that during three annual measurement occasions, the duration of Phase I at the onset of heavy-intensity exercise increased in both boys and girls (Fawkner & Armstrong, 2004a). The same study provided evidence of sexual dimorphism with prepubertal boys presenting a shorter-duration Phase I than that of prepubertal girls. This finding might be indicative of faster SV kinetics at the onset of exercise in boys than in girls—an observation previously noted using Doppler echocardiography (Rowland et al., 2000).

Very little is known about Phase I at the onset of exercise and whether it varies with the exercise intensity of the step change. Chronological age- and sex-related influences on Phase I and the underlying mechanisms require more rigorous exploration. Independent effects of biological maturity on Phase I do not appear to have been studied. Further investigation of the duration of Phase I across exercise domains is warranted.

### 8. Is the youth $p\dot{V}O_2$ kinetics response at the onset of exercise below the gas exchange threshold related to chronological age, biological maturity, or sex?

Initial investigations of $p\dot{V}O_2$ kinetics at the onset of moderate-intensity exercise indicated that the Phase II $\tau$ was fully mature in childhood, but several early studies were methodologically flawed (see Armstrong & Barker, 2009, for a critique), and more recent, better controlled studies have demonstrated that the primary component $\tau$ is significantly shorter in boys than in men and in girls than in women (Breese, Barker, Armstrong, Jones, & Williams, 2012; Fawkner, Armstrong, Potter, & Welsman, 2002; Leclair et al., 2013). In the only study to include girls, Fawkner et al. (2002) reported an absence of sexual dimorphism in $\tau$ despite significant sex differences in peak $\dot{V}O_2$. In contrast to earlier data from adults (see Poole & Jones, 2012, for review), they reported that peak $\dot{V}O_2$ is not related to the Phase II $\tau$ in youth, a finding later confirmed by others (Cleuziou et al., 2002).

A shorter Phase II $\tau$ and therefore higher aerobic contribution to adenosine triphosphate (ATP) resynthesis at the onset of exercise indicate that compared with adults, youth have an enhanced oxidative capacity, which may be due to greater oxygen delivery or to better oxygen utilization in the muscles. Peak $\dot{V}O_2$, which is postulated to be primarily dependent on oxygen delivery, is not related to the Phase II $\tau$, and there is no compelling evidence to indicate that increased delivery of oxygen would speed the $p\dot{V}O_2$ kinetics of
healthy youth at the onset of moderate-intensity exercise (Barker & Armstrong, 2017; Poole & Jones, 2012). However, a study that determined p\(\text{VO}_2\) kinetics, HR kinetics, deoxygenated myoglobin, and hemoglobin (HHb) kinetics and estimated capillary blood flow noted that compared with men, prepubertal boys presented a shorter Phase II \(\tau\) supported by both a more rapid adjustment in HHb kinetics and faster local blood flow. This finding infers that oxygen extraction and oxygen delivery might both have a role to play in children’s faster p\(\text{VO}_2\) kinetics at the onset of exercise below the GET (Leclair et al., 2013).

Evidence to tease out the mechanisms underpinning age-related differences in the Phase II \(\tau\) is limited. Robust data from girls are currently confined to one study, and the influence (if any) of the process of biological maturation has not been addressed. The dissociation of peak \(\text{VO}_2\) from the Phase II \(\tau\) in youth but not in adults is intriguing. More targeted research is needed to elucidate the p\(\text{VO}_2\) kinetic response to the onset of moderate-intensity exercise in youth.

9. Is the youth p\(\text{VO}_2\) kinetics response at the onset of exercise above the gas exchange threshold related to chronological age, biological maturity, or sex?

Resolution of youth p\(\text{VO}_2\) kinetics above the GET is proving to be challenging. As the range of potential exercise intensities is lower in youth than in adults, the scope of the metabolic transitions to exercise possible within each exercise domain is reduced and several studies do not adhere to strict definitions of the heavy and very heavy exercise domains, thus confounding the resolution of p\(\text{VO}_2\) responses. True p\(\text{VO}_2\) kinetic responses have also been masked in several studies through using suboptimal numbers of repeated transitions, not reporting confidence intervals, and analyzing data with a confusing array of mathematical models often with limited physiological rationales (see Armstrong & Barker, 2009, for a critique).

Data are sparse, but two robust longitudinal studies have established that at the onset of heavy-intensity exercise, the Phase II \(\tau\) is negatively related to chronological age in children and adolescents (Breese et al., 2010; Fawkner & Armstrong, 2004a). The first study demonstrated a significantly shorter Phase II \(\tau\) in 10-year-old prepubertal boys and prepubertal girls compared with the same children presented 2 years later. In the later study, which only included boys, researchers observed 14-year-olds presented a shorter Phase II \(\tau\) than they did at 16 years. The same research group found that in contrast to their findings at the onset of exercise below the GET, prepubertal boys presented a shorter Phase II \(\tau\) than prepubertal girls (Fawkner & Armstrong, 2004b). At all measurement occasions in the three studies (boys 7 measurement occasions, girls 4 measurement occasions), the Phase II \(\tau\) was not significantly related to peak \(\text{VO}_2\).

Early studies concluded that during heavy-intensity exercise, children demonstrate a negligible p\(\text{VO}_2\) SC and their responses could be modeled as a monoexponential process, but subsequent investigations have demonstrated empirically that a Phase III p\(\text{VO}_2\) SC does exist in children. Few data are available, but they are consistent in indicating that in youth, the p\(\text{VO}_2\) SC contributes about 9% to 12% of the end-exercise p\(\text{VO}_2\), increases in magnitude with chronological age, and is greater in girls than in boys (Breese et al., 2010; Fawkner & Armstrong, 2004a, 2004b). The influence of biological maturation on p\(\text{VO}_2\) kinetic responses at the onset of exercise above the GET has not been investigated.

In the very heavy exercise-intensity domain, boys have a shorter Phase II \(\tau\) and a smaller relative p\(\text{VO}_2\) SC than men and peak \(\text{VO}_2\) is not related to the Phase II \(\tau\) (e.g., Breese et al., 2012). No data from girls appear to have been published, and sexual dimorphism has therefore not been addressed in this exercise domain. In contrast to adult data, the Phase III p\(\text{VO}_2\) SC in youth has not been shown to project to peak \(\text{VO}_2\) but to stabilize at about 85% to 90% of peak \(\text{VO}_2\) (Barker, Bond, Toman, Williams, & Armstrong, 2012), which may be due simply to an early termination of exercise by children and adolescents through exhaustion, but confirmatory data are required.

Exploratory investigations have used priming exercise to elevate \(\dot{Q}\) and muscle oxygenation prior to exercise above the GET resulting in an unaltered Phase II \(\tau\) and a reduced p\(\text{VO}_2\) SC amplitude. This suggests that Phase II p\(\text{VO}_2\) kinetics above the GET are principally limited by intrinsic muscle metabolic factors and the p\(\text{VO}_2\) SC is sensitive to oxygen delivery (e.g., Barker, Jones, & Armstrong, 2010), but confirmatory research is required before conclusions can be drawn.

Breath-by-breath data in the severe-intensity exercise domain are limited to one study of 9- to 12-year-old boys and men in which, once Phase I had been excluded, p\(\text{VO}_2\) kinetics could be described by a monoexponential function (i.e., no p\(\text{VO}_2\) SC detected) in which chronological age-related differences in Phase II \(\tau\) were not present (Hebestreit et al., 1998).
Studies of the p\(\text{VO}_2\) kinetic response of youth at the onset of exercise above the GET are sparse and require confirmation before the pattern of change with chronological age can be confidently established. Data from girls and investigations including an estimate of stage of biological maturity are almost nonexistent. Empirical evidence to inform the theoretical bases of the mechanisms underlying youth p\(\text{VO}_2\) kinetics is limited. The introduction of experimental models such as priming exercise, manipulation of pedal rates, and control of respiratory gases (e.g., hypoxic/hyperoxic stimuli) is promising. The simultaneous analysis of p\(\text{VO}_2\) kinetics, HR kinetics, \(\dot{Q}\), and blood deoxygenation kinetics, supplemented with electromyography, is providing further insights into youth p\(\text{VO}_2\) kinetics (Barker & Armstrong, 2017). But the development of more noninvasive and innovative research models is essential to tease out and evidence the mechanisms of chronological age-, biological maturity-, and sex-related influences on p\(\text{VO}_2\) kinetics across exercise domains.

10. Is youth p\(\text{VO}_2\) kinetics trainable?

Training interventions that either shorten the Phase II \(\tau\) (reducing the oxygen deficit) or attenuate the SC (reducing the oxygen cost) can improve exercise tolerance. Adults’ Phase II \(\tau\) and SC have been shown to respond quickly and positively to both CIET and HIIT programs, although optimal training programs are not yet established (Poole & Jones, 2012). Data on youth p\(\text{VO}_2\) kinetics trainability are, however, sparse and largely reliant on four comparative studies of trained soccer players and swimmers compared to untrained youth from two research groups (McNarry & Armstrong, 2017).

Unnithan and his colleagues (Marwood, Roche, Rowland, Garrard, & Unnithan, 2010; Unnithan, Roche, Garrard, Holloway, & Marwood, 2015) compared responses at the onset of moderate-intensity exercise of 15-year-old male and female soccer players to those of untrained peers and reported the soccer players presented a shorter Phase II \(\tau\). As faster HR kinetics and capillary blood flow kinetics but similar HHb kinetics were observed in the trained boys, the authors postulated that the soccer players’ faster p\(\text{VO}_2\) kinetics could be attributed to both increased oxygen delivery and oxygen utilization. Intriguingly, the trained girls presented faster HHb kinetics, and despite not having estimates of oxygen delivery, Unnithan et al. (2015) speculated that their shorter Phase II \(\tau\) originated from enhanced oxygen utilization in the muscles, thus suggesting that unlike peak \(\dot{\text{VO}}_2\), there is sexual dimorphism in the response of p\(\text{VO}_2\) kinetics to training.

McNarry and her colleagues (McNarry, Welsman, & Jones, 2010; Winlove, Jones, & Welsman, 2010) contrasted the responses of swim-trained and untrained prepubertal and pubertal girls at the onset of heavy-intensity exercise and revealed a shorter Phase II \(\tau\) in both trained groups than their untrained peers during an arm-cranking exercise and a shorter Phase II \(\tau\) during a leg-cycling exercise in the trained pubertal girls. No differences between trained and untrained girls were observed in the magnitude of the p\(\text{VO}_2\) SC. On the basis of observing faster HR kinetics and HHb kinetics in the trained pubertal girls, McNarry et al. (2010) hypothesized that their shorter Phase II \(\tau\) was a function of both enhanced oxygen delivery to the muscles and oxygen utilization in the muscles.

In the only published intervention study, a 6-week games program was reported to significantly shorten the Phase II \(\tau\) of 9-year-old, prepubertal obese boys but had no significant effect on the Phase II \(\tau\) of normal-weight prepubertal boys. The peak VO\(_2\) and the magnitude of the SC were unaffected by training (McNarry, Lambrick, Westrupp, & Faulkner, 2015).

Knowledge of the trainability of youth p\(\text{VO}_2\) kinetics is meager and predominantly reliant on unconfirmed cross-sectional data. Well-designed intervention studies are needed to develop and evaluate training programs using a range of methodologies, including variations of CIET, HIIT, and combined CIET and HIIT. The influence of chronological age and biological maturity on the magnitude of changes in the Phase II \(\tau\) and the SC is unknown. Sexual dimorphism is untested. Recently developed noninvasive techniques and technologies need to be harnessed and utilized to explore the mechanisms underlying trainability in the parameters of p\(\text{VO}_2\) kinetics during growth and maturation.

Conclusions

Peak \(\dot{\text{VO}}_2\) is internationally recognized as the best single measure of youth aerobic fitness, but a debate over harmonizing definitions, terminology, methodology, and interpretation during growth and maturation persists. Lack of clarity has confused discussion of current youth aerobic fitness, comparisons with previous generations, and relationships with health-related behavior. Unique insights into youth aerobic fitness lie in the p\(\text{VO}_2\) kinetics response to a forcing exercise regimen, but in comparison with peak \(\dot{\text{VO}}_2\), the rigorous study of p\(\text{VO}_2\) kinetics is in its infancy and much more research into responses across exercise domains is required. The challenge is to not only map
out peak VO$_2$ and pVO$_2$ kinetics responses, but to identify and explain the underlying mechanisms and how they evolve in childhood and adolescence. The trainability of youth is no longer in dispute, and the presence of a maturation threshold remains to be proven. Optimal training programs for the enhancement of peak VO$_2$ and the parameters of pVO$_2$ kinetics need further development and evaluation. The mechanistic bases for training-induced improvements in aerobic fitness and the modulating effects of chronological age, biological maturity, and sex have yet to be resolved. The evolution of sophisticated modeling techniques and the introduction of noninvasive techniques and technology into pediatric exercise physiology provide promising avenues for further research into the elucidation of youth aerobic fitness.

**References**


Armstrong, N. (2012). Young people are fit and active—Fact or fiction? *Journal of Sport and Health Sciences, 1*, 131–140.


