Influence of training status and exercise modality on pulmonary O_2 uptake kinetics in pre-pubertal girls

Melitta A. Winlove[1], Andrew M. Jones[1] and Joanne R. Welsman[1]

[1] School of Sport and Health Sciences, St. Luke's Campus, University of Exeter, Heavitree Road, Exeter EX1 2LU, United Kingdom

Correspondence to:
Melitta A. Winlove
School of Sport and Health Sciences
St. Luke's Campus
University of Exeter
Heavitree Road
Exeter
EX1 2LU
United Kingdom
M.A.Winlove@exeter.ac.uk

Abstract

The limited available evidence suggests that endurance training does not influence the pulmonary oxygen uptake ($\dot{V}O_2$) kinetics of pre-pubertal children. We hypothesised that, in young trained swimmers, training status related adaptations in the $\dot{V}O_2$ and heart rate (HR) kinetics would be more evident during upper body (arm-cranking) than during leg cycling exercise. Eight swim-trained (T; 11.4 ± 0.7 years) and eight untrained (UT; 11.5 ± 0.6 years) girls completed repeated bouts of constant-work-rate cycling and upper body exercise at 40% of the difference between the gas exchange threshold and peak $\dot{V}O_2$. The phase II $\dot{V}O_2$ time constant was significantly shorter in the trained girls during upper body exercise (T: 25 ± 3 vs. UT: 37 ± 6 s; $P < 0.01$) but no training status effect was evident in the cycle response (T: 25 ± 5 vs. UT: 25 ± 7 s). The $\dot{V}O_2$ slow component amplitude was not affected by training status or exercise modality. The time constant of the HR response was significantly faster in trained girls during both cycle (T: 31 ± 11 vs. UT: 47 ± 9 s; $P < 0.01$) and upper body (T: 33 ± 8 vs. UT: 43 ± 4 s; $P < 0.01$) exercise. This study demonstrates for the first time that swim-training status influences upper body $\dot{V}O_2$ kinetics in pre-pubertal children, but that cycle ergometry responses are insensitive to such differences.

Key Words

Oxygen uptake kinetics; training;upper body and leg exercise; children.

Introduction

Pulmonary oxygen uptake ($\dot{V}O_2$) kinetics provide a useful, non-invasive assessment of the integrated capacity of the organism to transport and utilize O_2 to support the increased rate of energy turnover in the contracting myocytes (Whipp and Ward 1990). In adults, the influence of factors such as exercise intensity and exercise modality on $\dot{V}O_2$ kinetics have been well documented (for a detailed review see: Jones and Poole 2005). Exercise training is known to be a potent stimulus to $\dot{V}O_2$ kinetics in this population, resulting in reductions in both the phase II time constant (τ) and the amplitude of the subsequent $\dot{V}O_2$ 'slow component' (Bailey et al. 2009; Berger et al. 2006; Carter et al. 2000; Casaburi et al. 1987; Jones and Koppo 2005; Koppo et al. 2004; Phillips et al. 1995; Powers et al. 1985).

The $\dot{V}O_2$ kinetic response in children has been less comprehensively described and the influence of endurance training on $\dot{V}O_2$ kinetics in this population is unclear (Fawkner and Armstrong 2003). The available data suggest that training neither reduces τ (Cleuziou et al. 2002; Obert et al. 2000) nor reduces the amplitude of the $\dot{V}O_2$ slow component (Obert et al. 2000) in children. This apparent insensitivity to training clearly contrasts with findings in adults (e.g. Carter et al. 2000; Cleuziou et al. 2003; Koppo et al. 2004; Norris and Petersen 1998; Phillips et al. 1995). However, it is possible that this is a consequence of methodological limitations in paediatric studies such as the use of only single exercise transition to characterize $\dot{V}O_2$ kinetics, the prescription of exercise intensity as a fraction of the peak $\dot{V}O_2$, the use of mixed sex cohorts, or the employment of non-specific ergometry. The relatively small $\dot{V}O_2$ response amplitudes and relatively large inter-breath variability in $\dot{V}O_2$ in children requires that several repeat transitions are performed for $\dot{V}O_2$ kinetics to be confidently characterized (Fawkner and Armstrong 2007). Moreover, the relatively high inter-individual variability in the fraction of peak $\dot{V}O_2$ at which the gas exchange threshold (GET) occurs in children compared to adults (Fawkner and Armstrong 2007) means that it is important to consider both the GET and the peak $\dot{V}O_2$ when attempting to standardise the exercise intensity domain in which participants are exercising. There are suggestions that boys and girls exhibit subtle differences in $\dot{V}O_2$

kinetics, with boys suggested to exhibit faster $\dot{V}O_2$ kinetics and a smaller slow component amplitude (Fawkner and Armstrong 2004c). Therefore the analysis of both sexes together might be inappropriate. Another explanation for the reported insensitivity of children to training might be that some previous studies utilized non-specific testing modalities. Previous studies have all involved investigation of the effects of swimming training on $\dot{V}O_2$ kinetics during cycle ergometer exercise (Cleuziou et al. 2002; Obert et al. 2000). Considering the large contribution of the upper body to swimming (Ogita et al. 1996), a test modality such as upper body ergometry, which at least demonstrates a commonality of muscles exercised, might be more likely to demonstrate training influences on the physiological responses to exercise.

The purpose of this cross-sectional study was to examine the influence of training status and exercise modality on the $\dot{V}O_2$ and HR kinetics of pre- and early-pubertal girls during heavy-intensity exercise. We hypothesized that the $\dot{V}O_2$ and HR kinetics would be faster and that the $\dot{V}O_2$ slow component amplitude would be smaller in trained swimmers compared to an age-matched untrained control group. We also hypothesized, given the large contribution of the upper body to swimming, that training status-related differences would be more evident during upper body than during cycle ergometer exercise.

Methods

Participants

Eight endurance trained (T) girls and 8 untrained (UT) girls aged 10-12 years participated in this study. The T group, all competitive swimmers with a mean training volume of 8 (± 2.5) hours/week, were recruited from a local swimming club. The UT group comprised volunteers from local schools. Sexual maturity was assessed by self-report using the indices of pubic hair described by Tanner (1962). Age to peak height velocity was estimated to provide an additional indicator of physical maturity according to the equations of Mirwald *et al*. (2002) which are based on the

measurement of standing and seated height, weight, and date of birth as described below.

An anthropometrical evaluation was performed before the first test for all participants. Standing and seated height were measured to 0.1 cm using a Holtain stadiometer (Holtain, Crymych, Dyfed, UK) and body mass determined using Avery beam balance scales to 0.05 kg (Avery, Birmingham, UK). Skinfold thickness was assessed three times at four sites around the body (bicep, triceps, subscapular and supra-iliac crest) by the same researcher for all participants using Harpenden callipers (Baty International, Burgess Hill, UK), accurate to the nearest 0.2mm. The average of the three measurements was taken.

Participants were asked to arrive at the laboratory in a rested and fully hydrated state, at least 3 hours postprandial and to refrain from consuming caffeinated drinks in the 6 hours prior to the test. The methods employed during this study were approved by the institutional research ethics committee and all participants and their parents/guardians gave written informed consent and assent, respectively.

Measurements of peak $\dot{V}O_2$ and GET

On the first two visits to the laboratory, exercise mode-specific peak $\dot{V}O_2$ and gas exchange threshold (GET) were determined using an incremental ramp test to voluntary exhaustion on both cycle (Lode Excalibur, Netherlands) and upper body (Lode Angio, Netherlands) ergometers. Gas exchange variables (Metalyser 3B Cortex, Biophysik, Leipzig, Germany) and heart rate (Polar S610, Polar Electro Oy, Kempele, Finland) were measured on a breath-by-breath basis and displayed online**.** Prior to each test the gas analyser was calibrated using gases of known concentration and the turbine volume transducer was calibrated using a 3-litre syringe (Hans Rudolph, Kansas City, MO). The delay in the capillary gas transit and analyser rise time were accounted for relative to the volume signal, thereby time aligning the concentration and volume signals. The handle bar height, seat height and crank length (cycle ergometer) and electrically controlled seat height and distance (upper body

ergometer) were adapted to suit each child and the values recorded so they could be replicated throughout the testing series.

After a three minute warm-up consisting of unloaded pedalling or arm cranking, the resistance increased by 12 $W \cdot min^{-1}$ and 5 $W \cdot min^{-1}$ for cycle and upper body exercise, respectively, to attain a test of ~8-12 minutes in duration. Participants were instructed to maintain a cadence between 70 and 50 rpm on the cycle and upper body ergometer, respectively. Maximal tests were considered to have been achieved if in addition to subjective indications such as sweating, hyperpnoea and facial flushing, the respiratory exchange ratio (RER) was > 1.0 and there was a consistent reduction in cadence despite strong verbal encouragement. The data were interpolated to 1-s intervals and peak $\dot{V}O_2$ was taken as the highest 10-s stationary average during the test. The GET was determined by the V-slope method (Beaver et al. 1986) as the point at which carbon dioxide production began to increase disproportionately to $\dot{V}O_2$ as identified using purpose designed software developed using LabVIEW (National Instruments, Newbury, UK).

Constant work rate tests

The participants returned to the laboratory on a number of other occasions to complete step-change exercise tests on the upper body and cycle ergometers. Where multiple tests were performed on the same day, at least 1 hour separated the tests and the tests were ordered such that the first test involved a smaller muscle mass (upper body), thereby resulting in a smaller metabolic perturbation and faster recovery. On average, 4 cycle and 7 upper body transitions were completed, dependent on the number of transitions required to obtain 95% confidence intervals of < 4 s for cycling or < 4.5 s for upper body exercise. All constant-work-rate tests consisted of 4 minutes of unloaded pedalling or cranking followed instantaneously by a transition to a work rate extrapolated to require 40% of the difference between the GET and peak $\dot{V}O_2$ (40%Δ) for 8 minutes. On completion, the work rate instantaneously returned to no load and the participants pedalled or cranked for a further 6 minutes. Throughout the cycle ergometer and upper body tests, cadences of 70 ± 5 rpm and 50 ± 5 rpm were maintained respectively. Fingertip blood samples were taken 1 minute after

completion of the loaded phase and assayed for blood lactate concentration (YSI 1500, Yellow Springs Instruments, Yellow Springs, OH).

$\dot{V}O_2$ Kinetics Analysis

Initially, the breath-by-breath responses to each transition were examined to remove any errant breaths caused by coughing, swallowing, sighing etc using a 5 s moving average to identify points lying in excess of 4 SD from the local mean. Subsequently, each transition was interpolated to 1 s intervals, time aligned to the start of exercise and averaged.

To remove the influence of phase I on analysis of the subsequent response, the first 15 s of data were ignored. A single exponential model with a time delay (Eq.1) was then applied to the averaged response and parameters and their 95% confidence intervals determined by least squares linear regression analysis (Graphpad Prism, Graphpad Software, San Diego, CA).

$$\Delta \dot{V}O_{2(t)} = A_1 \cdot \left(1 - e^{-(t-\delta_1)/\tau_1}\right) \qquad \text{(Eq.1)}$$

where $\Delta\dot{V}O_2$ is the increase in $\dot{V}O_2$ at time *t* above the baseline value (calculated as the mean $\dot{V}O_2$ from the first 45 seconds of the last minute of baseline pedalling), and A_1, δ_1 and τ_1 are the primary component amplitude, time delay and time constant, respectively.

The fitting window was constrained to exclude all data after the visually determined onset of the $\dot{V}O_2$ slow component. This approach therefore avoids any possible influence of arbitrarily parameterizing the slow component. The onset of the $\dot{V}O_2$ slow component was determined using purpose designed LabVIEW software which iteratively fits a mono-exponential function to the $\dot{V}O_2$ data until the window encompasses the entire response. The resulting phase II time constants are plotted against time and the onset of the $\dot{V}O_2$ slow component identified as the point at which the phase II time constant consistently deviates from the previously “flat” profile

(Fawkner and Armstrong 2004b). The amplitude of the $\dot{V}O_2$ slow component was subsequently determined by calculating the difference between the end exercise $\dot{V}O_2$ and the primary amplitude plus baseline $\dot{V}O_2$. This was expressed both in absolute terms and relative to end exercise $\dot{V}O_2$.

HR Kinetics Analysis

As with the $\dot{V}O_2$ responses, the HR responses to each transition were interpolated to 1 second intervals, time aligned and averaged to produce a single data set. The resulting data set was fit with a single exponential with no time delay (Eq.2) with the fitting window starting at $t = 0$ and constrained to the onset of the $\dot{V}O_2$ slow component.

$$\Delta HR_{(t)} = A_1 \cdot \left(1 - e^{-(t/\tau_1)}\right) \quad \text{(Eq.2)}$$

where ΔHR is the increase in heart rate at time *t* above the baseline (calculated as the mean heart rate from the first 45 seconds of the last minute of baseline pedalling), and A_1 and τ_1 are the primary component amplitude and time constant, respectively.

Statistics

The allometric relationship between body mass and peak $\dot{V}O_2$ was determined using analysis of covariance (ANCOVA) on log transformed data (Welsman and Armstrong 2000). From the values of the regression slopes (allometric exponents) confirmed as common to all groups, power function ratios (Y/X^b) were computed. A two way ANOVA with repeated measures was used to analyse training status and exercise mode effects. Subsequent independent or paired samples t-tests with a Bonferonni correction were employed as appropriate to identify the location of significant effects. All data are presented as means ± SD. Statistical significance was accepted when $P < 0.05$.

Results

The physical characteristics and peak responses of the participants are presented in Tables 1 and 2. All children reported a maturity level of 1 or 2, indicting the study population was pre- or early-pubertal. Both groups had equal numbers of maturity stage 1 and 2 participants. Responses to the peak tests on each mode did not differ between groups before or after allometric scalling ($P > 0.05$). Peak values achieved on the cycle ergometer were significantly higher than the respective values achieved on the upper body ergometer within each group ($P < 0.005$), with the exception of peak RER, peak blood [lactate] and the fraction of peak $\dot{V}O_2$ at which the GET occurred which did not differ between exercise modes ($P > 0.05$).

Table 3 presents the $\dot{V}O_2$ responses to the constant-work-rate tests on the cycle ergometer and upper body ergometer. Between-group differences were evident in baseline $\dot{V}O_2$, with UT demonstrating a higher baseline value regardless of the exercise modality ($P < 0.01$). The UT had a significantly longer $\dot{V}O_2$ phase II τ (i.e. slower kinetics) during upper body exercise compared to T ($P < 0.001$) and also compared to their respective cycle ergometer response ($P < 0.01$). There was no correlation ($P > 0.05$) between the upper body and cycle ergometer phase II τ in T (r = -0.36) UT (r = -0.33) or when all the girls were analysed together (r = -0.21). The $\dot{V}O_2$ response of a representative participant from both groups is illustrated in Figure 1 for both cycle and upper body exercise. No training status-related differences were evident in the other $\dot{V}O_2$ related parameters during constant-work-rate exercise as shown in Table 3. No significant correlation was evident between peak $\dot{V}O_2$ and the phase II τ in T or UT girls or when the all the girls were analysed together for either cycle exercise (r = -0.47, -0.37 and -0.36) or upper body exercise (r = -0.18, -0.43 and -0.46).

The higher work rates employed during cycle exercise resulted in higher absolute $\dot{V}O_2$ values both at the end of the primary phase and at the end of exercise ($P < 0.001$). However, the gain of the primary component of $\dot{V}O_2$ was higher during upper body exercise ($P < 0.01$). T had a higher end-exercise blood [lactate] during cycle compared to upper body exercise ($P < 0.01$). A $\dot{V}O_2$ slow component was evident in all the girls' responses with the exception of one trained girl who failed to show a significant deviation from the steady state during upper body exercise. There were no

differences in the absolute or relative magnitude of the $\dot{V}O_2$ slow component with training status or exercise modality.

The parameters derived from the modelling of the HR responses to constant-work-rate exercise are presented in Table 4. UT had higher baseline HR regardless of the exercise modality ($P < 0.01$). On both modalities, the HR response was faster in T than UT ($P < 0.01$) but there was no influence of exercise modality in either group. The HR response of a representative participant from both groups is illustrated in Figure 2 for both cycle and upper body exercise. No correlation was found for either exercise mode between the $\dot{V}O_2$ phase II τ and the HR τ in either trained (cycle: r = 0.53; upper body: r = 0.22) or untrained (cycle: r = 0.44; upper body: r = 0.14) girls or when all the girls were analysed together (cycle: r = 0.35; upper body: r = 0.19).

Discussion

This is the first study to investigate the influence of training status and exercise modality on the $\dot{V}O_2$ and HR kinetic responses during step transitions to a heavy intensity work rate in children. We hypothesised that adaptations to swim training would be evident in the $\dot{V}O_2$ and HR kinetics measured during constant-work-rate exercise tests and that the effects would be more evident during upper body than during cycle ergometry. The results are partly consistent with our hypotheses as training status effects were evident in the $\dot{V}O_2$ kinetic response to upper body ergometry and in the HR kinetic response to both exercise modalities. Specifically, in response to the transition to a heavy-intensity work rate, trained girls had faster $\dot{V}O_2$ (upper body only) and HR kinetics (both modalities) compared to untrained girls. There was no effect of training status on the physiological responses to incremental exercise for either exercise modality.

Peak physiological responses

The peak physiological responses observed in the present study during both cycle and upper body ergometry demonstrate the insensitivity of peak values to training in children. Whilst the absence of an influence of training status on peak $\dot{V}O_2$ is not a

novel finding during cycle ergometry (Shephard 1992; Welsman et al. 1996), the physiological responses to upper body ergometry have not previously been investigated. This study therefore demonstrates that even when a more relevant, if not entirely sport-specific, exercise modality is used, incremental tests to the limit of tolerance are inappropriate for the investigation of training status differences, at least in pre- and early-pubertal girls. A limitation of this study is that no supra-maximal tests were conducted to verify if the peak $\dot{V}O_2$ values obtained from the incremental ramp tests were true peak values. The HR, RER and blood [lactate] criteria that are commonly applied for the acceptance of a $\dot{V}O_2$ 'max' both in adult and paediatric populations has recently been challenged (Barker et al. 2009; Poole et al. 2008b). Consequently, the peak $\dot{V}O_2$ values reported here cannot be definitively stated to reflect a maximal effort and are presented for consideration within the limits of reliability reported for peak $\dot{V}O_2$ determined using this traditional methodology (Welsman et al. 2005).

The similar peak $\dot{V}O_2$ values reported here between the T and UT girls may largely reflect genetic factors and/or that the appropriate hormonal milieu required for training adaptations in peak $\dot{V}O_2$ to be manifest is not sufficiently developed before puberty (Katch 1983). It is widely accepted that stroke volume (and hence cardiac output) is a primary determinant of peak $\dot{V}O_2$ (Nottin et al. 2002; Obert et al. 2003). It is interesting to note, therefore, that several studies have been unable to demonstrate changes in myocardial morphology following training in children (Nottin et al. 2004; Obert et al. 2009; Rowland et al. 2009). Since changes in cardiac structure including an increased thicknesses of the intraventricular and posterior walls thickness are commonly reported in adults (e.g. Caso et al. 2000; George et al. 1991), this might indicate that structural adaptations to cardiac muscle with training is somehow impeded in children. This age-related disparity may be related to a biological immaturity such as a lack of testosterone (Obert et al. 2003; Rowland et al. 1994) or to an overload limitation, either due to the lower blood pressures present in children during exercise (Nottin et al. 2002) or the shorter duration of training undertaken by children (Nottin et al. 2004). Although training has been reported to increase testosterone and growth hormone levels from puberty onwards, no difference in the

levels of these hormones has been shown pre-puberty (Daly et al. 1998; Tsolakis et al. 2003; Zakas et al. 1994).

Consistent with previous training studies in pre-pubertal children, we found no influence of training status on the fraction of peak $\dot{V}O_2$ at which the GET occurred (Cleuziou et al. 2002; Obert et al. 2000). These findings contrast with those commonly reported in adults where training increases the fraction of peak $\dot{V}O_2$ at which the GET occurs (Boone et al. 2008; Carter et al. 2000). The reason/s for this discrepancy is unclear from the present results. Exercise modality did not influence the percentage of peak $\dot{V}O_2$ at which the GET occurred, a finding which agrees with some adult studies (Koga et al. 1996; Schneider et al. 2002) but contrasts with others (Bhambhani et al. 1998; Davis et al. 1976).

Sub-maximal physiological responses: cycle exercise

In agreement with previous studies, swim training status did not alter the primary component $\dot{V}O_2$ kinetics nor affect the $\dot{V}O_2$ slow component amplitude in absolute or relative terms during cycle ergometer exercise (Cleuziou et al. 2002; Obert et al. 2000). The novelty of this study therefore lies not in the cycle ergometry results but in the confidence associated with those results, as this is the first study in children to employ a stringent and controlled methodology, the result of which was confidence intervals well within those suggested by Fawkner and colleagues (2007) for both the phase II τ and primary phase amplitude.

Our results are not, however, in agreement with those obtained in adults which show a reduced phase II τ during cycle exercise subsequent to training (e.g. Bailey et al. 2009; Berger et al. 2006; Figueira et al. 2008; Koppo et al. 2004) or consequently with our hypothesis. The cause of this disparity is not readily apparent but may be related to either an age-dependent change in the muscles' potential for oxygen utilisation (Fawkner and Armstrong 2004a) or to the shorter duration of training in children. Alternatively, the failure of this and previous studies to demonstrate differences in the $\dot{V}O_2$ kinetic response in relation to training or training status during cycle ergometry may be related to inappropriate testing modalities. Swimming is

widely accepted to have a significant upper body contribution (Ogita et al. 1996) and hence the testing of swimmers on a cycle ergometer might not be sufficiently specific. Whilst there is a variable contribution of the lower body to swimming, the present results could suggest there was very little effect of training on the legs of the participants tested. Additionally, the lack of difference in $\dot{V}O_2$ kinetics between the T and UT children during cycle exercise might reflect a similar level of habitual physical activity involving walking, running or cycling in the two groups.

Sub-maximal physiological responses: upper body exercise

To our knowledge, this is the first study to characterise $\dot{V}O_2$ kinetics during upper body exercise in children. The phase II τ values reported here for UT children are similar to those of Smith et al. (2006b) and considerably faster than those reported by Koga et al. (1996) and Koppo et al. (2002) for adults. No data are available for comparison with the trained girls phase II τ, even within the adult literature. The primary phase gain for both trained and untrained girls is within the range reported in adult studies (Koppo et al. 2002; Smith et al. 2006b).

This is the first study to report a significant training status-related difference in the $\dot{V}O_2$ kinetics of prepubertal girls. Specifically, the phase II τ was 32% faster for upper body ergometry in the T compared to the UT girls, a finding that emphasises the importance of test mode specificity and indicates that an exercise modality involving the upper body is more appropriate for testing swimmers. Considering that upper body ergometry does not precisely represent the movements involved in swimming, it is likely that greater differences between T and UT girls would be demonstrated if $\dot{V}O_2$ kinetics were to be characterised during a swimming test. Further work is required to develop methodologies to enable such measurements.

Resolution of the mechanisms responsible for the faster $\dot{V}O_2$ kinetics found in the swimmers of this study is impeded by the cross-sectional nature of the study. The comparison of T and UT participants means it is impossible to attribute the differences observed to training *per se*, as the differences may purely be a reflection of genetic traits which predisposed these children to success in swimming.

If physiological adaptations to training did contribute to the differences observed, the faster $\dot{V}O_2$ kinetics could potentially be related to an increased muscle O_2 delivery, greater muscle oxidative capacity, or to differences in muscle fibre type distribution or recruitment subsequent to training. HR kinetics, which may provide a crude estimate of muscle blood flow kinetics (MacPhee et al. 2005), were faster for both exercise modalities in the T girls. However, the $\dot{V}O_2$ and HR kinetics were not related in either group suggesting that O_2 oxygen delivery was not a limiting factor, a conclusion which agrees with adult studies (see Poole et al. 2008a, for review). Endurance training has been shown to enhance muscle oxidative capacity in adults through increases in mitochondrial volume and oxidative enzyme activities (Holloszy 1967; Mogensen et al. 2006). Whether similar adaptations to training are present in pre-pubertal children remains to be resolved, with information regarding the muscle oxidative capacity of T and UT children almost non-existent (Mahon 2008). The limited information available suggests that training may increase muscle oxidative enzyme activity in children (Eriksson et al. 1973; Fournier et al. 1982). An increased mitochondrial volume following endurance training would be predicted to result in faster $\dot{V}O_2$ kinetics (Meyer 1988). In the present study, we are unable to ascertain the physiological mechanism(s) which led to faster $\dot{V}O_2$ kinetics in T compared to UT girls. However, as in adults, this is likely a function of an enhanced integrated capacity to both transport and utilize O_2 (Jones and Koppo 2005; Poole et al. 2008a).

It is possible that the recruitment of muscle fibres of different types may influence the pulmonary phase II τ, since it has been suggested that type I fibres have a faster time constant for the rise in $\dot{V}O_2$ (Crow and Kushmerick 1982; Krustrup et al. 2008). Indirect evidence for an influence of muscle fibre type on $\dot{V}O_2$ kinetics has been provided by the demonstration of a negative correlation between the type I fibres percentage in the vastus lateralis and the pulmonary phase II τ during cycle exercise (Pringle et al. 2003). Such a negative association is pertinent to the current results because some studies report that endurance training induces an increase in the percentage of type I fibres (Russell et al. 2003; Saltin and Gollnick 1983) and/or alters the recruitment pattern to reduce the contribution of type II fibres. Therefore, the faster τ in the current study may be related to training-induced differences in the metabolic properties of the muscle fibres recruited to meet the exercise demand.

There was no significant correlation between the peak $\dot{V}O_2$ and the phase II τ in either the T or the UT group for either exercise mode. This is consistent with some previous studies in both paediatric (Cleuziou et al. 2002; Fawkner et al. 2002) and adult (Carter et al. 2000) populations, but differs from other studies which have suggested that higher peak $\dot{V}O_2$ values are associated with faster phase II $\dot{V}O_2$ kinetics in children (Cooper et al. 1985) and adults (Chilibeck et al. 1996; Powers et al. 1985). Given that both peak $\dot{V}O_2$ and $\dot{V}O_2$ kinetics are acknowledged as parameters of oxidative metabolic function, a relationship between the two might be expected at least in a sample of subjects with heterogeneous aerobic fitness (Poole et al. 2005). That this was not the case in the present study might be a function of the relatively small sample size but might also reflect differences in the factors which are predominantly limiting, i.e., central cardiovascular factors for peak$\dot{V}O_2$ and intrinsic muscle metabolic factors for $\dot{V}O_2$ kinetics (Poole et al. 2008a).

Influence of exercise modality on $\dot{V}O_2$ kinetics

To our knowledge, this is the first study to investigate the influence of exercise modality on the $\dot{V}O_2$ kinetics of T and UT children. The slower phase II τ during upper body ergometry compared to cycle ergometry in the UT girls is consistent with previous reports in adults (Koga et al. 1996; Koppo et al. 2002; Schneider et al. 2002). However, in the T girls there was no difference in the phase II τ between exercise modalities.

The musculature of the arm and leg differ in the percentage of type I muscle fibres and the capillary-to-fibre ratio, both being lower in the arm musculature (Johnson et al. 1973; Turner et al. 1997). Furthermore, the muscle perfusion pressure has been suggested to be lower during arm exercise due to a reduced "gravitational assist" to muscle blood flow compared to upright leg exercise (Jones and Burnley 2005; Koga et al. 1999; Koppo and Bouckaert 2005). These factors could negatively influence the rate of adjustment of $\dot{V}O_2$ to an increased metabolic demand and, therefore, the modality-related differences in the phase II τ in UT girls may be a reflection of these differences. Given the physiological differences in arm and leg musculature including

a higher proportion of type II fibres in the upper body, it is perhaps surprising that no differences were found in the amplitude of the $\dot{V}O_2$ slow component between exercise modalities. However, it is important to note that upper body exercise involves a contribution of accessory muscles, such as the shoulders and back, both in the generation of power and for stabilisation; it is possible that these factors obscure the relationship between fibre recruitment and $\dot{V}O_2$ kinetics (Smith et al. 2006a).

In conclusion, incremental exercise tests did not reveal any influence of training status on GET or peak $\dot{V}O_2$ regardless of exercise modality in pre-pubertal girls. However, T girls had faster phase II $\dot{V}O_2$ kinetics compared to UT girls during upper body but not cycle ergometry. As a consequence, the $\dot{V}O_2$ kinetics of T girls was not different between the exercise modalities, whereas UT girls had slower phase II $\dot{V}O_2$ kinetics during upper body exercise compared to leg exercise. These data therefore highlight the importance of test specificity in revealing training status differences and demonstrate, for the first time, that $\dot{V}O_2$ kinetics are faster in trained compared to untrained pre-pubertal girls.

References

Bailey SJ, Wilkerson DP, DiMenna FJ, Jones AM (2009) Influence of repeated sprint training on pulmonary O_2 uptake and muscle deoxygenation kinetics in humans. J Appl Physiol 106: 1875-1887

Barker AR, Williams CA, Jones AM, Armstrong N (2009) Establishing maximal oxygen uptake in young people during a ramp cycle test to exhaustion. Br J Sports Med. doi: 10.1136/bjsm.2009.063180

Beaver WL, Wasserman K, Whipp BJ (1986) A new method for detecting anaerobic threshold by gas-exchange. J Appl Physiol 60: 2020-2027

Berger NJA, Tolfrey K, Williams AG, Jones AM (2006) Influence of continuous and interval training on oxygen uptake on-kinetics. Med Sci Sports Exerc 38: 504-512

Bhambhani Y, Maikala R, Buckley S (1998) Muscle oxygenation during incremental arm and leg exercise in men and women. Eur J Appl Physiol 78: 422-431

Boone J, Koppo K, Bouckaert J (2008) The $\dot{V}O_2$ response to submaximal ramp cycle exercise: Influence of ramp slope and training status. Respir Physiol Neurobiol 161: 291-297

Carter H, Jones AM, Barstow TJ, Burnley M, Williams C, Doust JH (2000) Effect of endurance training on oxygen uptake kinetics during treadmill running. J Appl Physiol 89: 1744-1752

Casaburi R, Storer TW, Ben-Dov I, Wasserman K (1987) Effect of endurance training on possible determinants of $\dot{V}O_2$ during heavy exercise. J Appl Physiol 62: 199-207

Caso P, D'Andrea A, Galderisi M, Liccardo B, Severino S, De Simone L, Izzo A, D'Andrea L, Mininni N (2000) Pulsed doppler tissue imaging in endurance athletes: Relation between left ventricular preload and myocardial regional diastolic function. Am J Cardiol 85: 1131-1136

Chilibeck PD, Paterson DH, Petrella RJ, Cunningham DA (1996) The influence of age and cardiorespiratory fitness on kinetics of oxygen uptake. Can J Appl Physiol 21: 185-196

Cleuziou C, Lecoq AM, Candau R, Courteix D, Guenon P, Obert P (2002) Kinetics of oxygen uptake at the onset of moderate and heavy exercise in trained and untrained prepubertal children. Sci Sports 17: 291-296

Cleuziou C, Perrey S, Borrani F, Lecoq AM, Candau R, Courteix D, Obert P (2003) Dynamic responses of O_2 uptake at the onset and end of exercise in trained subjects. Can J Appl Physiol 28: 630-641

Cooper DM, Berry C, Lamarra N, Wasserman K (1985) Kinetics of oxygen-uptake and heart-rate at onset of exercise in children. J Appl Physiol 59: 211-217

Crow MT, Kushmerick MJ (1982) Chemical energetics of slow-twitch and fast-twitch muscles of the mouse. J Gen Physiol 79: 147-166

Daly RM, Rich PA, Klein R (1998) Hormonal responses to physical training in high-level peripubertal male gymnasts. Eur J Appl Physiol Occup Physiol 79: 74-81

Davis JA, Vodak P, Wilmore JH, Vodak J, Kurtz P (1976) Anaerobic threshold and maximal aerobic power for 3 modes of exercise. J Appl Physiol 41: 544-550

Eriksson BO, Gollnick PD, Saltin B (1973) Muscle metabolism and enzyme activities after training in boys 11-13 years old. Acta Physiol Scand 87: 485-497

Fawkner S, Armstrong N (2003) Oxygen uptake kinetic response to exercise in children. Sports Med 33: 651-669

Fawkner S, Armstrong N (2004a) Longitudinal changes in the kinetic response to heavy-intensity exercise in children. J Appl Physiol 97: 460-466

Fawkner S, Armstrong N (2004b) Modelling the $\dot{V}O_2$ kinetic response to heavy intensity exercise in children. Ergonomics 47: 1517-1527

Fawkner S, Armstrong N (2004c) Sex differences in the oxygen uptake kinetic response to heavy-intensity exercise in prepubertal children. Eur J Appl Physiol 93: 210-216

Fawkner S, Armstrong N (2007) Can we confidently study $\dot{V}O_2$ kinetics in young people? J Sport Sci Med 6: 277-285

Fawkner S, Armstrong N, Potter CR, Welsman J (2002) Oxygen uptake kinetics in children and adults after the onset of moderate-intensity exercise. J Sports Sci 20: 319-326

Figueira TR, Caputo F, Machado CEP, Denadai BS (2008) Aerobic fitness level typical of elite athletes is not associated with even faster $\dot{V}O_2$ kinetics during cycling exercise. J Sport Sci Med 7: 132-138

Fournier M, Ricci J, Taylor AW, Ferguson RJ, Montpetit RR, Chaitman BR (1982) Skeletal-muscle adaptation in adolescent boys - sprint and endurance training and detraining. Med Sci Sports Exerc 14: 453-456

George KP, Wolfe LA, Burggraf GW (1991) The athletic heart syndrome - a critical review. Sports Med 11: 300-330

Holloszy JO (1967) Biochemical adaptations in muscle - effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. J Biol Chem 242: 2278-&

Johnson MA, Polgar J, Weightma.D, Appleton D (1973) Data on distribution of fiber types in 36 human muscles - autopsy study. J Neurol Sci 18: 111-129

Jones AM, Burnley M (2005) Effect of exercise modality on $\dot{V}O_2$ kinetics. In: Jones AM, Poole DC (eds) Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London, pp. 95-114

Jones AM, Koppo K (2005) Effect of training on $\dot{V}O_2$ kinetics and performance. In: Jones AM, Poole DC (eds) Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London, pp. 373-397

Jones AM, Poole DC (2005) Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London

Katch VL (1983) Physical conditioning of children. J Adolesc Health 3: 241-246

Koga S, Shiojiri T, Shibasaki M, Fukuba Y, Fukuoka Y, Kondo N (1996) Kinetics of oxygen uptake and cardiac output at onset of arm exercise. Respir Physiol 103: 195-202

Koga S, Shiojiri T, Shibasaki M, Kondo N, Fukuba Y, Barstow TJ (1999) Kinetics of oxygen uptake during supine and upright heavy exercise. J Appl Physiol 87: 253-260

Koppo K, Bouckaert J (2005) Prior arm exercise speeds the $\dot{V}O_2$ kinetics during arm exercise above the heart level. Med Sci Sports Exerc 37: 613-619

Koppo K, Bouckaert J, Jones AM (2002) Oxygen uptake kinetics during high-intensity arm and leg exercise. Respir Physiol Neurobiol 133: 241-250

Koppo K, Bouckaert J, Jones AM (2004) Effects of training status and exercise intensity on phase II $\dot{V}O_2$ kinetics. Med Sci Sports Exerc 36: 225-232

Krustrup P, Secher NH, Relu MU, Hellsten Y, Soderlund K, Bangsbo J (2008) Neuromuscular blockade of slow twitch muscle fibres elevates muscle oxygen uptake and energy turnover during submaximal exercise in humans. J Physiol-London 586: 6037-6048

MacPhee SL, Shoemaker JK, Paterson DH, Kowalchuk JM (2005) Kinetics of O_2 uptake, leg blood flow, and muscle deoxygenation are slowed in the upper compared with lower region of the moderate-intensity exercise domain. J Appl Physiol 99: 1822-1834

Mahon AD (2008) Aerobic training. In: Armstrong N, van Mechelen W (eds) Paediatric Exercise Science and Medicine. Oxford University Press, Oxford, pp. 513-529

Meyer RA (1988) A linear-model of muscle respiration explains monoexponential phosphocreatine changes. Am J Physiol 254: C548-C553

Mirwald RL, Baxter-Jones ADG, Bailey DA, Beunen GP (2002) An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc 34: 689-694

Mogensen M, Bagger M, Pedersen PK, Fernstrom M, Sahlin K (2006) Cycling efficiency in humans is related to low UCP3 content and to type I fibres but not to mitochondrial efficiency. J Physiol-London 571: 669-681

Norris SR, Petersen SR (1998) Effects of endurance training on transient oxygen uptake responses in cyclists. J Sports Sci 16: 733-738

Nottin S, Nguyen LD, Terbah M, Obert P (2004) Left ventricular function in endurance-trained children by tissue Doppler imaging. Med Sci Sports Exerc 36: 1507-1513

Nottin S, Vinet A, Stecken F, N'Guyen LD, Ounissi F, Lecoq AM, Obert P (2002) Central and peripheral cardiovascular adaptations to exercise in endurance-trained children. Acta Physiol Scand 175: 85-92

Obert P, Cleuziou C, Candau R, Courteix D, Lecoq AM, Guenon P (2000) The slow component of O_2 uptake kinetics during high-intensity exercise in trained and untrained prepubertal children. Int J Sports Med 21: 31-36

Obert P, Mandigouts S, Nottin S, Vinet A, N'Guyen LD, Lecoq AM (2003) Cardiovascular responses to endurance training in children: effect of gender. Eur J Clin Invest 33: 199-208

Obert P, Nottin S, Baquet G, Thevenet D, Gamelin FX, Berthoin S (2009) Two months of endurance training does not alter diastolic function evaluated by TDI in 9-11-year-old boys and girls. Br J Sports Med 43: 132-135

Ogita F, Hara M, Tabata I (1996) Anaerobic capacity and maximal oxygen uptake during arm stroke, leg kicking and whole body swimming. Acta Physiol Scand 157: 435-441

Phillips SM, Green HJ, MacDonald MJ, Hughson RL (1995) Progressive effect of endurance training on $\dot{V}O_2$ kinetics at the onset of submaximal exercise. J Appl Physiol 79: 1914-1920

Poole DC, Barstow TJ, McDonough P, Jones AM (2008a) Control of oxygen uptake during exercise. Med Sci Sports Exerc 40: 462-474

Poole DC, Kindig CA, Behnke BJ, Jones AM (2005) Oxygen uptake kinetics in different species: a brief review. Equine Comp Exerc Physiol 2: 1-15

Poole DC, Wilkerson DP, Jones AM (2008b) Validity of criteria for establishing maximal O_2 uptake during ramp exercise tests. Eur J Appl Physiol 102: 403-410

Powers SK, Dodd S, Beadle RE (1985) Oxygen uptake kinetics in trained athletes differing in $\dot{V}O_2$ max. Eur J Appl Physiol 54: 306-308

Pringle JSM, Doust JH, Carter H, Tolfrey K, Campbell IT, Jones AM, Sakkas GK (2003) Oxygen uptake kinetics during moderate, heavy and severe intensity 'submaximal' exercise in humans: the influence of muscle fibre type and capillarisation. Eur J Appl Physiol 89: 289-300

Rowland T, Bougault V, Walther G, Nottin S, Vinett A, Obert P (2009) Cardiac responses to swim bench exercise in age-group swimmers and non-athletic children. J Sci Med Sport 12: 266-272

Rowland TW, Unnithan VB, Macfarlane NG, Gibson NG, Paton JY (1994) Clinical manifestations of the athletes heart in prepubertal male runners. Int J Sports Med 15: 515-519

Russell AP, Wadley G, Hesselink MKC, Schaart G, Lo S, Leger B, Garnham A, Kornips E, Cameron-Smith D, Giacobino JP, Muzzin P, Snow R, Schrauwen P (2003) UCP3 protein expression is lower in type I, IIa and IIx muscle fiber types of endurance-trained compared to untrained subjects. Pflugers Arch 445: 563-569

Saltin B, Gollnick PD (1983) Skeletal muscle adaptability: significance for metabolism and performance. In: Peachy LD (ed) Handbook of Physiology. Physiological Society, Bethesda, MD, pp. 555-631

Schneider DA, Wing AN, Morris NR (2002) Oxygen uptake and heart rate kinetics during heavy exercise: a comparison between arm cranking and leg cycling. Eur J Appl Physiol 88: 100-106

Shephard RJ (1992) Effectiveness of training programmes for prepubescent children. Sports Med 13: 194-213

Smith PM, Amaral I, Doherty M, Price MJ, Jones AM (2006a) The influence of ramp rate on $\dot{V}O_2$ peak and "excess" $\dot{V}O_2$ during arm crank ergometry. Int J Sports Med 27: 610-616

Smith PM, McCrindle E, Doherty M, Price MJ, Jones AM (2006b) Influence of crank rate on the slow component of pulmonary O_2 uptake during heavy arm-crank exercise. Appl Physiol Nutr Metab 31: 292-301

Tanner JM (1962) Growth of Adolescence. Blackwell Scientific, Oxford

Tsolakis C, Vagenas G, Dessypris A (2003) Growth and anabolic hormones, leptin, and neuromuscular performance in moderately trained prepubescent athletes and untrained boys. J Strength Cond Res 17: 40-46

Turner DL, Hoppeler H, Claassen H, Vock P, Kayser B, Schena F, Ferretti G (1997) Effects of endurance training on oxidative capacity and structural composition of human arm and leg muscles. Acta Physiol Scand 161: 459-464

Welsman J, Bywater K, Farr C, Welford D, Armstrong N (2005) Reliability of peak $\dot{V}O_2$ and maximal cardiac output assessed using thoracic bioimpedance in children. Eur J Appl Physiol 94: 228-234

Welsman JR, Armstrong N (2000) Statistical techniques for interpreting body size-related exercise performance during growth. Pediatr Exerc Sci 12: 112-127

Welsman JR, Armstrong N, Chedzoy S, Withers S (1996) Aerobic training in 10 year old and adult females. Med Sci Sports Exerc 28: 3

Whipp BJ, Ward SA (1990) Physiological determinants of pulmonary gas-exchange kinetics during exercise. Med Sci Sports Exerc 22: 62-71

Zakas A, Mandroukas K, Karamouzis G, Panagiotopoulou G (1994) Physical training, growth hormone and testosterone levels and blood pressure in prepubertal, pubertal and adolescent boys. Scand J Med Sci Sports 4: 113-118

Table 1. Morphological characteristics of participants

	Trained	**Untrained**
Age (y)	11.4 ± 0.7	11.5 ± 0.6
Stature (m)	1.48 ± 0.06	1.52 ± 0.05
Mass (kg)	39.9 ± 6.9	43.2 ± 8.6
Sum of skinfolds (mm)	36.4 ± 14.6	43.8 ± 25.6
Years to PHV (y)	-0.46 ± 0.5	-0.31 ± 0.45

Values are mean ± SD. No significant differences were present. N = 8

Table 2. Peak physiological responses to exercise on a cycle and upper body ergometer in trained and untrained girls

	Cycle Ergometry		**Upper body Ergometry**	
	Trained	Untrained	Trained	Untrained
Peak $\dot{V}O_2$ ($L.min^{-1}$)	1.69 ± 0.23	1.60 ± 0.30	1.13 ± 0.13 #	1.09 ± 0.19 ##
Peak $\dot{V}O_2$ ($mL.kg^{-1}.min^{-1}$)	42.9 ± 4.9	37.8 ± 7.7	29.0 ± 5.5 #	25.4 ± 2.9 ##
Peak $\dot{V}O_2$ ($mL.kg^{-0.56}.min^{-1}$)	311 ± 30	285 ± 47	260 ± 33 #	242 ± 31
Peak HR ($b.min^{-1}$)	192 ± 7	195 ± 8	177 ± 6 #	180 ± 17 ##
Peak RER	1.11 ± 0.08	1.18 ± 0.13	1.07 ± 0.05	1.16 ± 0.12
Peak blood [lactate] (mM)	5.0 ± 0.9	5.3 ± 1.2	3.7 ± 0.6	3.9 ± 0.7
Peak WR (W)	142 ± 12	132 ± 25	59 ± 6	58 ± 11
GET ($L.min^{-1}$)	0.84 ± 0.06	0.88 ± 0.17	0.53 ± 0.07 #	0.57 ± 0.10 ##
GET (% peak $\dot{V}O_2$)	51 ± 7	58 ± 3	50 ± 9	54 ± 5

Values are mean ± SD. HR, heart rate; RER, respiratory exchange ratio; blood [lactate], blood lactate concentration; WR, work rate; GET, gas exchange threshold. N = 8

*Significant difference between modes in trained children (P < 0.01)*

*Significant difference between modes in untrained children (P < 0.01)*

Table 3. Oxygen uptake kinetics and blood [lactate] during heavy-intensity exercise on a cycle and upper body ergometer in trained and untrained girls

	Cycle Ergometry		**Upper Body Ergometry**	
	Trained	Untrained	Trained	Untrained
Baseline $\dot{V}O_2$ ($L.min^{-1}$)	0.44 ± 0.04	0.55 ± 0.06 *	0.34 ± 0.02 #	0.40 ± 0.05 **, ##
Phase II time delay (s)	11 ± 3	12 ± 3	12 ± 2	9 ± 4
Phase II τ (s)	25 ± 5	25 ± 7	25 ± 3	37 ± 6 **, ##
95% confidence interval (s)	3 ± 1	4 ± 0	4 ± 1	4 ± 0
Phase II amplitude ($L.min^{-1}$)	0.83 ± 0.16	0.64 ± 0.17	0.45 ± 0.07 #	0.51 ± 0.18
Phase II gain ($mLO_2.min^{-1}.W^{-1}$)	9.2 ± 0.9	8.9 ± 2.7	12.2 ± 2.5 #	12.5 ± 1.9 ##
Slow component amplitude ($L.min^{-1}$)	0.08 ± 0.04	0.06 ± 0.03	0.05 ± 0.4	0.05 ± 0.02
Slow component amplitude (% end exercise $\dot{V}O_2$)	10 ± 4	9 ± 5	9 ± 5	8 ± 2
End-exercise $\dot{V}O_2$ ($L.min^{-1}$)	1.35 ± 0.19	1.26 ± 0.17	0.83 ± 0.09 #	0.95 ± 0.23 ##
Blood [lactate] (mM)	3.9 ± 0.9	4.1 ± 1.4	2.7 ± 0.6 #	3.3 ± 1.0

Values are mean ± SD. τ, time constant; blood [lactate], blood lactate concentration. N = 8

* *Significant difference between trained and untrained in cycle ergometer response (P < 0.01*

** *Significant difference between trained and untrained in upper body response (P < 0.01)*

\# *Significant difference between modes in trained children (P < 0.01)*

\## *Significant difference between modes in untrained children (P < 0.01)*

Table 4. Heart rate kinetics during heavy-intensity exercise on a cycle and upper body ergometer in trained and untrained girls

	Cycle Ergometry		**Upper Body Ergometry**	
	Trained	Untrained	Trained	Untrained
HR baseline ($b.min^{-1}$)	91 ± 8	112 ± 9 *	93 ± 6	101 ± 5 **,##
HR phase II τ (s)	31 ± 11	47 ± 9 *	33 ± 8	43 ± 4 **
HR phase II amplitude ($b.min^{-1}$)	62 ± 8	54 ± 10	48 ± 7 #	41 ± 10 ##
End-exercise HR ($b.min^{-1}$)	169 ± 9	179 ± 9	151 ± 12 #	157 ± 17 ##
95% confidence interval (s)	1 ± 0	2 ± 1	1 ± 0	2 ± 1

Values are mean ± SD. HR, heart rate; τ, time constant. N = 8

* *Significant difference between trained and untrained in cycle ergometer response (P < 0.01)*

** *Significant difference between trained and untrained in upper body response (P < 0.01)*

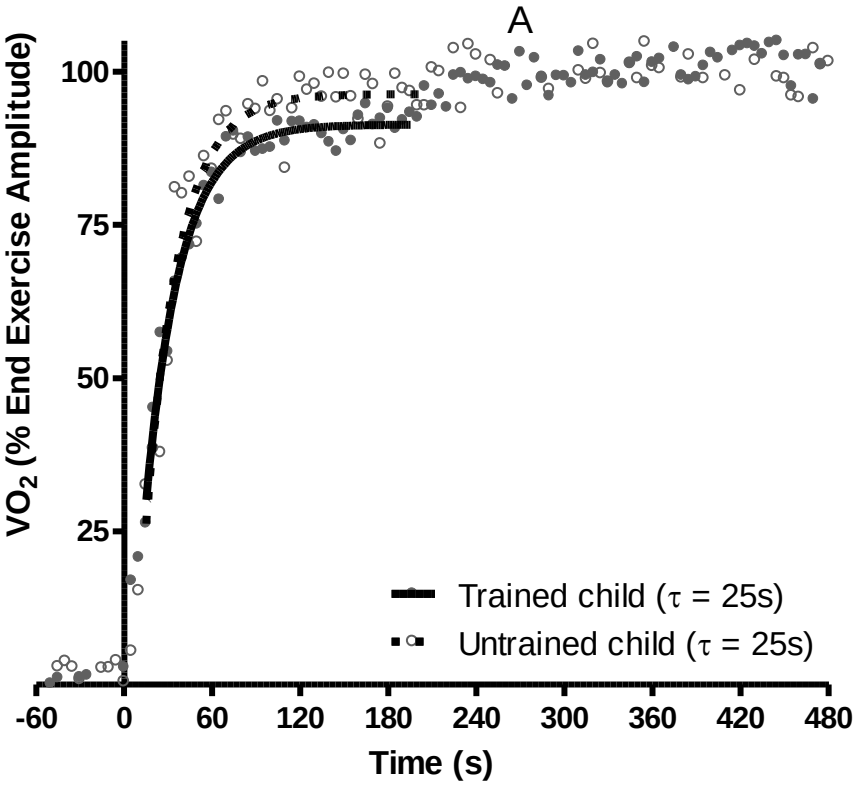
\# *Significant difference between modes in trained children (P < 0.01)*

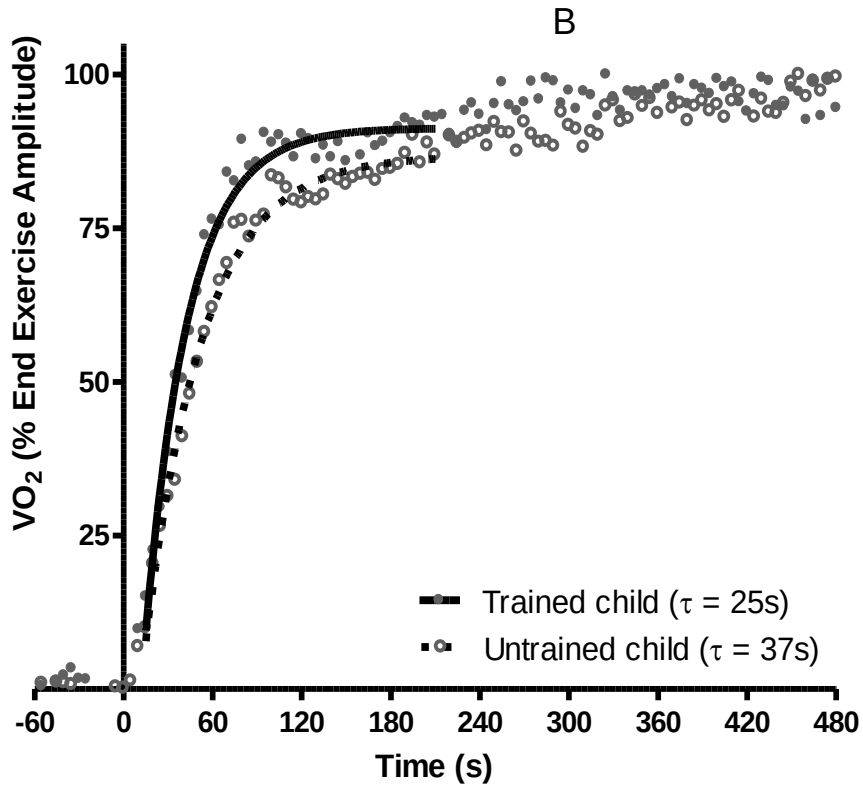
\#\# *Significant difference between modes in untrained children (P < 0.01)*

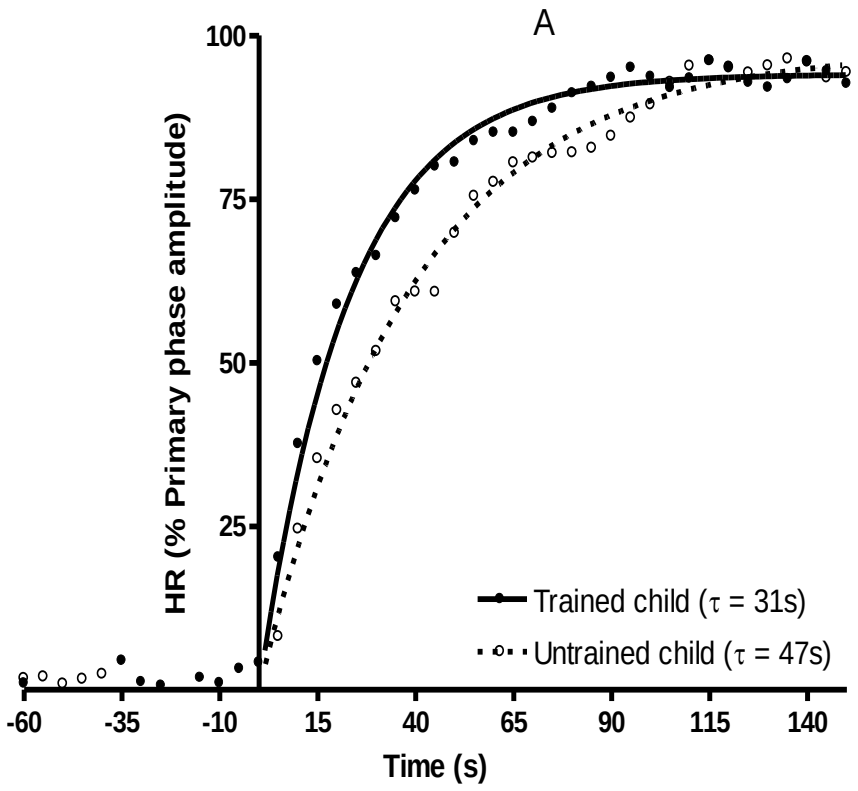
Figures

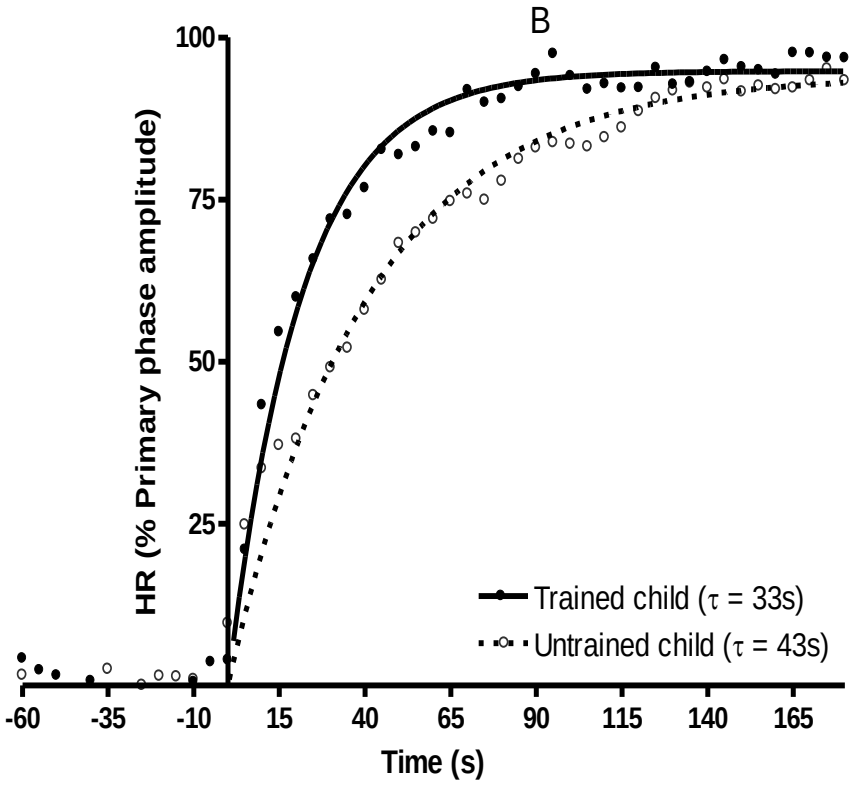
Fig. 1. Pulmonary oxygen uptake response to a step increment in work rate from an unloaded baseline to a heavy intensity work rate (40%Δ) in a representative trained and untrained participant. Graph A shows the response during cycle ergometer exercise and graph B the response to upper body ergometer exercise. The data expressed as a percentage of the end exercise amplitude. The trained girls are shown as closed circles and the untrained girls are shown as open circles. The solid and dashed lines represent the mono-exponential model fit of the data. Note the significantly faster τ in the trained participant during upper body exercise. For clarity, data are displayed as 5-s bin averages

Fig. 2. Heart rate response to a step increment in work rate from an unloaded baseline to a heavy intensity work rate (40%Δ) in a representative trained and untrained participant. Graph A shows the response during cycle ergometer exercise and graph B the response to upper body ergometer exercise. The data expressed as a percentage of the primary phase amplitude. The trained girls are shown as closed circles and the untrained girls are shown as open circles. The solid and dashed lines represent the mono-exponential model fit of the data. Note the significantly faster τ in the trained participant during both exercise modalities. For clarity, data are displayed as 5-s bin averages


A
100
75
50
25
VO2 (% End Exercise Amplitude)
Trained child (τ = 25s)
Untrained child (τ = 25s)
-60
0
60
120
180
240
300
360
420
480
Time (s)


B
100
75
50
25
VO2 (% End Exercise Amplitude)
Trained child (τ = 25s)
Untrained child (τ = 37s)
-60
0
60
120
180
240
300
360
420
480
Time (s)


A
100
75
50
25
HR (% Primary phase amplitude)
Trained child (τ = 31s)
Untrained child (τ = 47s)
-60
-35
-10
15
40
65
90
115
140
Time (s)


B
100
75
50
25
HR (% Primary phase amplitude)
Trained child (τ = 33s)
Untrained child (τ = 43s)
-60
-35
-10
15
40
65
90
115
140
165
Time (s)