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

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Abstract

The influence of training status on the oxygen uptake ($\dot{V}O_2$) response to heavy intensity exercise in pubertal girls has not previously been investigated. We hypothesised that whilst training status-related adaptations would be evident in the $\dot{V}O_2$, heart rate (HR) and deoxyhemoglobin ([HHb]) kinetics of pubertal swimmers during both lower and upper body exercise, they would be more pronounced during upper body exercise. Eight swim-trained (T; 14.2±0.7 years) and eight untrained (UT; 14.5±1.3 years) girls completed a number of constant-work-rate transitions on cycle and upper body ergometers 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 (τ) was significantly shorter in the trained girls during both cycle (T: 21 ± 6 vs. UT: 35 ± 11 s; P<0.01) and upper body exercise (T: 29 ± 8 vs. UT: 44 ± 8 s; P<0.01). The $\dot{V}O_2$ slow component was not influenced by training status. The [HHb] τ was significantly shorter in the trained girls during both cycle (T: 12 ± 2 vs. UT: 20 ± 6 s; P<0.01) and upper body exercise (T: 13 ± 3 vs. UT: 21 ± 7 s; P<0.01), as was the HR τ (cycle, T: 36 ± 5 vs. UT: 53 ± 9 s; upper body, T: 32 ± 3 vs. UT: 43 ± 2; P<0.01). This study suggests that both central and peripheral factors contribute to the faster $\dot{V}O_2$ kinetics in the trained girls and that differences are evident in both lower and upper body exercise.

Keywords: Oxygen uptake kinetics; near-infrared spectroscopy; training, children; adolescents; upper body exercise

Introduction

Following a sudden increase in the external work rate, pulmonary oxygen uptake ($\dot{V}O_2$) increases in a predictable, exercise-intensity dependent manner. Below the gas exchange threshold (GET), the $\dot{V}O_2$ response is characterised by 3 phases: an initial cardiodynamic phase which reflects the rapid elevation in cardiac output and pulmonary blood flow, a second phase during which $\dot{V}O_2$ increases exponentially (reflecting the increasing muscle $\dot{V}O_2$; Grassi et al. 1996; Krustrup et al. 2009), and a final steady-state phase which is typically achieved after 2-3 minutes of constant-work-rate exercise (Whipp et al. 1982; Whipp and Wasserman 1972). Above the GET the attainment of a steady state is delayed, or even precluded, by the presence of a supplementary "slow component" of $\dot{V}O_2$ (Barstow and Molé 1991; Whipp and Wasserman 1972).

The $\dot{V}O_2$ kinetic response has been demonstrated to be highly adaptive in adults, with one of the most potent interventions being exercise training. Endurance training has been shown to result in a faster phase II time constant (τ) and a reduced contribution of the slow component to the total increase in $\dot{V}O_2$ (e.g. Bailey et al. 2009; Koppo et al. 2004; Powers et al. 1985). The temporal features of the $\dot{V}O_2$ response have similarly been shown to be influenced by training status in pre-pubertal children, provided that an appropriate test modality is used (Winlove et al. 2010), although this remains controversial (Cleuziou et al. 2002; Obert et al. 2000).

There is a dearth of studies investigating the influence of training status on $\dot{V}O_2$ kinetics in pubertal populations. In the only previous study, adolescent male football players demonstrated significantly faster $\dot{V}O_2$ kinetics during moderate intensity cycle exercise (Marwood et al. 2010). Whether training status similarly influences the τ or slow component of $\dot{V}O_2$ during heavy intensity exercise in adolescents is unknown. This question warrants investigation given reports in adults that the effects of training status on $\dot{V}O_2$ kinetics may be exercise-intensity dependent (Bailey et al. 2009; Carter et al. 2000; Krustrup et al. 2004).

The mechanistic bases for training status-related differences in $\dot{V}O_2$ kinetics are unclear. While $\dot{V}O_2$ kinetics is accepted to be regulated by both muscle O_2 delivery and O_2 utilisation, the relative importance of each factor in different populations, at different intensities and in different exercise modalities remains a topic of debate (Jones and Burnley, 2005; Poole et al. 2008; Tschakovsky and Hughson 1999). In adults, it appears that the training-induced speeding of the phase II τ may be predominantly related to an enhanced O_2 extraction at the muscle (Bailey et al. 2009; Krustrup et al. 2004) due to an increased mitochondrial volume and oxidative enzyme activity (Krustrup et al. 2004; Phillips et al. 1995) and/or to an enhanced matching of perfusion to metabolic demand (Murias et al. 2010). Bulk muscle blood flow has also been shown to be enhanced following training (Laughlin and Roseguini 2008; Shoemaker et al. 1996) and therefore cannot be eliminated as a potential explanatory variable (McKay et al. 2009). Information on the extent to which training status-related differences in $\dot{V}O_2$ kinetics are related to central or peripheral factors in adolescents is sparse. Marwood et al (2010) have recently reported that during moderate intensity exercise male adolescent football players had faster heart rate kinetics but unaltered deoxygenated haemoglobin/myoglobin ([HHb]) kinetics compared to untrained subjects. The authors interpreted these results as evidence that training enhanced both muscle O_2 delivery and fractional O_2 extraction (Marwood et al. 2010). The mechanistic basis for any differences in $\dot{V}O_2$ kinetics during heavy intensity exercise in adolescents has yet to be investigated, with available techniques necessarily indirect due to the ethical considerations associated with testing young populations.

A fundamental limitation of many of the studies investigating the influence of training status on $\dot{V}O_2$ kinetics in adolescents is a lack of commonality between the muscles trained and the muscles tested. Indeed, the importance of exercise modality to the investigation of training status influences on $\dot{V}O_2$ kinetics has previously been reported in pre-pubertal girls where effects were evident in upper body but not lower body exercise (Winlove et al. 2010).

The purpose of this cross-sectional study was to assess the influence of training status on the kinetics of $\dot{V}O_2$, HR and muscle deoxygenation in pubertal girls during heavy-intensity exercise. We hypothesised that, in accordance with findings in adults, the $\dot{V}O_2$ kinetics would be faster and that the $\dot{V}O_2$ slow component would be relatively smaller in trained swimmers compared to an age-matched untrained control group. We also hypothesised, given the large upper body contribution to swimming (Ogita et al. 1996), that training status-related differences would be more evident during upper body (arm crank ergometer) than during lower body (cycle ergometer) exercise.

Methods

Participants

Eight endurance-trained (T) girls and 8 untrained (UT) girls aged 13-15 years participated in this study. The T group, all competitive swimmers with a mean training volume of 12 (± 2) hours/week and who had been swimming for an average of 5.2 (± 0.7) years, 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). All children reported a maturity level of 3 or 4, indicating the study population was pubertal. Age to peak height velocity was estimated to provide an additional indicator of physical maturity according to the equations of Mirwald *et al*. (2002).

All participants were anthropometrically evaluated during the first visit to the laboratory. Standing and seated height was measured to 0.1 cm using a Holtain stadiometer (Holtain, Crymych, Dyfed, UK) and body mass was determined to 0.05 kg using Avery beam balance scales (Avery, Birmingham, UK). Skinfold thickness was assessed three times at four sites around the body (biceps, 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 0.2 mm. The average of the three measurements was taken. Table 1 presents the participants' physical characteristics.

Participants were asked to arrive at the laboratory in a rested and fully hydrated state, at least 3 hours postprandial and to have avoided consuming caffeinated drinks for 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.

Incremental Test

On the first two visits to the laboratory, exercise mode-specific peak $\dot{V}O_2$ and gas exchange threshold (GET) were determined using a ramp incremental test to voluntary exhaustion on both cycle (Lode Excalibur, Netherlands) and upper body (Lode Angio, Netherlands) ergometers. The handle bar height, seat height and crank length (cycle ergometer) and electrically controlled seat height and distance (upper body ergometer) were adjusted to suit each participant 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 (equivalent to 10W at 70 rpm according to the manufacturer's guidelines), the resistance increased by 20 $W \cdot min^{-1}$ and 10 $W \cdot min^{-1}$ for cycle and upper body exercise, respectively, to attain a test of ~8-12 minutes in duration (Buchfuhrer et al. 1983). Participants were instructed to maintain a cadence within the range of 70 ± 5 and 50 ± 5 rpm on the cycle and upper body ergometer, respectively. The peak $\dot{V}O_2$ was defined 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 written 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" tests on the upper body and cycle ergometers for the determination of $\dot{V}O_2$ kinetics. 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, 3 cycle and 4 upper body transitions were completed, depending on the number of transitions required to obtain 95% confidence intervals of

< 4 s for the phase II $\dot{V}O_2$ τ. All constant-work-rate tests consisted of 4 minutes of unloaded pedalling or cranking followed by an 'instantaneous' transition to a work rate calculated to require 40% of the difference between the GET and peak $\dot{V}O_2$ (40%Δ) for 8 minutes. At 8 minutes the work rate returned to an unloaded baseline at which 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.

Measurements

Throughout all the tests, 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 analysers were 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. During each transition, 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).

For at least one transition for each exercise modality, the oxygenation status of the right *m. vastus lateralis* (cycle) or right *m. tricep brachii* (upper body) was monitored using a commercially available near-infrared system (NIRO 300, Hamamatsu Photonics KK, Hiugashi-ku, Japan). This system consists of an emission probe which emits four wavelengths of light (776, 826, 845 and 905 nm) and a photon detector. The intensity of incident and transmitted light was recorded continuously at 2 Hz and used to estimate the concentration changes relative to baseline levels for oxygenated, deoxygenated and total haemoglobin. The [HHb] signal was used as an indicator of O_2 extraction within the field of interrogation (DeLorey et al. 2003; Ferreira et al. 2007; Grassi et al. 2003; Jones et al. 2006). The contribution of myoglobin to the NIRS signal is currently unresolved (Seiyama et al. 1988; Masuda et al. 2010). Therefore, the [HHb] signal described throughout this paper should be considered to

refer to the combined concentration of both deoxygenated haemoglobin and myoglobin.

The muscle was initially cleaned and the probes placed in a rubber holder which was adhered to the skin at the midpoint of the muscle. To ensure the holder and its probes remained stationary during exercise and to minimise the interference of extraneous light with the near-infrared signal a bandage was wrapped around the arm/leg. The position of the holder relative to the fibular head or ulna head was recorded to enable accurate replication in subsequent tests. The NIRS signal was zeroed with the participant at rest in a seated position with the muscle stationary and relaxed.

$\dot{V}O_2$ 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 mono-exponential model with a time delay (Eq.1) was then applied to the averaged response and kinetic 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 s 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 (Rossiter et al. 2001). 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 $\dot{V}O_2$. The functional gain of the primary $\dot{V}O_2$ response was also calculated by dividing the primary phase amplitude by the change in work rate.

[HHb] & HR Kinetics Analysis

The [HHb] and HR responses to exercise were also modelled. The responses to each transition were interpolated to 1 s intervals, time aligned and averaged to produce a single data set. The resulting [HHb] response was fitted with a mono-exponential with a time delay (Eq.1) whereas the HR response was modelled by a mono-exponential without a time delay (Eq.2). For both responses the fitting window started at $t = 0$ and was constrained to the onset of the "slow component".

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

where ΔHR is the increase in heart rate at time t above the baseline (calculated as the mean heart rate from the first 45 s of the last minute of baseline pedalling), and A_1 and τ_1 are the primary component amplitude and time constant, respectively. The [HHb] time delay (TD) and τ were summed, giving the [HHb] mean response time (MRT), which provides information on the overall [HHb] response over the fundamental phase of the response.

Statistics

The allometric relationship between body mass and peak $\dot{V}O_2$ was determined using analysis of covariance (ANCOVA) on log transformed data. 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. Pearson product-moment correlation coefficients were used to assess the strength of relationships between variables. All data are presented as means ± SD. Statistical significance was accepted when $P < 0.05$.

Results

The physiological responses to ramp incremental exercise on the cycle and upper body ergometers are summarised in Table 2. The trained girls demonstrated significantly higher absolute and relative peak $\dot{V}O_2$ for both exercise modalities. Allometric scaling did not alter the difference in peak $\dot{V}O_2$ between trained and untrained girls for either exercise modality. Similarly, peak work rate was significantly higher in the trained girls for both cycle and upper body ergometry. Peak heart rate and the fraction of peak $\dot{V}O_2$ at which the GET occurred were not affected by training status. Cycle ergometry elicited significantly higher response values compared to upper body ergometry, with the exception of peak heart rate and the fraction of peak $\dot{V}O_2$ at which the GET occurred. The difference between trained and untrained girls peak $\dot{V}O_2$ was less during cycle (~28% higher in trained) than during upper body (~36% higher in trained) ergometry.

$\dot{V}O_2$ kinetics

The parameters determined from the monoexponential modelling revealed a significant influence of training status on the $\dot{V}O_2$ kinetics, as presented in Table 3 and illustrated in Figure 1. Specifically, the phase II τ was significantly lower and the phase II amplitude significantly greater in the trained girls during both cycle and upper body ergometry. The $\dot{V}O_2$ slow component response was not affected by training status for either exercise modality. The temporal aspects of the $\dot{V}O_2$ response

were not affected by exercise modality in contrast to the amplitude related parameters, such as the baseline $\dot{V}O_2$, phase II amplitude and end-exercise $\dot{V}O_2$, which were significantly higher during cycle ergometry. Exercise modality also influenced the primary gain, with upper body ergometry associated with a higher O_2 cost per Watt compared to cycle ergometry. A significant correlation was evident between the phase II τ during cycle and upper body ergometry ($r = 0.57$; $P < 0.05$).

[HHb] kinetics

The [HHb] kinetics, summarised in Table 4 and illustrated in Figure 2, were influenced by training status, with the τ and MRT being significantly shorter in the trained participants irrespective of exercise modality. The influence of exercise modality was limited, with the only difference being the shorter [HHb] time delay and greater amplitude during upper body ergometry.

Heart Rate Kinetics

The HR kinetics, summarised in Table 5, were significantly affected by training status: both a lower baseline and a shorter τ were evident in the trained girls. The trained status was also associated with a larger HR amplitude during arm crank ergometry. Exercise modality influenced the amplitude but not the kinetics of the HR response, with a greater amplitude and a higher baseline combining to elicit a significantly higher end exercise HR during cycle ergometry. There was a significant correlation between the HR τ during the cycle and upper body exercise ($r = 0.69$; $P < 0.01$).

Discussion

This study is the first to investigate the influence of training status and exercise modality on the $\dot{V}O_2$, HR and [HHb] kinetics of pubertal girls. Additionally, this is the first study to report the influence of training status in adolescents on $\dot{V}O_2$ kinetics during heavy intensity exercise. Consistent with our hypotheses, training status significantly influenced the $\dot{V}O_2$, HR and [HHb] kinetics. Specifically, in response to the transition to a heavy-intensity work rate, trained girls exhibited a smaller phase II $\dot{V}O_2$ τ, HR τ and [HHb] τ (i.e. faster kinetics) although there was no difference in the

relative magnitude of the $\dot{V}O_2$ slow component between trained and untrained girls. However, contrary to our hypothesis, the influence of training status was equally evident during both upper and lower body exercise.

Prior to discussing the possible physiological mechanisms responsible for the influence of training status on the $\dot{V}O_2$ kinetics during both exercise modalities, it is important to acknowledge the limitations of the current study. The most significant impediment to interpretation of the results is the cross-sectional design of this study which precludes the attribution of the differences observed to training *per se*. While the trained girls were most certainly "trained" (i.e. 12 hours of swim training per week for 5 years), the observed differences in their responses might also reflect genetic traits which predisposed these adolescents to success in swimming.

Ramp Incremental Exercise

The influence of training status was clearly evident in the incremental ramp test responses, with the trained girls having a significantly higher peak $\dot{V}O_2$ and work rate during both cycle and upper body ergometry. Although an influence of training status on peak cycle ergometry responses is not a novel finding (Mahon and Vaccaro 1989; Rowland et al. 1991), the current results extend these findings to a previously unconsidered exercise modality (upper body ergometry). A higher peak $\dot{V}O_2$ in trained children is largely attributable to an enhanced peak cardiac output consequent solely to an enhanced stroke volume as peak heart rates do not differ. The mechanistic basis of the higher peak stroke volume in trained children remains equivocal, with evidence to suggest both morphological (Ayabakan et al. 2006; Nottin et al. 2004; Obert et al. 2009; Rowland et al. 2009) and/or functional (Rowland et al. 1998) differences according to training status. In the current study, the trained swimmers' peak $\dot{V}O_2$ was 21% and 28% higher than the untrained girls during cycle and upper body ergometry, respectively. This difference is considerably larger than the 7-12% difference found in other training studies in adolescents (Mahon and Vaccaro 1989; Rowland et al. 1991). This difference likely arises due to the longitudinal nature of the previous studies and, consequently, the considerably shorter training history of the trained populations under investigation. Support for this conclusion is provided by the only other cross-sectional study in pubertal populations which reported a 17%

difference in the peak $\dot{V}O_2$ between footballers and untrained adolescents during cycle ergometry (Marwood et al. 2010).

The absence of a training status effect on the GET, either in absolute terms or as a fraction of peak $\dot{V}O_2$, contrasts with previous findings in adolescents (Marwood et al. 2010) and adults (Boone et al. 2008; Simon et al. 1986) but agrees with reports in pre-pubertal children (Cleuziou et al. 2002; Obert et al. 2000; Winlove et al. 2010). The explanation for the discrepancy is currently unclear, although it could be related to the lower glycolytic activity reported in children and adolescents (Kuno et al. 1995; Taylor et al. 1997; Zanconato et al. 1993). When expressed in absolute values, the GET was higher during cycle than upper body ergometry, in agreement with previous paediatric (Winlove et al. 2010) and adult studies (Davis et al. 1976; Koga et al. 1996; Schneider et al. 2002). However, the influence of exercise modality was removed when the GET was expressed as a fraction of peak $\dot{V}O_2$, as has been previously reported (Koga et al. 1996; Schneider et al. 2002; Winlove et al. 2010).

Constant-work-rate tests: influence of training status

Training status significantly affected the primary component $\dot{V}O_2$ kinetics during cycle ergometry, in agreement with previous work in both adolescents (Marwood et al. 2010) and adults (Koppo et al. 2004; Figueira et al. 2008; Powers et al. 1985). Although this significant influence on the cycle ergometry response is in contrast to previous findings in pre-pubertal children (Cleuziou et al. 2002; Obert et al. 2000; Winlove et al. 2010), the significant influence of training status on the upper body $\dot{V}O_2$ primary component response is in agreement with the pre-pubertal literature (Winlove et al. 2010). No studies are available in adolescents or adults for comparison to the current upper body ergometer results.

The shorter $\dot{V}O_2$ τ in the trained swimmers may be related to an enhanced delivery and/or fractional extraction of O_2. The faster HR τ in trained participants is in agreement with previous research in both adolescent (Marwood et al. 2010) and pre-pubertal (Winlove et al. 2010) populations, although this is the first study to investigate the HR kinetics during heavy intensity cycle and upper body ergometry in adolescents. If HR kinetics are accepted to provide a crude estimate of muscle blood

flow kinetics, as suggested during knee extension exercise (MacPhee et al. 2005), these results suggest that bulk O_2 delivery to the muscle was enhanced in the trained state. This would be consistent with previous studies conducted in adult populations reporting faster conduit artery blood flow kinetics and greater vascular conductance following training (Krustrup et al. 2004; Shoemaker et al. 1996). However, it is important to note that an increased bulk O_2 delivery in the trained state does not necessarily imply that O_2 availability was limiting in the untrained state. Indeed such a suggestion seems unlikely given the evidence available in young adults (DeLorey et al. 2004; Jones et al. 2006; Wilkerson et al. 2006).

Alternatively, the smaller phase II τ may be related to the influence of training status on muscle fractional O_2 extraction (as reflected by the [HHb] response). Specifically, the [HHb] τ and MRT were significantly shorter in trained girls, although the TD was unaffected by training status. Whilst the latter finding is in agreement with previous reports, the faster τ and MRT observed in the present study is in contrast to the study of Marwood et al (2010) in which the τ and MRT were reported to be unaffected by training status. The [HHb] response is generally accepted to reflect fractional O_2 extraction at the muscle (DeLorey et al. 2003; Ferreira et al. 2007; Grassi et al. 2003); therefore the shorter [HHb] τ in the trained swimmers indicates a more rapid increase in O_2 extraction towards the new steady-state. In adults, a training-induced reduction of the [HHb] τ (e.g., Bailey et al. 2009) has been attributed to an enhanced muscle oxidative capacity consequent to an increased mitochondrial volume and oxidative enzyme activity (Holloszy 1967; Mogensen et al. 2006). Although an increased muscle oxidative capacity has been reported in trained children (Eriksson et al. 1973; Fournier et al. 1982), there is insufficient information available regarding the effects of training on muscle fibre type and oxidative capacities in children and adolescents to allow conclusions to be drawn as to the mechanisms responsible for the enhanced O_2 extraction kinetics in the trained girls. Therefore, although the current results do not permit the complete elucidation of the factors limiting $\dot{V}O_2$ kinetics in adolescents, it is likely that the faster $\dot{V}O_2$ kinetics in the trained girls are a function of both a faster O_2 delivery and greater O_2 extraction, as similarly concluded in adults (Jones and Koppo 2005; Poole et al. 2008; McKay et al., 2009).

The influence of training status was isolated to the primary component, with no influence evident on the amplitude of the slow component during either exercise modality whether expressed in absolute or relative terms. The absence of a training status influence is in agreement with studies in pre-pubertal children (Cleuziou et al. 2002; Obert et al. 2000; Winlove et al. 2010) but contrasts with the reduction typically seen with training in adults (e.g. Bailey et al. 2009; Koppo et al. 2004; Powers et al. 1985). The explanation for this seemingly age-related influence of training status on the slow component is not readily apparent. However, in light of the putative association between the slow component and muscle fibre type recruitment (Poole et al.1994; Whipp, 1994), future investigations into the basis for this age-related effect may benefit from the inclusion of techniques (EMG, MRI) to assess differences in muscle activity between trained and untrained populations.

Constant-work-rate tests: influence of exercise modality

The influence of exercise modality on the $\dot{V}O_2$, HR and [HHb] kinetics has not previously been examined in a pubertal population. One of the most striking features of this study was the lack of exercise modality effect on the $\dot{V}O_2$ τ, a finding which contrasts with the longer phase II τ reported during upper compared to lower body exercise in adults (Koppo et al. 2002; Schneider et al. 2002). Typically, a longer upper body $\dot{V}O_2$ τ is attributed to a lower percentage of type I fibres in the upper body musculature (Gollnick et al. 1972; Johnson et al. 1973; Turner et al. 1997) in combination with a decreased perfusion pressure due to a reduced "gravitational assist" (Koga et al. 1999; Koppo and Bouckaert 2005). Since there is no evidence to suggest that either of these factors would be different in adolescents compared to adults, the explanation for this discrepancy is unclear. Exercise modality did not influence the [HHb] or HR τ values, in contrast to the longer HR τ reported in adults during upper compared to lower body exercise (Koga et al. 1996; Schneider et al. 2002). An explanation for this difference between pubertal and adult populations is not apparent from the current results. Exercise modality did, however, influence the [HHb] TD which was significantly shorter during upper body ergometry, indicating a reduced period of matching of O_2 delivery and utilisation.

In contrast to our previous study in pre-pubertal children (Winlove et al. 2010), there was no interaction between exercise modality and training status in the current study, with the influence of training status evident during both exercise modalities. Although conclusions are limited by the cross-sectional nature of these studies, it is possible that this difference between pre-pubertal and pubertal girls is attributable to the presence of a maturational threshold (Katch 1983) and/or the longer training history and greater training volume reported by the pubertal girls. This greater training load may have satisfied the minimum threshold stimulus required to elicit significant alterations in the $\dot{V}O_2$ kinetic response to both lower and upper body ergometry (Bailey et al. 2009; Berger et al. 2006). In contrast, the lower training load of the pre-pubertal children may have only surpassed the threshold stimulus required for upper body exercise, a threshold which is likely to be reduced relative to the respective lower body threshold due to differences in habitual activity.

In conclusion, training status significantly influenced the physiological responses of pubertal girls to both ramp incremental and constant-work-rate tests in two exercise modalities. Specifically, the $\dot{V}O_2$ kinetics of the trained girls was significantly faster than the untrained girls during both upper and lower body exercise. The faster HR and [HHb] kinetics in the trained girls may indicate that both central and peripheral factors are influenced by training status and contribute to the shorter $\dot{V}O_2$ τ in the trained state. These results contrast with the minimal influences generally observed in pre-pubertal populations, a difference likely attributable to both a greater stage of maturation and a longer training history in adolescents. Unlike pre-pubertal populations, the specificity of the exercise test modality to the training modality is not crucial in revealing the influence of training status in adolescents.

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Table 1. Physical characteristics of participants

	Trained	**Untrained**
Age (y)	14.2 ± 0.7	14.5 ± 1.3
Stature (m)	1.66 ± 0.04	1.61 ± 0.06
Mass (kg)	54.0 ± 5.1	58.7 ± 12.1
Sum of skinfolds (mm)	34.0 ± 10.7	48.7 ± 23.3
Estimated years past PHV (y)	2.0 ± 0.4	2.4 ± 0.6

Values are mean ± SD. PHV, peak height velocity. 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 $\dot{V}O_2$ ($L \cdot min^{-1}$)	2.51 ± 0.27	1.98 ± 0.26 *	1.88 ± 0.26 #	1.36 ± 0.21 *#
Peak $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	46.6 ± 5.0	34.5 ± 2.2 *	35.0 ± 6.0 #	24.0 ± 4.9 *#
Peak HR ($b \cdot min^{-1}$)	194 ± 5	196 ± 6	186 ± 10	180 ± 15
Peak blood [lactate] (mM)	7.8 ± 1.5	6.5 ± 2.1	5.1 ± 1.8	5.4 ± 2.1
Peak WR (W)	227 ± 23	180 ± 26 *	99 ± 9 #	71 ± 13 *#
GET ($L \cdot min^{-1}$)	1.51 ± 0.23	1.12 ± 0.26	1.00 ± 0.29 #	0.69 ± 0.29 #
GET (% peak $\dot{V}O_2$)	60 ± 5	62 ± 4	53 ± 10	56 ± 12

Values are mean ± SD. $\dot{V}O_2$, oxygen uptake; HR, heart rate; blood [lactate], blood lactate concentration; WR, work rate; GET, gas exchange threshold. N = 8

* *Significant difference between trained and untrained children within an exercise modality (P < 0.01)*

\# *Significant difference between modes within trained or 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 $\dot{V}O_2$ ($L \cdot min^{-1}$)	0.58 ± 0.09	0.64 ± 0.10	0.36 ± 0.05 #	0.42 ± 0.07 #
Phase II time delay (s)	12 ± 3	10 ± 4	13 ± 4	9 ± 6
Phase II τ (s)	21 ± 6	35 ± 11 *	29 ± 8	44 ± 8 *
95% confidence interval (s)	2 ± 0	3 ± 0	2 ± 0	4 ± 0
Phase II amplitude ($L \cdot min^{-1}$)	1.33 ± 0.24	0.92 ± 0.22 *	0.86 ± 0.13 #	0.50 ± 0.15 *#
Phase II gain ($mLO_2 \cdot min^{-1} \cdot W^{-1}$)	9.3 ± 0.7	8.2 ± 1.0	13.1 ± 1.3 #	12.1 ± 1.9 #
Slow component amplitude ($L \cdot min^{-1}$)	0.12 ± 0.10	0.14 ± 0.10	0.07 ± 0.05	0.08 ± 0.06 #
Slow component amplitude (% end)	8 ± 4	14 ± 8	7 ± 4	13 ± 10
End-exercise $\dot{V}O_2$ ($L \cdot min^{-1}$)	2.03 ± 0.29	1.71 ± 0.26	1.29 ± 0.15 #	1.00 ± 0.20 *#
Blood [lactate] (mM)	3.5 ± 0.5	5.6 ± 1.0 *	2.9 ± 0.7	4.0 ± 1.0

Values are mean ± SD. $\dot{V}O_2$, oxygen uptake; τ, time constant; blood [lactate], blood lactate concentration. N = 8

** Significant difference between trained and untrained children within an exercise modality (P < 0.01)*

 # Significant difference between modes within trained or untrained children (P < 0.01)

Table 4. Deoxyhemoglobin/myoglobin 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
[HHb] TD (s)	6 ± 2	7 ± 2	3 ± 1 #	3 ± 2 #
[HHb] τ (s)	12 ± 2	20 ± 6 *	13 ± 3	21 ± 7 *
95% confidence interval (s)	3 ± 0	2 ± 1	2 ± 1	2 ± 1
[HHb] MRT (s)	17 ± 2	27 ± 8 *	16 ± 3	24 ± 8 *
[HHb] Amplitude (AU)	71 ± 57	102 ± 59	166 ± 108 #	200 ± 114 #

Values are mean ± SD. [HHb], deoxyheamoglobin/myoblobin; τ, time constant; MRT, mean response time. N = 8

* *Significant difference between trained and untrained children within an exercise modality (P < 0.01)*

\# *Significant difference between modes within trained or untrained children (P < 0.01)*

Table 5. 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 ($b \cdot min^{-1}$)	93 ± 8	114 ± 10 *	87 ± 6	99 ± 7 *#
HR τ (s)	36 ± 5	53 ± 9 *	32 ± 3	43 ± 2 *
95% confidence interval (s)	1 ± 0	2 ± 1	1 ± 1	2 ± 1
HR amplitude ($b \cdot min^{-1}$)	65 ± 9	58 ± 6	58 ± 9	37 ± 17 *#
End-exercise HR ($b \cdot min^{-1}$)	170 ± 13	182 ± 10	159 ± 16	147 ± 17 #

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

* *Significant difference between trained and untrained children within an exercise modality (P < 0.01)*

\# *Significant difference between modes within trained or untrained children (P < 0.01)*

Figure legends

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. Panel A shows the response during cycle ergometer exercise and panel B the response to upper body ergometer exercise. The data are expressed as a percentage of the end exercise amplitude. The trained girl's data are shown as closed circles and the untrained girl's data are shown as open circles. The solid and dashed lines represent the mono-exponential model fit to the data. Note the faster τ in the trained participant during both exercise modes. 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. Panel A shows the response during cycle ergometer exercise and panel B the response to upper body ergometer exercise. The data are expressed as a percentage of the end exercise amplitude. The trained girl's data are shown as closed circles and the untrained girl's data are shown as open circles. The solid and dashed lines represent the mono-exponential model fit to the data. Note the faster τ in the trained participant during both exercise modalities. For clarity, data are displayed as 5-s bin averages.

Fig. 3. Deoxyhemoglobin/myoblobin 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. Panel A shows the response during cycle ergometer exercise and panel B the response to upper body ergometer exercise. The data are expressed as a percentage of the primary phase amplitude. The trained girl's data are shown as closed circles and the untrained girl's data are shown as open circles. The solid and dashed lines represent the mono-exponential model fit to the data. Note the faster τ in the trained participant during both exercise modalities. For clarity, data are displayed as 5-s bin averages

Figure 1

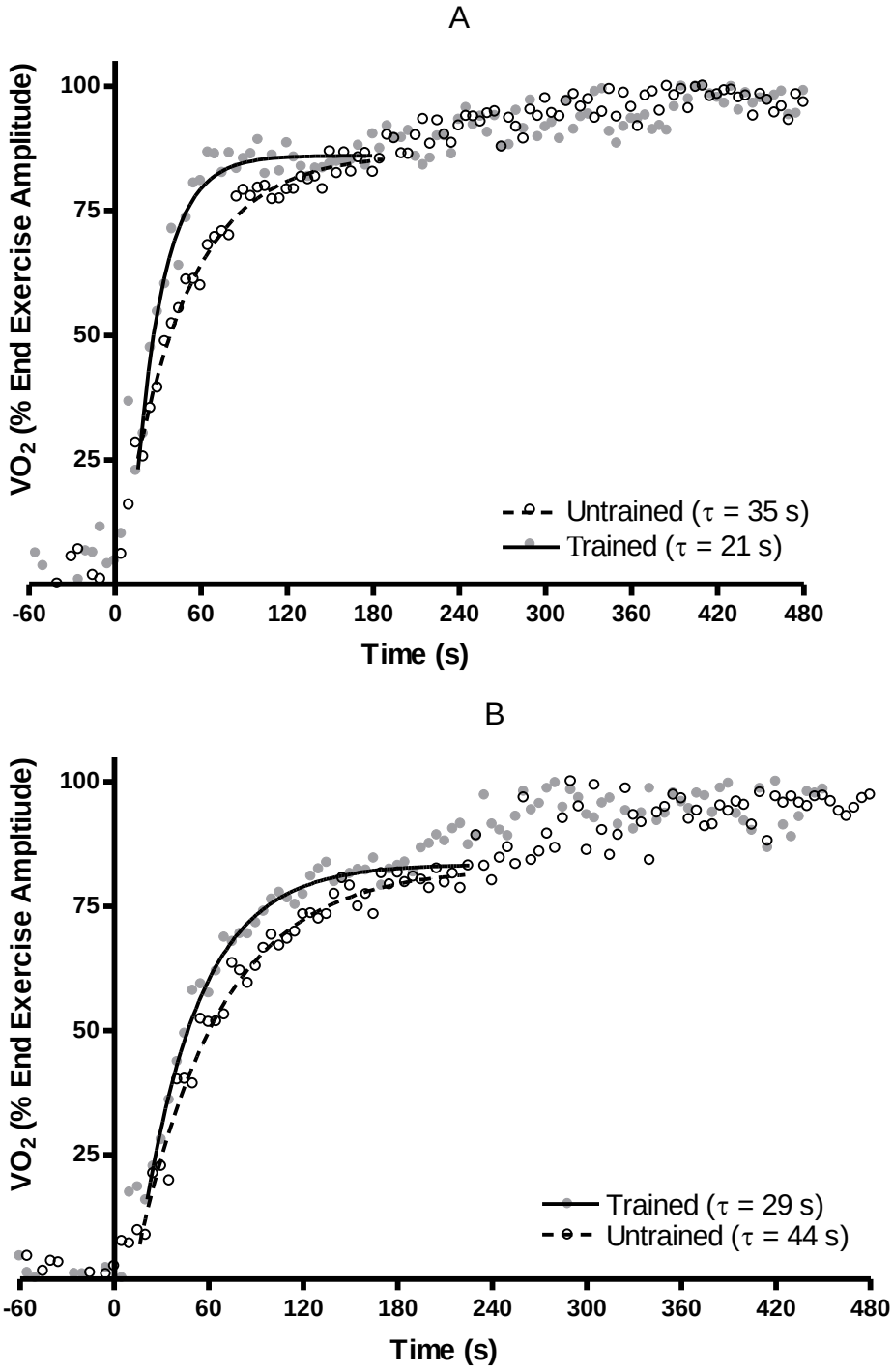

A
100
75
50
25
VO2 (% End Exercise Amplitude)
Untrained (τ = 35 s)
Trained (τ = 21 s)
-60
0
60
120
180
240
300
360
420
480
Time (s)
B
100
75
50
25
VO2 (% End Exercise Ampltiude)
Trained (τ = 29 s)
Untrained (τ = 44 s)
-60
0
60
120
180
240
300
360
420
480
Time (s)

Figure 2

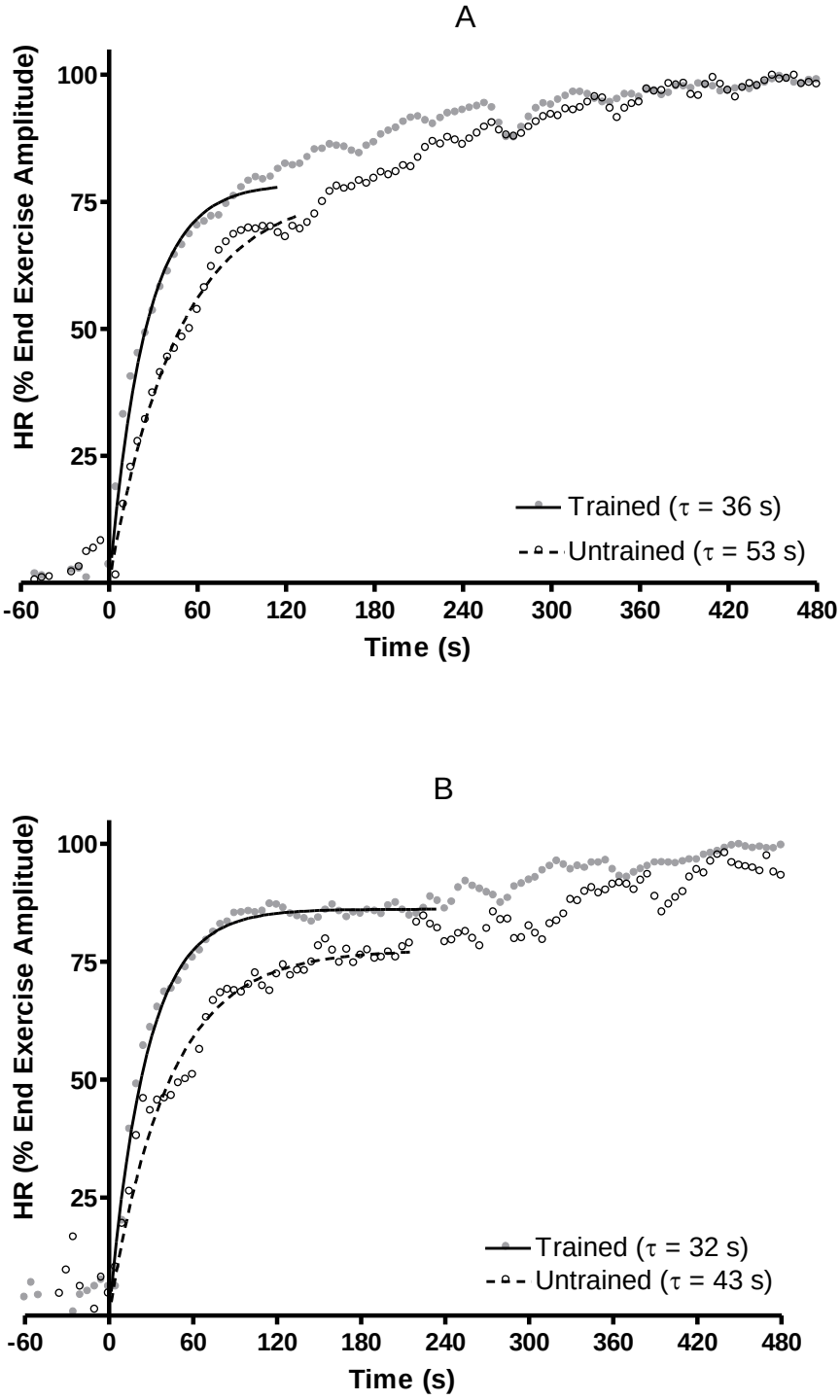
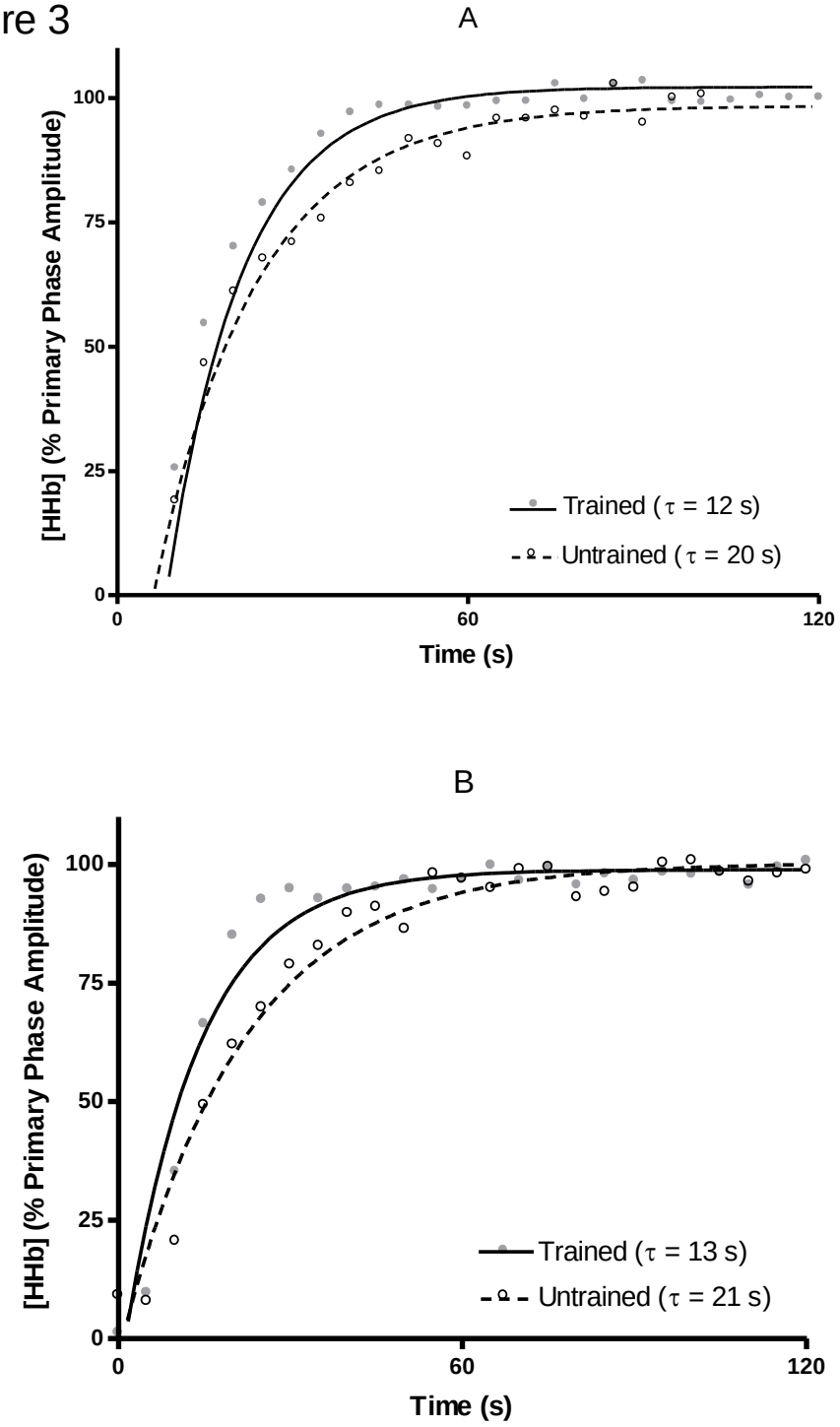

A
HR (% End Exercise Amplitude)
100
75
50
25
-60
0
60
120
180
240
300
360
420
480
Time (s)
Trained (τ = 36 s)
Untrained (τ = 53 s)
B
HR (% End Exercise Amplitude)
100
75
50
25
-60
0
60
120
180
240
300
360
420
480
Time (s)
Trained (τ = 32 s)
Untrained (τ = 43 s)

Figure 3


A
[HHb] (% Primary Phase Amplitude)
100
75
50
25
0
0
60
120
Time (s)
Trained (τ = 12 s)
Untrained (τ = 20 s)
B
[HHb] (% Primary Phase Amplitude)
100
75
50
25
0
0
60
120
Time (s)
Trained (τ = 13 s)
Untrained (τ = 21 s)