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Influence of training status and maturity on pulmonary O_2 uptake recovery kinetics following cycle and upper body exercise in girls

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Abstract

The influence of training status on pulmonary $\dot{V}O_2$ recovery kinetics, and its interaction with maturity, has not been investigated in young girls. Sixteen pre-pubertal (Pre: trained (T, 11.4±0.7 years), 8 untrained (UT, 11.5±0.6 years)) and sixteen pubertal (Pub: 8T, 14.2±0.7 years; 8 UT, 14.5±1.3 years) girls completed repeat transitions from heavy intensity exercise to a baseline of unloaded exercise, on both an upper and lower body ergometer. The $\dot{V}O_2$ recovery time constant was significantly shorter in the trained pre-pubertal and pubertal girls during both cycle (Pre: T, 26±4 vs. UT, 32±6; Pub: T, 28±2 vs. UT, 35±7 s; both $P<0.05$) and upper body exercise (Pre: T, 26±4 vs. UT, 35±6; Pub: T, 30±4 vs. UT, 42±3 s; both $P<0.05$). No interaction was evident between training status and maturity. These results demonstrate the sensitivity of $\dot{V}O_2$ recovery kinetics to training in young girls and challenge the notion of a "maturational threshold" in the influence of training status on the physiological responses to exercise and recovery.

Keywords: children, training, recovery, $\dot{V}O_2$, maturity, NIRS

Introduction

The parameters characterising the pulmonary $\dot{V}O_2$ response to a sudden change in metabolic demand provide an insight into the bioenergetic processes sustaining exercise performance (19, 44). These parameters, which provide a surrogate measure of muscle oxygen consumption kinetics (19, 25), have been extensively investigated in adults and, within the last decade, there has been increased research attention in pre-pubertal children and pubertal adolescents. By comparison, the $\dot{V}O_2$ recovery kinetics, which closely reflect muscle phosphocreatine (PCr) kinetics (3, 45), a marker of muscle oxidative capacity, have received less attention.

Characterisation of the $\dot{V}O_2$ recovery kinetics from heavy intensity exercise remains unclear. In adults, there are contradictory findings regarding the mono- or bi-exponentiality of the response profile (5, 7, 10, 42), as well as the relative speed of the recovery response compared to the on-response (5, 17, 42, 43). The extent to which pulmonary $\dot{V}O_2$ kinetics reflect muscle $\dot{V}O_2$ kinetics in recovery from exercise has also been challenged (25). Relatively little information is available on the $\dot{V}O_2$ recovery profile in young people; however, what does exist suggests a mono-exponential decline that is slower than the $\dot{V}O_2$ on-response in pre-pubertal children (3, 55) and male pubertal adolescents (26).

Endurance training has been shown to result in faster $\dot{V}O_2$ on-kinetics in pre-pubertal children (52), adolescents (32, 36) and adults (2, 5). However, whilst the $\dot{V}O_2$ off-kinetics of adults have been shown to be speeded by training (4, 13), a recent report suggests no influence of training status on the $\dot{V}O_2$ off-kinetics of adolescents (31). This discrepancy may be indicative of a maturational threshold which must be exceeded for training status influences to be manifest (19). Alternatively, it is possible that it may be a reflection of an insufficient training volume in the adolescents or of an inappropriate test modality. The latter is supported by recent findings regarding the influence of training status on the $\dot{V}O_2$ on-kinetics of pre-pubertal swimmers, where significant

effects were only evident during upper-body and not lower-body exercise (52), in accord with the predominantly upper body nature of swimming (40).

Marwood et al. (28) reported a dissociation in the influence of training status on the $\dot{V}O_2$ and deoxyhaemoglobin ([HHb]) off-kinetics, with only the latter being significantly faster in trained adolescents. The [HHb] signal from near-infrared spectroscopy (NIRS) reflects the balance between oxygen supply and oxygen utilisation within a specific, localised area of the microcirculation and myocytes of the muscle, thereby providing an index of fractional oxygen extraction. The authors interpreted these results to be indicative of slower blood flow recovery kinetics in trained participants. The consideration of heart rate (HR) kinetics, which have been reported to reflect cardiac output kinetics and thus possibly bulk oxygen delivery (29), may allow this possibility to be investigated further.

The purpose of the present cross-sectional study was to investigate the influence of training status and maturity on the $\dot{V}O_2$ recovery kinetics from heavy intensity exercise in girls. To ameliorate concerns regarding disparities between training and testing modalities, both the lower and upper body responses were investigated. We hypothesised that the influence of training status on the $\dot{V}O_2$ off-kinetics would reflect those found in the on-kinetics of these participants as reported previously (36, 52). Specifically, we hypothesised that both the trained pre-pubertal and pubertal girls would exhibit a faster $\dot{V}O_2$ recovery response during upper body exercise than their untrained counterparts but that only the trained pubertal girls would exhibit faster $\dot{V}O_2$ recovery kinetics during lower body exercise.

Methods

Participants and anthropometry

Sixteen pre-pubertal (8 trained, 8 untrained) and sixteen pubertal (8 trained, 8 untrained) girls participated in this study. The trained girls (T) were all competitive swimmers; the pre-pubertal girls

(Pre) trained 8 ± 2.5 hours/week and the pubertal girls (Pub) trained 12 ± 2 hours/week. The untrained girls (UT) comprised volunteers from local schools. These girls were all participants in previously published studies investigating the on-kinetics response to exercise (36, 52); the present study details an analysis of the off-transition from these exercise bouts.

Prior to the first test, an anthropometrical evaluation was performed for all participants. 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 an 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 recorded. Table 1 presents the participants' physical characteristics. Sexual maturity was assessed by self-report using the indices of pubic hair proposed by Tanner (48). Age to peak height velocity was also estimated to provide an additional indicator of physical maturity according to the equations of Mirwald *et al*. (39).

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.

Experimental procedures

On the first two of multiple visits to the laboratory, participants completed a ramp incremental test to volitional exhaustion on either the lower (cycle; Lode Excalibur, Netherlands) or upper (arm crank; Lode Angio, Netherlands) body ergometer for the determination of the exercise mode-specific peak $\dot{V}O_2$ and gas exchange threshold (GET). 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.

The ramp incremental test comprised a three minute warm-up of 'unloaded' pedalling or arm-cranking (equivalent to 10W at 70 rpm according to the manufacturer's guidelines) following which the resistance increased at a pre-determined rate to attain a test duration of 8-12 minutes. The ramp rate was 12 $W \cdot min^{-1}$ or 20 $W \cdot min^{-1}$ for pre-pubertal and pubertal girls, respectively, for cycle ergometry, and 5 $W \cdot min^{-1}$ or 10 $W \cdot min^{-1}$, respectively, for the upper body ergometry. 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 (4) 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).

On subsequent visits, participants completed step transitions from a heavy intensity work rate to either unloaded pedalling or arm-cranking on the upper or lower body ergometer, respectively, for the determination of $\dot{V}O_2$ recovery kinetics. 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) which was sustained for 8 minutes. At 8 minutes, the work rate returned to the unloaded baseline at which the participants pedalled or cranked for a further 6 minutes. When multiple tests were completed on the same day, at least 1 hour separated the tests and the tests were ordered such that the first test involved the smaller muscle mass (upper body), thereby resulting in a smaller metabolic perturbation and faster recovery. Repeat transitions were completed until the 95% confidence intervals associated with the phase II τ of the on response were <4.5 s, as recommended by Fawkner et al. (12). Given the lower breath-to-breath noise inherent to the $\dot{V}O_2$ off-kinetics relative to the $\dot{V}O_2$ on-kinetics (16), this principle

ensured acceptable confidence intervals were also associated with the recovery-phase τ (cycle, 2.9 ± 1.2 s; upper body, 2.7 ± 1.7 s). To achieve this, 3-10 exercise transitions were completed on each exercise modality.

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.

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 (9, 14, 18). The contribution of myoglobin to the NIRS signal is currently unresolved (33, 46). Therefore, the [HHb] signal described throughout this paper should be considered to refer to the combined concentration of both deoxygenated haemoglobin and myoglobin.

The skin over 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 between the greater trochanter and the lateral condyle of the tibia. The distance from the lateral condyle of the tibia was recorded to allow accurate replication in future tests. 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.

$\dot{V}O_2$ Recovery 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. To identify the most appropriate model fit describing the averaged $\dot{V}O_2$ recovery response, a mono-exponential model with a time delay (Eq. 1) was compared to a bi-exponential with a single, common time delay (Eq. 2) and their 95% confidence intervals determined using least-squares linear regression analysis (Graphpad Prism, Graphpad Software, San Diego, CA).

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

$$\Delta VO_{2(t)} = A_1 \cdot \left(1 - e^{-(t-\delta_1)/\tau_1}\right) + A_2 \cdot \left(1 - e^{-(t-\delta_1)/\tau_2}\right) \quad \text{(Eq. 2)}$$

where $\Delta\dot{V}O_2$ is the decrease in $\dot{V}O_2$ at time *t* below the baseline value (calculated as the mean $\dot{V}O_2$ from the last 45 s of loaded pedalling), and A, δ and τ are the primary component amplitude, time delay and time constant, respectively. The subscripts 1 and 2 denote the primary and slow components of the dynamic pulmonary $\dot{V}O_2$ response.

[HHb] & HR Recovery Kinetics Analysis

The recovery kinetics of [HHb] and HR 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.3). For both responses the fitting window started at $t = 0$.

$$\Delta HR_{(t)} = A_1 \cdot \left(1 - e^{-(t-\delta_1)/\tau_1}\right) \qquad \text{(Eq.3)}$$

where ΔHR is the decrease in heart rate at time t below the baseline (calculated as the mean heart rate from the last 45 s of loaded 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.

Statistical Analysis

Following identification of a normal distribution, homogenous variation and an absence of skewness or kurtosis, a three way ANOVA with repeated measures was used to analyze training status, exercise modality and exercise phase (on vs. off) effects. Subsequent independent or paired samples t-tests were employed as appropriate to identify the location of significant differences. For those parameters influenced by training status, the interaction between training status and sexual maturity was investigated using a factorial ANOVA. Standardised mean difference scores were calculated according to Cohen's *d* index (*d*). Pearson product-moment correlation coefficients were used to assess the strength of relationships between variables. The best fitting model to describe the $\dot{V}O_2$ recovery kinetics was determined on the basis of the R^2 values, the residual sum of squares, and the F value. All data are presented as means ± SD. Statistical significance was accepted if $P<0.05$.

Results

Influence of training status

The parameters derived from the monoexponential modelling revealed a significant influence of training status on the $\dot{V}O_2$ recovery kinetics for pre-pubertal and pubertal girls, as presented in Table 2

and illustrated in Figure 1 and 2. Specifically, during both exercise modalities the τ of the $\dot{V}O_2$ recovery phase was significantly shorter in the trained pre-pubertal (lower body: *d*, 1.2; upper body: *d*, 1.8) and pubertal (lower body: *d*, 1.2; upper body: *d*, 3.2) girls than their untrained counterparts. Additional influences of training status were evident in the end-recovery $\dot{V}O_2$, which was significantly lower in the trained pre-pubertal girls after both exercise modalities, and in the $\dot{V}O_2$ recovery amplitude, which was greater in the trained pubertal girls during both upper and lower body exercise than in their untrained equivalents (as a consequence of the higher absolute WR during exercise).

The temporal parameters of the HR and [HHb] recovery, summarised in Table 3 and illustrated in Figures 2 and 3, respectively, were not significantly influenced by training status during either exercise modality in the pre-pubertal girls or during upper body exercise in the pubertal girls. Following lower body exercise, the trained pubertal girls exhibited a significantly faster [HHb] τ and MRT but HR τ was not different. There was no correlation between the $\dot{V}O_2$ recovery τ and the HR recovery τ or [HHb] recovery τ.

Influence of maturity

With the exception of the $\dot{V}O_2$ recovery amplitude during both exercise modalities, the magnitude of training status differences was similar in both maturity groups and, consequently, no interactions were evident between training status differences and maturity for the majority of the $\dot{V}O_2$, HR and [HHb] parameters.

Influence of exercise phase

The monoexponential model was accepted as a superior description of the pulmonary $\dot{V}O_2$ recovery data based on higher R^2 values, higher residual sum of squares and an insignificant F-test for the higher order model for all participants during both exercise modalities. An asymmetry between the

on- and off- $\dot{V}O_2$ response profiles for heavy exercise was therefore evident since the on-kinetics derived from the same participants exhibited two phases (36, 52). No asymmetry was evident in the temporal description of the on- and off- transients however, with a similar $\dot{V}O_2$ τ_{on} and τ_{off}, irrespective of training status or maturity, as shown in Table 2. In contrast, both the HR and [HHb] kinetics were influenced by exercise phase during both modalities and regardless of maturity, with a faster response to the onset than cessation of exercise, as shown in Table 3. A significant correlation was present between the $\dot{V}O_2$ τ_{on} and $\dot{V}O_2$ τ_{off} irrespective of exercise modality in both pre-pubertal (lower body: r = 0.73, $P<0.01$; upper body r = 0.80, $P<0.01$) and pubertal girls (lower body: r = 0.67, $P<0.01$; upper body r = 0.59, $P<0.05$). In contrast, there was no significant correlation between the τ describing the on and off transitions for either HR or [HHb].

Discussion

This is the first study to investigate the influence of training status and maturity on the $\dot{V}O_2$ recovery dynamics of young girls. We hypothesised that the influence of training status on the $\dot{V}O_2$ off-kinetics would reflect those previously reported for the on-kinetics of these participants, i.e. that during upper body exercise both pre-pubertal and pubertal trained girls would exhibit faster $\dot{V}O_2$ kinetics than their untrained counterparts but that only pubertal trained girls would exhibit faster $\dot{V}O_2$ kinetics during lower body exercise (36, 52). However, this hypothesis was not supported as significant training status effects were evident during *both* exercise modalities in pre-pubertal and pubertal girls. This suggests that $\dot{V}O_2$ on- and off-kinetics may differ in their sensitivity to training status in young girls. Further novel findings were that the influence of training status was independent of maturity stage, thereby challenging the notion of a maturational threshold which must be surpassed for the influence of training status to become evident. Finally, the $\dot{V}O_2$ and HR recovery τ were dissociated at all stages of maturity and during both exercise modalities.

Prior to a discussion of the present findings it is apposite to acknowledge two limitations of the present study. Firstly, the methodologies employed were non-invasive, as necessitated by the ethical constraints associated with the population investigated, and consequently indirect. Secondly, the cross-sectional design precluded observed training status differences being attributable to training *per se* as they may also reflect genetic traits which predisposed these girls to success in swimming.

Influence of training status

The faster $\dot{V}O_2$ recovery τ found here in trained compared to untrained pre-pubertal and pubertal girls during both exercise modalities is in agreement with findings in adults (5, 16). However, the present findings contradict those recently reported in male adolescents (31). This discrepancy may be related to gender, training volume, exercise modality or exercise intensity. Gender has previously been suggested to influence the on-kinetics of pre-pubertal children (13); the applicability of such findings to pubertal adolescents, or the recovery response, is yet to be investigated. The lack of an influence of training status in the adolescents of Marwood et al. may be related to the lower training volume completed by their participants (6 $h \cdot wk^{-1}$ vs 12 $h \cdot wk^{-1}$) or to a disparity between training and testing modalities (runners completed cycle ergometry testing). However, the latter is perhaps unlikely as these modalities demonstrate a commonality in the muscles used, similar to those implemented in the present study where training status influences were demonstrated. The interaction between exercise intensity and the demonstration of training status effects has not been investigated in young people, but a significant interaction has been reported in adults: higher intensity exercise being associated with greater influences of training status (2, 6, 24). Thus, it is possible that the disparity in the influence of training status observed in the present study and that of Marwood et al. is attributable to the use of heavy and moderate intensity constant work-rate exercise, respectively.

The present findings in pre-pubertal girls that the influence of training status on $\dot{V}O_2$ recovery kinetics is not modality specific contrasts the findings in the on-kinetics of the same participants, where a significant influence of training status was only evident during upper body ergometry (52). The

explanation for this asymmetry between exercise phases is presently unclear but may be indicative of a dichotomy in the sensitivity of the on- and off-transients to the influence of training status.

In the recovery from exercise, pulmonary $\dot{V}O_2$ kinetics has been reported to closely reflect muscle PCr kinetics (3, 45), which are considered to reflect the rate of mitochondrial respiration and to provide an index of skeletal muscle oxidative capacity (1, 22). The relationship between PCr recovery kinetics and oxidative capacity has been supported by findings of a faster PCr recovery τ in endurance trained adults (28, 53), who are typically characterised by an increased muscle mitochondrial content and oxidative enzyme activity (20, 34). An increased mitochondrial volume following endurance training would be anticipated to result in faster $\dot{V}O_2$ kinetics (37), associated with an improved matching of perfusion to demand and/or to a reduced contribution of substrate level phosphorylation to energy turnover. Resolution is still required as to whether similar adaptations in muscle oxidative capacity are present in children and/or adolescents (30). However, the limited information available suggests that training may increase muscle oxidative enzyme activity in children (11, 15). Thus, the faster $\dot{V}O_2$ recovery time constant in the present trained participants may be attributable to a greater muscle oxidative capacity relative to their untrained counterparts. This is supported by the trend for a faster [HHb] recovery τ in the trained girls at both stages of maturity and during both exercise modalities. Alternatively, the faster $\dot{V}O_2$ recovery in the trained girls may be associated with an enhanced O_2 delivery (27, 47). However, the lack of training status effect on the HR recovery τ, which may provide a crude estimate of muscle blood flow kinetics (29), and the absence of any correlation between the HR and $\dot{V}O_2$ recovery τ do not support this interpretation.

Influence of maturity

This is the first study to investigate the interaction between training status and maturity during the recovery phase from a step-transition in metabolic demand. It has been suggested that the influence of training status increases with maturity (21). However, we found no interaction between training status

and maturity for either exercise modality. In agreement with other studies (8, 35, 50), these findings contradict the notion of a maturational threshold below which significant physiological adaptations to training cannot occur. Studies purporting to support the concept of a maturational threshold may rather be a reflection of a lower (and perhaps insufficient) training volume in the younger participants (23, 38, 41). However, it should be acknowledged that the importance of sex or sport in determining the presence, or absence, of a maturational threshold remains to be investigated. For example, the influence of the hormones associated with the onset of puberty (49, 54) may be more important in boys or in more "anaerobic" sports. Nevertheless, the growing evidence against the presence of a maturational threshold has considerable practical implications since it suggests that training may be equally beneficial before and during puberty as it is after puberty.

Influence of exercise or recovery phase

Characterisation of the pulmonary $\dot{V}O_2$ responses to a step transition to and from heavy intensity exercise revealed asymmetrical response profiles regardless of participant group or exercise modality. Specifically, the $\dot{V}O_2$ response to the on-transition evidenced both a primary and slow phase (36, 52), whereas the recovery response exhibited a mono-exponential decline to end-recovery values. This mono-exponential decline agrees with previous studies in pre-pubertal children (3, 55), pubertal adolescents (26) and adults (5, 42, 43) during lower body exercise, but this is the first study to report upon the on-off response symmetry during upper body exercise. The different behaviour of pulmonary $\dot{V}O_2$ at the onset of exercise and recovery may be partly attributable to the relationship between the dynamics of $\dot{V}O_2$ and phosphocreatine breakdown-synthesis (26, 42).

Whilst beyond the scope of the present study, it is interesting to note the implications of the presence of a $\dot{V}O_2$ slow component only at the onset of, and not recovery from, heavy intensity exercise with regard to the mechanistic basis for this additional, slowly developing phase (7, 42). These implications include the likelihood that the $\dot{V}O_2$ slow component development during exercise is consequent to the

'progressive' recruitment of additional (type II) muscle fibres as exercise proceeds (51), and is not strongly related to additional O_2 costs associated with increased muscle temperature or additional cardiac or respiratory work (42).

The present findings regarding a similar $\dot{V}O_2$ τ describing the $\dot{V}O_2$ on- and off-kinetics are in contrast with those previously reported in pre-pubertal children (3) and adolescents (26). Interestingly however, they are in accord with the similar PCr on and off-kinetics recently reported in pre-pubertal children (3). This temporal symmetry suggests a similar mechanism is responsible for regulating both the on- and off-responses.

Conclusion

This is the first study to investigate the influence of training status on the recovery kinetics of pulmonary $\dot{V}O_2$, HR and [HHb] in young girls. The $\dot{V}O_2$ recovery kinetics were significantly faster in the trained pre-pubertal and pubertal girls relative to their untrained counterparts, irrespective of exercise modality. This finding contrasts the exercise modality-specific influence reported in pre-pubertal girls during the on transient, indicating that the recovery phase kinetics may be more sensitive to training status influences. The magnitude of training status differences were not modulated by maturity, challenging the concept of a maturational threshold which must be surpassed for training status effects to be evident.

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Table 1. Participants' anthropometric characteristics

	Pre-pubertal girls		Pubertal girls	
	Trained	Untrained	Trained	Untrained
Age (y)	11.4 ± 0.7	11.5 ± 0.6	14.2 ± 0.7	14.5 ± 1.3
Stature (m)	1.48 ± 0.06	1.52 ± 0.05	1.66 ± 0.04	1.61 ± 0.06
Mass (kg)	39.9 ± 6.9	43.2 ± 8.6	54.0 ± 5.1	58.7 ± 12.1
Sum of skinfolds (mm)	36.4 ± 14.6	43.8 ± 25.6	34.0 ± 10.7	48.7 ± 23.3
Years to/past PHV (y)	-0.46 ± 0.5	-0.31 ± 0.45	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. Monoexponential model derived parameters describing the pulmonary oxygen uptake off-kinetics following heavy-intensity exercise on a cycle and upper body ergometer in trained and untrained pre-pubertal and pubertal girls

	Pre-pubertal girls		Pubertal girls	
	Trained	Untrained	Trained	Untrained
		Lower body		
$\dot{V}O_2$ TD (s)	9 ± 1	8 ± 3	10 ± 4	9 ± 3
$\dot{V}O_2$ τ_{off} (s)	26 ± 4	32 ± 6 *	28 ± 2	35 ± 7 *
$\dot{V}O_2$ Amp ($l \cdot min^{-1}$)	0.88 ± 0.16	0.72 ± 0.15	1.43 ± 0.31	1.00 ± 0.23 *
		Upper body		
$\dot{V}O_2$ TD (s)	10 ± 3	8 ± 3	9 ± 4	8 ± 3
$\dot{V}O_2$ τ_{off} (s)	26 ± 4	35 ± 6 *	30 ± 4	42 ± 3 *#
$\dot{V}O_2$ Amp ($l \cdot min^{-1}$)	0.51 ± 0.09 #	0.56 ± 0.19 #	0.93 ± 0.15 #	0.66 ± 0.29 *#

Values are mean ± SD. $\dot{V}O_2$, oxygen uptake; τ, time constant; Amp, amplitude; ER, end recovery. N = 8.

* *Significant difference between trained and untrained girls within a pubertal stage (P<0.05)*

\# *Significant difference between exercise modalities within training and pubertal group (P<0.05)*

Table 3. Monoexponential model derived parameters describing the heart rate and deoxyhaemoglobin/myoglobin recovery kinetics from heavy-intensity exercise on a cycle and upper body ergometer in trained and untrained pre-pubertal and pubertal girls

	Pre-pubertal girls		Pubertal girls	
	Trained	Untrained	Trained	Untrained
		Lower body		
HR τ_{off} (s)	52 ± 13	64 ± 13	67 ± 16	67 ± 11
HR Amp ($b \cdot min^{-1}$)	59 ± 6	54 ± 6	63 ± 11	56 ± 6
ER HR ($b \cdot min^{-1}$)	108 ± 9	125 ± 7 *	110 ± 3	124 ± 12 *
[HHb] TD (s)	3.2 ± 1.8	6.0 ± 3.1	3.9 ± 3.8	1.6 ± 3.5
[HHb] τ_{off} (s)	29 ± 10	41 ± 10	24 ± 10	50 ± 7 *
[HHb] MRT (s)	32 ± 9	47 ± 13 *	28 ± 10	51 ± 7 *
		Upper body		
HR τ_{off} (s)	35 ± 14 #	41 ± 13 #	36 ± 13 #	34 ± 16 #
HR Amp ($b \cdot min^{-1}$)	54 ± 9	48 ± 11 #	62 ± 11	42 ± 14 *#
ER HR ($b \cdot min^{-1}$)	98 ± 8 #	107 ± 8 *#	92 ± 8 #	105 ± 9 *#
[HHb] TD (s)	1.1 ± 3.8 #	5.9 ± 8	4.5 ± 2.8	4.6 ± 7.8
[HHb] τ_{off} (s)	34 ± 13	40 ± 12	31 ± 17	38 ± 15
[HHb] MRT (s)	33 ± 12	47 ± 17	36 ± 17	43 ± 14

Values are mean ± SD. HR, heart rate; [HHb], deoxyhaemoglobin; τ, time constant; Amp, amplitude; ER, end recovery; MRT, mean response time. N = 8.

* *Significant difference between trained and untrained girls within a pubertal stage (P<0.05)*

\# *Significant difference between exercise modalities within training and pubertal group (P<0.05)*

Table 4. Monoexponential model derived parameters describing the oxygen uptake, heart rate and deoxyhaemoglobin/myoglobin kinetics at the onset of heavy-intensity exercise on a cycle and upper body ergometer in trained and untrained pre-pubertal and pubertal girls

	Pre-pubertal girls		Pubertal girls	
	Trained	Untrained	Trained	Untrained
		Lower body		
$\dot{V}O_2$ τ_{on} (s)	25 ± 5	25 ± 7	21 ± 6	35 ± 11*
HR τ_{on} (s)	31 ± 11	47 ± 9*	36 ± 5	53 ± 9*
[HHb] τ_{on} (s)	16 ± 4	24 ± 10	12 ± 2	20 ± 6*
		Upper body		
$\dot{V}O_2$ τ_{on} (s)	25 ± 3	37 ± 6*	29 ± 8	44 ± 8*
HR τ_{on} (s)	16 ± 4	24 ± 12*	12 ± 2	20 ± 6*
[HHb] τ_{on} (s)	14 ± 3	12 ± 3	13 ± 3	21 ± 7*

Data is from previously published studies (36, 52) in which the same girls were also participants. Values are mean ± SD. $\dot{V}O_2$, oxygen uptake; HR, heart rate; [HHb], deoxyhaemoglobin; τ, time constant; N = 8.

* *Significant difference between trained and untrained girls within a pubertal stage (P<0.05)*

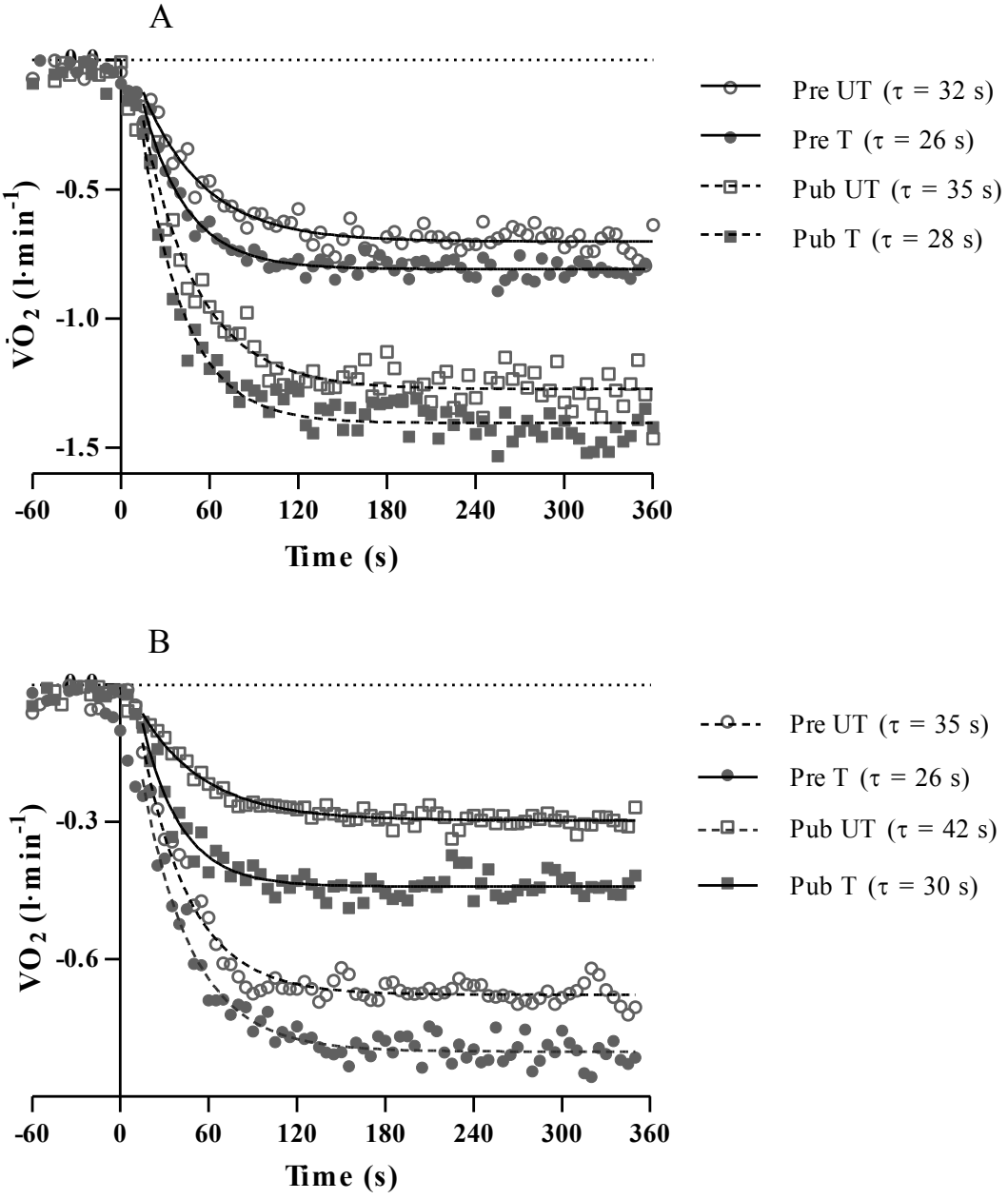
\# *Significant difference between exercise modalities within training and pubertal group (P<0.05)*

Figure Legends

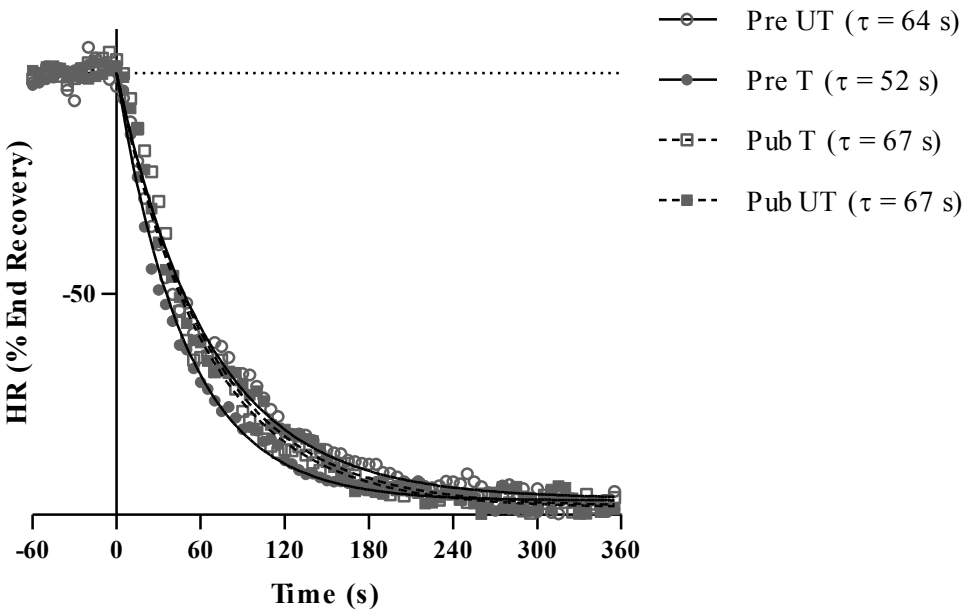
Fig. 1. Pulmonary oxygen uptake response to a step decrement in work rate from a heavy intensity work rate (40%Δ) to an unloaded baseline in a representative trained (closed symbols) and untrained (open symbols) pre-pubertal (circles) and pubertal (squares) participant during lower (A) and upper body (B) exercise. For clarity, data are displayed as 5-s bin averages

Fig. 2. Heart rate response to a step decrement in work rate from a heavy intensity work rate (40%Δ) to an unloaded baseline in a representative trained (closed symbols) and untrained (open symbols) pre-pubertal (circles) and pubertal (squares) participant during lower (A) and upper body (B) exercise. For clarity, data are displayed as 5-s bin averages

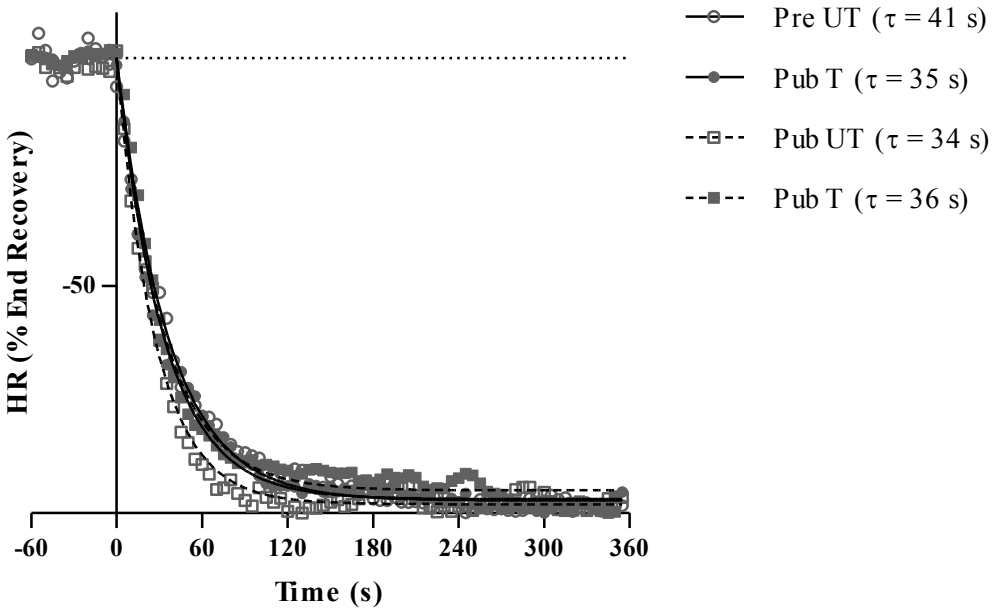
Fig. 3. Deoxyhaemoglobin ([HHb]) response to a step decrement in work rate from a heavy intensity work rate (40%Δ) to an unloaded baseline in a representative trained (closed symbols) and untrained (open symbols) pre-pubertal (circles) and pubertal (squares) participant during lower (A) and upper body (B) exercise. For clarity, data are displayed as 5-s bin averages

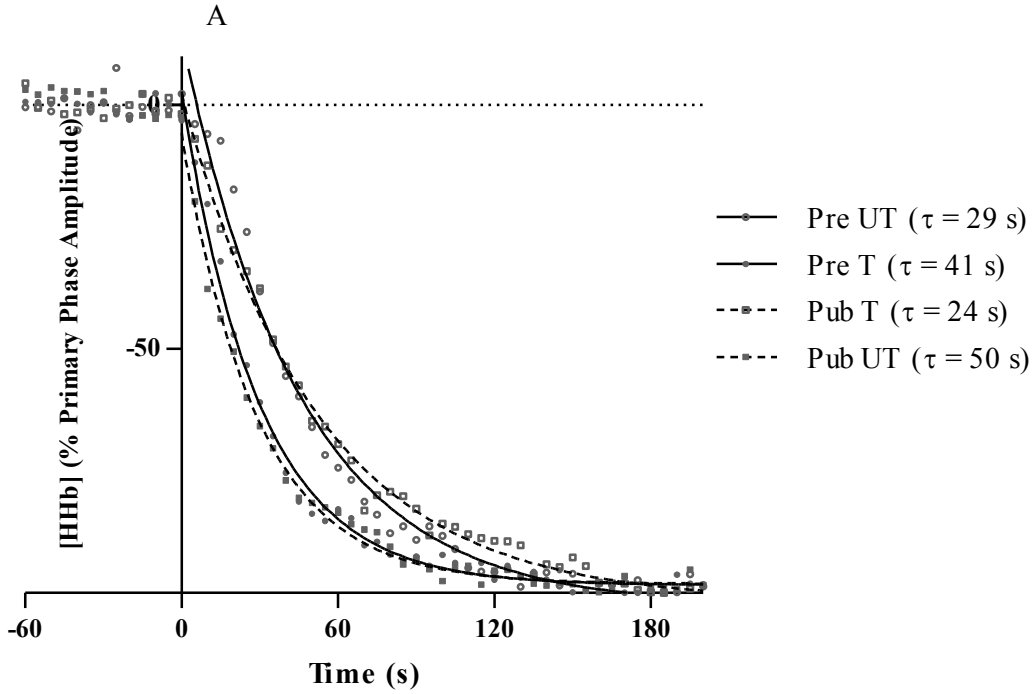

A
0.0
-0.5
-1.0
-1.5
$\dot{V}O_2$ (l·min^{-1})
-60
0
60
120
180
240
300
360
Time (s)
Pre UT (τ = 32 s)
Pre T (τ = 26 s)
Pub UT (τ = 35 s)
Pub T (τ = 28 s)
B
0.0
-0.3
-0.6
VO_2 (l·min^{-1})
-60
0
60
120
180
240
300
360
Time (s)
Pre UT (τ = 35 s)
Pre T (τ = 26 s)
Pub UT (τ = 42 s)
Pub T (τ = 30 s)

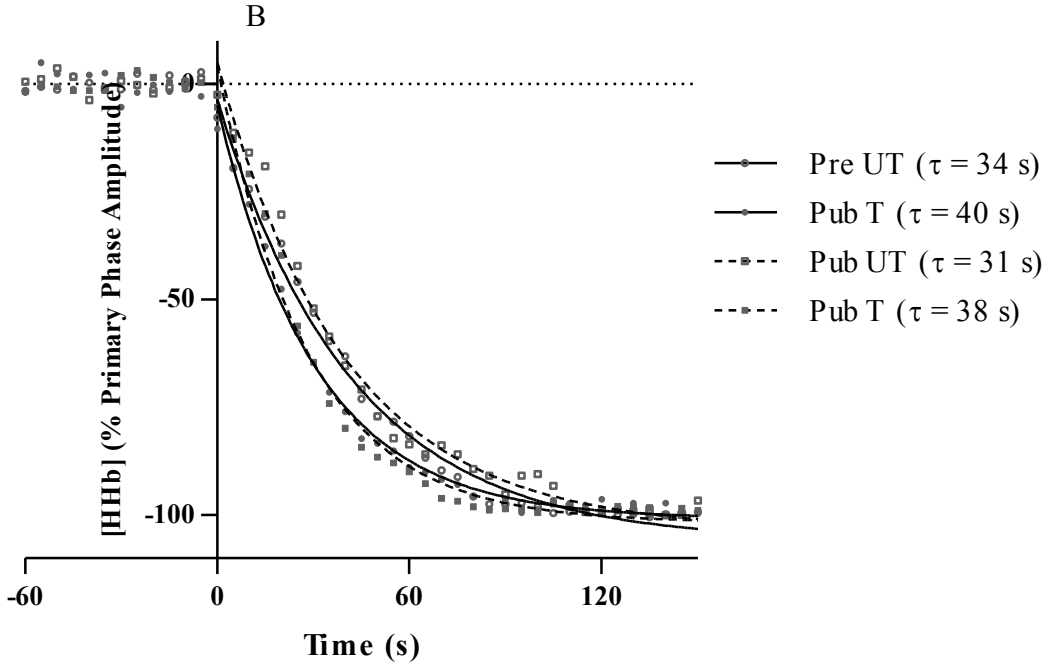
A


Pre UT (τ = 64 s)
Pre T (τ = 52 s)
Pub T (τ = 67 s)
Pub UT (τ = 67 s)
HR (% End Recovery)
-50
-60
0
60
120
180
240
300
360
Time (s)

B


Pre UT (τ = 41 s)
Pub T (τ = 35 s)
Pub UT (τ = 34 s)
Pub T (τ = 36 s)
HR (% End Recovery)
-50
-60
0
60
120
180
240
300
360
Time (s)


A
[HHb] (% Primary Phase Amplitude)
0
-50
-60
0
60
120
180
Time (s)
Pre UT (τ = 29 s)
Pre T (τ = 41 s)
Pub T (τ = 24 s)
Pub UT (τ = 50 s)


B
[HHb] (% Primary Phase Amplitude)
0
-50
-100
-60
0
60
120
Time (s)
Pre UT (τ = 34 s)
Pub T (τ = 40 s)
Pub UT (τ = 31 s)
Pub T (τ = 38 s)