The influence of a six week exercise intervention on the pulmonary oxygen uptake kinetics in pre-pubertal obese and normal weight children

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Abstract

Purpose: The pulmonary oxygen uptake ($\dot{V}O_2$) response is deleteriously influenced by obesity in pre-pubertal children, as evidenced by a slower phase II response relative to their normal-weight counterparts. To date, no studies have investigated the ability of an exercise intervention to ameliorate this deleterious influence. Therefore, the purpose of the present study was to investigate the influence of a six week, high-intensity games orientated intervention on the $\dot{V}O_2$ kinetic response of pre-pubertal obese (OB) and normal-weight (NW) children during heavy intensity exercise.

Methods: Thirteen NW and fifteen OB children participated in a twice weekly exercise intervention involving repeated bouts of 6-minutes of high-intensity, games-orientated exercises followed by 2 minutes of recovery. Sixteen NW and eleven OB children served as a control group. At baseline and post-intervention, each participant completed a graded-exercise test to volitional exhaustion and constant work rate heavy intensity exercise (40% of the difference between the gas exchange threshold and peak $\dot{V}O_2$).

Results: Following the intervention, OB children demonstrated a significantly reduced phase II τ (Pre: 30±8 cf. Post: 24±7 s), MRT (Pre: 50±10 cf. Post: 38±9 s) and phase II amplitude (Pre: 1.51±0.30 cf. Post: 1.34 ± 0.27). In contrast, the intervention did not elicit any changes in the dynamic $\dot{V}O_2$ response in NW children.

Conclusions: The present findings demonstrate that a six-week, high-intensity intervention can have a significant positive impact on the dynamic $\dot{V}O_2$ response of obese pre-pubertal children.

Keywords: High intensity; games; $\dot{V}O_2$ kinetics; exercise intensity; training; BMI

Introduction

The prevalence of childhood obesity has reached epidemic proportions (Watts et al. 2005), with the World Health Organisation (WHO) recognising it as a major public health challenge of the 21st century (WHO 2010). This is of paramount importance considering that obesity is strongly associated with the occurrence of comorbities such as insulin resistance, type II diabetes mellitus, coronary artery disease, stroke and heart failure (Poirier et al. 2006). Recent statistics suggest that ~30% of children are overweight or obese (Townsend et al. 2013). Furthermore, childhood obesity is known to track strongly into adolescence and adulthood, with evidence suggesting that 80% of obese adolescents will become obese adults (Schonfeld-Warden et al. 1997). Exercise has been widely recognised as pivotal to resolving this obesity epidemic, however, there is little agreement within the literature as to effective exercise interventions. Regular physical activity and exercise participation for children, therefore, may be important in attenuating or even alleviating the severity of childhood obesity.

Pulmonary oxygen uptake ($\dot{V}O_2$) kinetics describe the finite rate of adjustment to a sudden change in metabolic demand, thus providing a functional evaluation of the integration of the pulmonary, cardiovascular and skeletal systems (Whipp et al. 1990). During moderate intensity exercise below the gas exchange threshold (GET), $\dot{V}O_2$ rises in a near-exponential manner (the so-called phase II response), attaining a steady-state within 2-3 minutes in young, healthy adults (Whipp et al. 1972; Whipp et al. 1982). The dynamic $\dot{V}O_2$ response becomes appreciably more complex during exercise above the GET, with attainment of a steady state delayed, or even precluded, by the presence of a supplementary "slow component" of $\dot{V}O_2$ (Whipp and Wasserman 1972; Barstow et al. 1991), that is thought to originate in the exercising muscle (Poole et al. 1991; Jones et al. 2011). This $\dot{V}O_2$ response has been shown to be highly sensitive in children, for example, a higher cardiorespiratory fitness is associated with a faster phase II time constant (τ) (McNarry et al. 2010; Winlove et al. 2010). Conversely, $\dot{V}O_2$ kinetics have also been shown to be deleteriously influenced by obesity during

weight-bearing exercise (Lambrick et al. 2013). The findings of this latter study are particularly pertinent given the utilisation of a weight-bearing exercise (i.e., walking, running) which is an important part of the daily activities of children. However, despite the sensitivity of $\dot{V}O_2$ kinetics and the pivotal role of exercise and physical activity as an intervention to resolve obesity, no study to date has investigated the influence of an exercise intervention on the $\dot{V}O_2$ response. Rather the reliance on peak $\dot{V}O_2$ as an indicator of aerobic fitness, despite the common recognition that this parameter can lack sensitivity (Impellizzeri et al. 2005; Meyer et al. 2005), may explain, at least in part, the discrepancy in the literature with regard to the trainability of pre-pubertal children (e.g. Welsman et al. 1997; Obert et al. 2009; Rowland et al. 2009). The importance of using appropriate modalities and parameters to assess the influence of training and interventions is especially evident in paediatric populations, particularly when considering that a meta-analysis demonstrated that aerobic endurance training may only elicit a 5-6% increase in peak $\dot{V}O_2$ (Baquet et al. 2003). The equivocal findings of previous studies may also be attributable to differences in the duration, intensity and/or type of exercise programme. Whilst the majority of previous studies have utilised continuous, sustained bouts of exercise, such bouts fail to reflect the highly sporadic nature of children's play patterns which are characterised by bursts of high-intensity, intermittent exercise (Bailey et al. 1995).

Therefore, the purpose of the present study was to investigate the effect of a short-term exercise intervention on the pulmonary oxygen uptake response of obese and normal weight children. To ensure participant adherence and the relevance of the intervention to the daily physical activity patterns of children, a high-intensity games model was used. We hypothesised that the intervention would significantly speed the $\dot{V}O_2$ kinetics in the obese children. Furthermore, we also hypothesised that the intervention would not influence the $\dot{V}O_2$ kinetics of the normal weight children due to their assumed higher baseline aerobic fitness.

Methods

Participants

Twenty-six obese (OB) children (9.3±0.9 y, 143.5±8.8 cm, 48.9±10.6 kg, 23.5±3.6 kg·m^2, 13 boys) and twenty-nine healthy normal weight (NW) children (9.2±0.8 y, 137.5±8.8 cm, 32.2±5.6 kg, 16.9±1.6 kg·m^2, 17 boys) volunteered for this study. BMI percentiles were used to classify children as either NW (5th to 85th percentile) or OB (≥ 95th percentile) (Cole et al. 2012). The participants were pre-pubescent, as determined by their predicted age at peak height velocity (Mirwald et al. 2002). On average, children took part in two or more weekly bouts of physical activity (i.e., football, netball, bike riding), as determined by parental-reports of activity status. Reported physical activity did not differ between the obese and normal weight children. Child assent and parent/guardian consent were obtained prior to participation. This research was conducted in agreement with the guidelines and policies of the institutional ethics committee.

Procedure

Participants took part in four laboratory-based exercise sessions; two maximal graded-exercise tests (GXT) to peak oxygen consumption ($\dot{V}O_2$ peak) and two repeated constant work rate exercise tests, all of which were conducted on a treadmill (True 825, Fitness Technologies, St Louis, USA).

Standing- and seated height (measured to the nearest 0.1cm; SECA, Hamburg, Germany), body weight, fat-free mass (measured to the nearest 0.1kg) and body fat percentage (% BF; measured with bioelectrical impedance analysis: InBody Biospace 230, Los Angeles, USA) were obtained on the initial visit to the laboratory. Following this, participants completed a baseline GXT to $\dot{V}O_2$ peak. After a minimum 72 hour interim period, participants completed a baseline constant work-rate exercise test. OB and NW participants were then randomised to either a 6-week child-specific, high-intensity exercise intervention, completed during school hours, or to a usual care control group which incorporated normal classroom activities. On completion of the exercise programme, all participants completed a post-intervention GXT to $\dot{V}O_2$peak and constant work-rate exercise test, similar to baseline.

Throughout all exercise tests, the treadmill grade was set at 1%. On-line respiratory gas analysis was undertaken continuously using a breath-by-breath automatic gas exchange system (Sensormedics Corporation, Yorba Linda, CA, USA). Children wore a paediatric facemask while they exercised to allow respiratory variables to be monitored continuously. A paediatric wireless chest strap telemetry system (Polar Electro T31, Kempele, Finland) measured heart rate (HR) throughout each exercise test. All physical and physiological data were concealed from the participants for the duration of each test.

Graded exercise test

Children were firstly familiarised to a range of treadmill speeds (4, 6 & 8 $km \cdot h^{-1}$) and the testing equipment. Habituation to the apparatus was considered essential to reduce any potential bias in the measurements. The GXT followed a discontinuous incremental protocol to ascertain peak oxygen uptake ($\dot{V}O_2$) and maximal HR (HRmax) (Lambrick et al. 2011; Lambrick, Faulkner et al. 2013). During this test participants completed a series of 1 minute exercise bouts with 1-minute recovery periods interspersed between each active bout of exercise until participants reported volitional exhaustion (For more details, see Lambrick, Faulkner et al. 2013) . The $\dot{V}O_2$peak was taken as the highest 10-s average value attained in the test. The GET was determined by the V-slope method (Beaver et al. 1986). Using the $\dot{V}O_2$ values reported at GET and $\dot{V}O_2$peak, the running speeds equivalent to 90 % GET (moderate exercise) and 40% delta (difference between GET and $\dot{V}O_2$peak; heavy exercise) were calculated for the constant work rate tests with the mean response time (MRT) for $\dot{V}O_2$ during ramp exercise accounted for (i.e., two-thirds of the ramp rate was deducted from the work rate at the GET and $\dot{V}O_2$peak (Whipp et al. 1981).

Constant work rate tests

For the determination of $\dot{V}O_2$ kinetics, participants completed a series of "step" tests. The protocol required participants to complete one moderate and one heavy intensity exercise bout, each of 6 min duration. Each bout was preceded by 5 min of rest followed by an abrupt transition to the target speed.

Randomisation

Participants were randomised to either a 6-week exercise programme (EX) or to a usual care control group (CON). OB and NW participants were randomly assigned using simple randomisation procedures (computerised random numbers). However, to ensure equal distribution of OB and NW children to both EX and CON the randomisation procedure was implemented separately for each group (OB, NW). Details of the allocated group were given on a piece of paper contained within sequentially numbered, opaque sealed envelopes. The randomisation procedures were prepared by an investigator with no involvement in the trial. Following randomisation, there were no significant differences between the exercise and control groups with regard to their anthropometrics, peak or constant work rate responses.

Exercise intervention

Participants randomised to EX completed a supervised high-intensity, child-specific, discontinuous games programme for 6 weeks. This included participation in twice weekly 60 minute exercise sessions, during which children were physically active for 40 minutes. There was a minimum 48 hour recovery period between sessions. These sessions were conducted outside of the children's normal Physical Education lessons (1 $hr \cdot wk^{-1}$) and were therefore considered supplementary exercise.

A two week pilot study demonstrated that children could manage repeated bouts of 6-minutes of high-intensity exercise followed by 2 minutes of recovery. To increase motivation, enjoyment and adherence, and to ensure children took part in each game at a high-intensity, different games (n=14) were used for each 6-minute exercise period (Howe et al. 2010). The 2-minute recovery period allowed researchers to provide instructions concerning the subsequent game activity. During each session, children took part in 6 child-specific games and a 4-minute circuit which included ladder running, step-ups, skipping, star jumps, high knees, shuttle runs, jumping jacks and lateral jumps. HR was constantly monitored throughout each exercise session (Polar Team^2 system, POLAR, Oulu, Finland).

The CON group continued to attend their weekly Physical Education classes, but no additional exercise sessions were implemented.

Data analysis

The $\dot{V}O_2$peak from the GXT is reported in absolute ($L \cdot min^{-1}$) and relative terms ($mL \cdot kg^{-1} \cdot min^{-1}$; $mL \cdot kg^{LBM} \cdot min^{-1}$[where LBM is $\dot{V}O_2$peak expressed relative to lean body mass]). Furthermore, the influence of body size was accounted for using analysis of covariance (ANCOVA) on log transformed data to determine the allometric relationship between body mass and peak $\dot{V}O_2$. Common allometric exponents were confirmed for all groups and power function ratios (Y/X^b) were computed.

Initially, the breath-by-breath $\dot{V}O_2$ responses to each step transition were visually 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 standard deviations 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 the cardiodynamic phase on the analysis of the subsequent response, the first 20s of data were ignored. Subsequently, following baseline correction, a mono-exponential model with a time delay (Eq.1) was then applied to this averaged response:

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

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 min of baseline), and A_1, δ_1 and τ_1 are the primary component amplitude, time delay (which was allowed to vary freely), and time constant, respectively. Kinetic variables (A_1, δ_1 and τ_1) and their 95% confidence intervals were determined by least squares non-linear regression analysis (Graphpad Prism, Graphpad Software, San Diego, CA). A mono-exponential model was ultimately used for both moderate and heavy intensity exercise as a bi-exponential ($\Delta\dot{V}O_{2(t)} = A_1 \cdot$

$(1 - e^{-(t-\delta_1)/\tau_1}) + A_2 \cdot (1 - e^{-(t-\delta_2)/\tau_2})$) during heavy exercise was found to produce an inferior and ambiguous fit.

Given the failure of the bi-exponential model to describe the $\dot{V}O_2$ response during heavy intensity exercise, the onset of the $\dot{V}O_2$ slow component was determined using purpose designed LabVIEW software which iteratively fits a monoexponential function to the $\dot{V}O_2$ data until the window encompasses the entire response. The resulting phase II time constants were 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. The amplitude of the $\dot{V}O_2$ peak slow component was subsequently determined by calculating the difference between the end exercise $\dot{V}O_2$ and the sum of the primary amplitude and 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 phase II $\dot{V}O_2$ response during both exercise intensities was also calculated by dividing the primary phase amplitude by the change in speed. The mean response time (MRT) was calculated by fitting a single exponential curve to the data with no time delay from the onset of exercise to the end of exercise. Finally, the oxygen deficit was determined using the MRT (expressed as a fraction of a minute) and the phase II amplitude.

Gaussian distribution was confirmed by the Shapiro-Wilks test. The influence of gender was assessed by an independent sample t-test which revealed no differences during either the incremental or constant work rate tests. Therefore, the data for boys and girls were pooled for all further analyses. A series of three-factor repeated measures analysis of variance (ANOVA), with weight status (NW, OB) and exercise condition (EX, CON) as the between-participant factors and test (baseline, post-intervention) as the within-participant factor, were used to analyse maximal data from the GXT to $\dot{V}O_2$peak and the physiological data reported from the constant work rate tests (for both moderate & heavy intensity exercise). Where statistical differences were observed, post-hoc analyses using Tukey's HSD were performed to identify where these differences lay. Where applicable, intention-to-treat analysis, whereby individual data recorded from the baseline assessment from a single

participant was carried forward and used in place of any missing data from the post-intervention assessment, was used on all relevant repeated-measures statistical procedures. All statistical analyses were conducted using PASW Statistics 21 (SPSS, Chicago, IL). All data are presented as means ± SD. Statistical significance was accepted when $P \leq 0.05$.

Results

Descriptive statistics

Participant descriptives are summarised in Table 1. There were no anthropometrical differences following randomisation between the exercise and control groups. The obese children demonstrated a significantly higher weight (OB, 49.2 ± 8.9 cf. NW, 32.5 ± 8.9 kg; $P < 0.05$), BMI (OB, 23.3 ± 2.9 cf. NW, 17.0 ± 2.9 kg·m^{-2}; $P < 0.05$) and %BF (OB, 32.3 ± 7.0 cf. NW, 20.7 ± 6.9%; $P < 0.05$) than their normal weight counterparts throughout the entire study. Irrespective of weight status, body mass increased from baseline to post-intervention (40.4 ± 8.6 cf. 41.4 ± 9.3 kg, respectively; $P < 0.05$)

Maximal physiological responses

The physiological responses to the incremental exercise test are summarised in Table 2. Both at baseline and post-intervention, OB children demonstrated a higher $\dot{V}O_2$peak than NW children when expressed in absolute terms (2.4 ± 0.4 cf. 2.0 ± 0.4 L·min^{-1}, respectively; $P < .001$), but no differences were observed when allometrically expressed relative to body mass. A significant increase in absolute $\dot{V}O_2$ peak was observed between baseline and post-intervention (2.1 ± 0.4 cf. 2.2 ± 0.4 L·min^{-1}, respectively; $P < .001$), but relative peak $\dot{V}O_2$, was unchanged. Furthermore, there were no main effects or interactions for HR (all $P > .05$), GET (L·min^{-1}) or the fraction of VO_2peak at which the GET occurred, irrespective of weight status (OB, NW) or exercise condition (EX, CON) ($P > .05$).

VO_2 kinetics: Moderate intensity exercise

During the moderate intensity exercise (Table 3), a trend was observed for a Test by Exercise Condition interaction for the phase II amplitude (P=0.061). Follow-up analysis demonstrated a lower phase II amplitude at the post-intervention than baseline assessment for EX (0.44±0.09 cf. 0.47±0.09 $L \cdot min^{-1}$, respectively) but not for CON (0.46±0.09 cf. 0.45±0.09 $L \cdot min^{-1}$, respectively). The phase II amplitude and gain were significantly higher in the OB children (all P<.01).

VO_2 kinetics: Heavy intensity exercise

The physiological data from the Heavy intensity exercise are reported in Table 4.

Phase II amplitude

A significant Test (baseline, post-intervention) by Exercise Condition (EX, CON) by weight status (OB, NW) interaction was observed for the phase II amplitude (P<.05). Post hoc analysis revealed the following: a significant decrease in the OB children phase II amplitude between baseline and post-intervention for EX (P<.01), a significant increase in the OB children phase II amplitude between baseline and post-intervention for those in CON (P<.05), a significantly lower phase II amplitude for OB children at post-intervention for EX compared to CON (P<.001) and that NW children elicited a lower phase II amplitude than OB children, regardless of the Exercise Condition or Test (all P<.001).

Phase II gain

A significant Test by Exercise Condition by weight status interaction was observed for the phase II gain (P<.05). Post hoc analysis demonstrated the following: a significant decrease in the phase II gain between baseline and post-intervention for OB children taking part in EX (P<.001), significantly lower values for OB children taking part in EX than OB children randomised to CON at the post-

intervention assessment (P<.001), and that NW children (EX & CON) elicited lower values than OB children (EX & CON) at both the baseline and post-intervention assessments (P<.001).

MRT

A Test by Exercise Condition interaction was observed for MRT, with a significant decrease reported from baseline to post-intervention for EX (47.4±8.8 to 40.5±9.7 s, respectively), but not for CON (45.7±8.9 to 46.1±9.8 s, respectively) (P < .05).

Phase II time delay

A significant Test by Exercise Condition by weight status interaction was observed for the phase II time delay with the greatest change reported between the baseline and post-intervention assessment for OB children (P<.05).

Phase II τ

A significant Test by Exercise Condition by weight status interaction was also revealed for τ (P<.05). Post hoc analysis demonstrated a greater decrease in the OB children phase II τ between baseline and post-intervention for EX than all other conditions (P<.01), as shown in Figure 1.

Slow Component

A slow component was observed in all participants during heavy intensity exercise, but was not influenced by weight status, Exercise Condition or Test, irrespective of the method of expression (all P>.05).

Discussion

The main finding of the present study was that a six-week high-intensity, games-orientated intervention significantly influenced the $\dot{V}O_2$ kinetic response of obese pre-pubertal children. Specifically, and in accordance with our hypothesis, the obese children demonstrated a significantly faster phase II τ and MRT and reduced phase II amplitude during heavy intensity exercise. The intervention did not influence the dynamic $\dot{V}O_2$ response in the normal weight children. These findings demonstrate that postive improvements in the dynamic $\dot{V}O_2$ response of obese children may be elicited following regular participation in just six weeks of high-intensity exercise.

The current study is the first to investigate the influence of an exercise intervention on the dynamic $\dot{V}O_2$ response of obese pre-pubertal children. In this study, a slower phase II τ was observed at baseline in the obese children, supporting some (Lambrick, Faulkner et al. 2013), but not all (Cooper et al. 1990; Rasmussen et al. 2000; Unnithan et al. 2007), previous research. Interestingly, a significant speeding of the phase II τ (20%) and MRT (24%) and reduction of the phase II amplitude (11%) was observed in obese children during heavy intensity exercise following the exercise intervention. Whilst direct comparisons are precluded, it is pertinent to note that long-term, intensive training has been reported to elicit a phase II τ that is 32% faster than that observed in untrained counterparts (Winlove, Jones et al. 2010). This is remarkably similar to that observed in the present study and demonstrates the efficacy of our short-term exercise intervention; a programme which reflects the high-intensity nature of children's play patterns (Bailey, Olson et al. 1995). These findings further support those which suggest peak $\dot{V}O_2$ may lack sensitivity to changes in fitness (Impellizzeri, Marcora et al. 2005; Meyer, Lucia et al. 2005) and highlight the superior sensitivity of $\dot{V}O_2$ kinetics.

It is of interest to note that the influence of this exercise programme was weight-status dependent; specifically no change was observed following the intervention in the normal weight children irrespective of exercise intensity. As exercise-induced improvements are inversely related to the initial baseline fitness of an individual (Eliakim et al. 1996; Mandigout et al. 2001), the relatively high level

of fitness of the normal-weight children at the start of the programme may plausibly have necessitated a greater training stimulus for significant effects of the intervention to be observed in this population.

In healthy individuals during upright exercise, the dynamic $\dot{V}O_2$ response is purported to be predominantly determined by factors inherent to the mitochondria. Specifically, the delayed activation of oxidative phosphorylation at the onset of exercise is suggested to be partially attributable to determinants such as pyruvate dehydrogenase (Grassi et al. 2002), nitric oxide (Jones et al. 2003; Jones et al. 2004) and the PC-Cr shuttle concept of regulation (Grassi 2005). However, there may be circumstances, such as certain pathologies or exercise conditions that are associated with an altered microvascular O_2 partial pressure of a muscle fibre or the fibre type recruitment profile, in which rate of the $\dot{V}O_2$ response is determined by oxygen delivery. Consequently, it is now widely accepted that these two factors may co-exist on a continuum, distinguished by a "tipping point" in muscle oxygen delivery. Therefore, the faster $\dot{V}O_2$ kinetics observed post-intervention in the obese children in the present study may be attributable to an improved muscle oxygen delivery and/or to a greater muscle oxidative capacity. Indeed, while information regarding the muscle oxidative capacity of children is almost non-existent (Mahon 2008), the limited information available suggests that training may elicit an increased muscle oxidative enzyme activity (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). Whether similar adaptations occurred in the present obese population remains to be elucidated. However, when considering the mechanistic basis for the significant speeding of the phase II response in the present obese children, it may be pertinent to consider the greater proportion of type II muscle fibres reported in obese participants (Kriketos et al. 1997). Type II muscle fibres are characterised by a higher glycolytic but lower oxidative capacity than type I fibres (Crow et al. 1982; Krustrup et al. 2008). In addition, evidence suggests that type II fibres are less efficient, fatigue more easily and elicit lower microvascular O_2 partial pressures, both across the transient phase and during steady state exercise (Coyle et al. 1992; Behnke et al. 2003; McDonough et al. 2005). These differences are likely to exert considerable influence on the overall $\dot{V}O_2$ kinetics

exhibited, as supported by the inherently slower $\dot{V}O_2$ response reported in type II relative to type I muscle fibres (Crow and Kushmerick 1982; Krustrup, Secher et al. 2008). Thus, although it is presently uncertain whether the aforementioned findings are applicable to paediatric populations, an altered proportion or recruitment pattern of type II muscle fibres in the obese children post-intervention may contribute to the faster phase II τ observed.

In accordance with our previous study (Lambrick, Faulkner et al. 2013), a similar $\dot{V}O_2$ slow component amplitude was observed between the obese and normal weight children, irrespective of weight status, exercise participation (EX vs. CON) or assessment session (baseline, post-intervention). Although the aetiology of the slow component remains to be conclusively elucidated, ~90% of this component has been shown to arise from within the exercising muscle (Poole, Schaffartzik et al. 1991; Rossiter et al. 2002b), with the recruitment of additional (less efficient) type II muscle fibres widely purported (Rossiter et al. 2002a; Krustrup et al. 2004). These findings are therefore contrary to what we would hypothesise, as a reduced reliance on type II fibres might be expected to be associated with a reduced slow component magnitude. However, since muscle fibre type distribution and abundance was not assessed in the present study, further interpretation is beyond the scope of the current study. It is prudent to note, however, that the similar slow component amplitudes observed in the present participants might be a reflection of a slower development of this component in the obese individuals, i.e. the slow component might not have been fully manifest at 6 minutes in the obese children, artificially causing it to appear diminished.

The exercise intervention utilised in the present study demonstrated high rates of adherence (94%) and consequently proved highly successful in eliciting significant physiological adaptations. However, the present study is not without its limitations. Bias may have been introduced into the study as a result of voluntary participant recruitment, i.e., children who 'like' exercise wished to be involved in the study and as such, this may be reflected in the high peak $\dot{V}O_2$ values observed pre-intervention, irrespective of weight-status. In terms of the findings, whilst a high baseline fitness may

384 explain the absence of an effect within the normal-weight children, it also indicates that the
385 intervention programme may have an even greater influence in a more representative obese
386 population. No measures of habitual physical activity were taken post-intervention, therefore it is not
387 presently possible to account for the effect of any potential changes in habitual physical activity and
388 their influence of the results observed. Finally, It is suggested that several repeat transitions are
389 needed for reliable characterisation of $\dot{V}O_2$ kinetic response (Lamarra et al. 1987). Although the
390 current study only utilised one transition, the confidence intervals during heavy intensity exercise are
391 nonetheless within those advocated by Fawkner and Armstrong (2007).
392
393 In conclusion, the present study demonstrates that a six week, high-intensity, games-orientated
394 intervention significantly improved the $\dot{V}O_2$ kinetic response of obese, pre-pubertal children.
395 Specifically, during heavy intensity exercise, the obese children demonstrated a 20% faster primary
396 phase response, 24% faster MRT and an 11% lower phase II amplitude. These adaptations would be
397 expected to reduce metabolic perturbation and fatigue development in the transition from a lower to a
398 higher metabolic rate, and may therefore improve exercise tolerance. Furthermore, if sustained, they
399 may directly translate to a reduced O_2 cost of daily activities and thereby potentially enhance
400 functional capacity and quality of life.
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543 Figure 1. Pulmonary oxygen uptake response during heavy intensity exercise in a representative A)
544 obese and B) normal-weight participant pre (open circles) and post (closed circles) the exercise
545 intervention.