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The influence of body weight on the pulmonary oxygen uptake kinetics in pre-pubertal children during moderate- and heavy intensity treadmill exercise

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Abstract

Purpose: To assess the influence of obesity on the oxygen uptake ($\dot{V}O_2$) kinetics of pre-pubertal children during moderate- and heavy intensity treadmill exercise. We hypothesised that obese (OB) children would demonstrate significantly slower $\dot{V}O_2$ kinetics than their normal weight (NW) counterparts during moderate- and heavy intensity exercise. **Methods:** 18 OB (9.8±0.5 y; 24.1±2.0 kg·m^2) and 19 NW (9.7±0.5 y; 17.6±1.0 kg·m^2) children completed a graded-exercise test to volitional exhaustion and two submaximal constant work rate treadmill tests at moderate (90% gas exchange threshold) and heavy (Δ40%) exercise intensities. **Results:** Bodyweight significantly influenced the $\dot{V}O_2$ kinetics during both moderate- and heavy exercise intensities ($P < 0.05$). During moderate intensity exercise, the phase II τ (OB: 30±13 cf. NW: 22±7 s), mean response time (MRT; OB: 35±16 cf. NW: 25±10 s), phase II gain (OB: 156±21 cf. NW: 111±18 mLO_2·kg^{-1}·km^{-1}) and oxygen deficit (OB: 0.36±0.11 cf. NW: 0.20±0.06 L) were significantly higher in the OB children (all $P < 0.05$). During heavy intensity exercise, the τ (OB: 33±9 cf. NW: 27±6 s; $P < 0.05$) and phase II gain (OB: 212±61 cf. NW: 163±23 mLO_2·kg^{-1}·km^{-1}; $P < 0.05$) were similarly higher in the OB children. A slow component was observed in all participants during heavy intensity exercise but was not influenced by weight status. **Conclusion:** This study demonstrates that weight status significantly influences the dynamic $\dot{V}O_2$ response at the onset of treadmill exercise in children and highlights that the deleterious effects of being obese are already manifest pre-puberty.

Keywords

Paediatric; Oxygen uptake; Obese; Moderate intensity exercise; Heavy intensity exercise

Introduction

The prevalence of childhood obesity has increased at an alarming rate and is currently considered one of the most serious public health challenges of the 21st century (WHO 2010). Existing literature suggests that overweight or obesity in youth may likely persist into adulthood (Reilly et al. 2003; Singh et al. 2008), increasing the risk of developing non-communicable diseases, like diabetes and cardiovascular disease, at an earlier age (Franks et al. 2010). Childhood obesity also contributes to severe cardiorespiratory deconditioning, leading to limitations in motor activity, physical functioning and exercise tolerance, and consequentially resulting in a poorer quality of life (Gidding et al. 2004). Despite our understanding of the preventable nature of overweight and obesity-related diseases and the significant role that exercise can have in terms of intervention strategies (Chen 2012), the specific influence of obesity on exercise performance and its mechanistic basis remains to be determined (Loftin et al. 2005; Salvadego et al. 2010; Unnithan et al. 2007).

Pulmonary oxygen uptake ($\dot{V}O_2$) kinetics, which describe the dynamic response to a sudden change in external work rate, has been well characterised (for a detailed review see: Jones and Poole 2005) and has shown to be dependent upon exercise intensity above the gas exchange threshold (GET; Koppo et al. 2004; McNarry et al. 2012; Wilkerson et al. 2004). The approximately exponential increase in $\dot{V}O_2$, referred to as phase II or the primary component, is generally accepted to reflect the oxygen consumption within the muscle (Grassi et al. 1996; Krustrup et al. 2009), thereby providing a non-invasive insight into the metabolic activity of the exercising muscle. During exercise above the GET, the attainment of a steady state $\dot{V}O_2$ is delayed or even precluded by the presence of an additional, slowly developing component of $\dot{V}O_2$, aptly termed the $\dot{V}O_2$ slow component, that also emanates primarily from the exercising muscle (Barstow and Mole 1991; Poole et al. 2008; Poole et al. 1991; Whipp and Wasserman 1972).

Although less extensively investigated in children, research suggests that the dynamic $\dot{V}O_2$ response is highly sensitive to both advantageous (e.g. training Marwood et al. 2010; McNarry et al. 2010; Winlove et al. 2010) and deleterious (e.g. pathology Hebestreit et al. 2005; Nadeau et al. 2009) influences. Accordingly, the potential influence of obesity on the $\dot{V}O_2$ response is of growing interest, not only because of its increasing prevalence but because of the functional effect that excess body fat may have on skeletal muscle. For example, obese individuals exhibit a considerable functional limitation in motor activity yet their skeletal muscles are exposed to an inherent chronic strength training stimuli due to the

excess weight (Salvadego et al. 2010). It has been suggested that obese children and adolescents are characterised by slower $\dot{V}O_2$ kinetics than their normal weight counterparts, likely due to impairments in skeletal muscle oxidative metabolism (Potter et al. 2013; Salvadego et al. 2010). Conversely, earlier research has found no differences in the $\dot{V}O_2$ kinetics of obese and normal weight children (Cooper et al. 1990; Unnithan et al. 2007) or adolescents (Loftin et al. 2005). Such ambiguous findings may be a consequence of methodological limitations, including the use of only a single exercise transition to characterise $\dot{V}O_2$ kinetics (Loftin et al. 2005; Unnithan et al. 2007), the prescription of exercise intensity as a fraction of peak $\dot{V}O_2$ (Salvadego et al. 2010) or at a set work rate (Unnithan et al. 2007), or the use of mixed sex and/or biological maturity stage cohorts (Cooper et al. 1990; Unnithan et al. 2007). As children demonstrate relatively small $\dot{V}O_2$ response amplitudes and large inter-breath $\dot{V}O_2$ variability, it is necessary to perform repeat transitions in order for $\dot{V}O_2$ kinetics to be confidently characterized (Fawkner and Armstrong 2007). Furthermore, the relatively high inter-individual variability in the fraction of peak $\dot{V}O_2$ at which the GET occurs in children compared to adults (Fawkner and Armstrong 2007) means that it is important to consider both the GET and the peak $\dot{V}O_2$ when attempting to standardise the exercise intensity domain in which participants are exercising. It is also of particular interest to note that previous research has assessed the $\dot{V}O_2$ kinetic response during non-weight bearing exercise (Cooper et al. 1990; Potter et al. 2013; Salvadego et al. 2010; Unnithan et al. 2007) and as such, the mechanistic influence of weight-bearing exercise (walking, running) on $\dot{V}O_2$ kinetics has been, to date, overlooked in children. Furthermore, the nature of the $\dot{V}O_2$ kinetic response in obese children during heavy intensity exercise is yet to be examined in detail (Potter et al. 2013).

The purpose of the present study was therefore to examine the influence of obesity on the $\dot{V}O_2$ kinetics of pre-pubertal children during moderate- and heavy intensity treadmill exercise. We hypothesised that obese children would have characteristically slower $\dot{V}O_2$ kinetics, irrespective of the exercise intensity, compared with normal weight children.

Methods

Participants

Nineteen healthy normal weight (NW) children (9.7 ± 0.5 y, 140.9 ± 6.1 cm, 35.0 ± 3.5 kg, 17.6 ± 1.0 kg·m^2, 10 boys) and eighteen obese (OB) children (9.8 ± 0.5 y, 145.7 ± 6.9

cm, 51.5 ± 7.0 kg, 24.1 ± 2.0 kg·m^2, 9 boys) volunteered for this study. BMI percentiles were used to classify children as either NW (5th to 85th percentile) or OB (≥ 95th percentile) (Cole and Lobstein 2012). To ascertain that children were asymptomatic of illness, disease and pre-existing injuries, parents / guardians completed a standardised health screening questionnaire. 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 three laboratory-based exercise sessions within a thermo-neutral environment (temperature: 21.8 ± 1.9 °C; humidity: 38.4 ± 7.0 %; air pressure: 1005 ± 9 N·m^2); a maximal graded-exercise test (GXT) to peak oxygen consumption ($\dot{V}O_2$peak) and two repeated constant work rate exercise tests. A minimum 72 hour interim period was imposed between these 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. Girth measures of the waist, hip, forearm, biceps, quadriceps and calf (Lufkin W606PM, Apex Tools Group, Maryland, USA) were taken from the right side of each participant.

Throughout all exercise tests, the treadmill grade was set at 1% to parallel the suggested oxygen cost of running outside (Jones and Doust 1996). 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 (oxygen uptake [$\dot{V}O_2$], carbon dioxide [$\dot{V}CO_2$], minute ventilation [$\dot{V}_E$], respiratory exchange ratio [RER]). 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 km·h^{-1}, 6 km·h^{-1} & 8 km·h^{-1}) and the testing equipment (facemask, HR monitor). Habituation to the apparatus was

considered essential to reduce any potential bias in the measurements. The GXT followed a discontinuous incremental protocol to ascertain $\dot{V}O_2$peak and maximal heart rate (HRmax) (Lambrick et al. 2011). Children commenced the test at 4 km·h^{-1} and walked at that intensity for 1 minute. The treadmill speed was then slowed to a stop (0 km·h^{-1}) to allow a 1 minute recovery before the treadmill speed was increased to 6 km·h^{-1} for a further minute of exercise. Increments of 1 km·h^{-1} continued with this protocol until a speed of 8 km·h^{-1} was accomplished. Thereafter, increments in running speeds of 0.5 km·h^{-1} occurred (8.5, 9.0, 9.5, 10.0 km·h^{-1}, etc.) until volitional exhaustion. A 1 minute recovery period was implemented following each minute of active exercise for the duration of the test. The $\dot{V}O_{2peak}$ was taken as the highest 10-s average value attained before the subject's volitional exhaustion 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 (Δ; 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 peak $\dot{V}O_2$ (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, which was performed two times on separate days, comprised one moderate- and one heavy intensity transition, each of 6 min duration. Each transition was preceded by 5 min of rest followed by an abrupt transition to the target speed. Both the moderate- and heavy intensity exercise elicited running exercise in both normal and obese children.

Data analysis

The $\dot{V}O_{2peak}$ from the GXT is reported in absolute (L·min^{-1}) and relative terms (mL·kg^{-1}·min^{-1}; mL·kg^{LBM}·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 $\dot{V}O_2$ (Welsman and Armstrong 2000). 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. 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) in which the best fit was defined by minimisation of the residual sum of squares and minimal variation of residuals around the Y-axis (Y = 0). 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 (Fawkner and Armstrong 2004; 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 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 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. To account for the influence of body size, the phase II amplitude was allometrically scaled prior to the calculation of the oxygen deficit which was also expressed as a percentage of the phase II amplitude. The O_2 deficit was only determined for moderate intensity exercise because the presence of the $\dot{V}O_2$ slow component precludes the determination of the O_2 deficit during heavy intensity exercise.

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. Following this, the results of the incremental test were analysed to assess the influence of weight status using an independent samples t-test. The remaining data were analysed using a mixed 'between-within' analysis of variance (ANOVA), with weight status as a between-participant factor and exercise intensity as a within-participant factor. Pearson product moment correlation coefficients were used to analyse the degree of association between key variables. All statistical analyses were conducted using PASW Statistics 18 (SPSS, Chicago, IL). All data are presented as means ± SD. Statistical significance was accepted when $P \leq 0.05$.

Results

The physiological responses to the incremental exercise test are summarised in Table 1. The OB children demonstrated a significantly higher absolute- but lower relative peak $\dot{V}O_2$ (both $P < 0.05$). When $\dot{V}O_2$peak was expressed relative to lean body mass, or when allometrically scaled, no significant differences were observed between groups ($P > 0.05$), although there was a trend for normal weight children to achieve a higher allometrically scaled peak $\dot{V}O_2$. The peak heart rate and the fraction of peak $\dot{V}O_2$ at which the GET occurred were both unaffected by weight status ($P > 0.05$). Moderate intensity exercise corresponded to a group mean speed of 8±1 $km \cdot h^{-1}$ and heavy intensity exercise to 10±1 $km \cdot h^{-1}$.

$\dot{V}O_2$ kinetics

The parameters determined from the monoexponential modelling revealed a significant influence of weight status on the $\dot{V}O_2$ kinetics during both exercise intensities, as presented in Table 2 and illustrated in Figure 1. Specifically, during moderate intensity exercise, the phase II τ, MRT and phase II gain were significantly higher in the OB children. Furthermore, the oxygen deficit was higher in the OB children, whether expressed in absolute (OB: 0.36 ± 0.11 cf. NW: 0.20 ± 0.06 L; $P < 0.05$) or relative terms (OB: 61 ± 25 cf. NW: 45 ± 12 %; $P < 0.05$). During heavy intensity exercise, the τ and phase II gain were similarly higher in OB children and there was a trend for the MRT to be longer. A slow component was observed in all participants during heavy intensity exercise but was not influenced by weight status, irrespective of the method of expression. Peak $\dot{V}O_2$ was significantly related to the phase II τ during both moderate ($r = 0.39$, $P < 0.05$) and heavy intensity exercise ($r = 0.33$, $P < 0.05$).

In the NW children, both the temporal and amplitude related parameters were significantly influenced by exercise intensity, being greater during heavy compared to moderate intensity exercise ($P < 0.05$). In the OB children, only the amplitude and MRT were significantly greater during heavy intensity exercise ($P < 0.05$).

Discussion

This study demonstrated that the dynamic $\dot{V}O_2$ response of pre-pubertal children is influenced by weight status during moderate- and heavy intensity treadmill exercise. Although absolute $\dot{V}O_2$peak was shown to be higher in obese participants, there was no discernible difference in $\dot{V}O_2$peak when expressed relative to lean body mass. This similarity in relative oxygen consumption is particularly interesting when considering the differences in $\dot{V}O_2$ kinetic response between these obese and normal weight children. Specifically, during moderate- and heavy intensity exercise, the phase II τ was 27% and 18% slower, respectively, in obese children compared to their normal weight counterparts. The obese children also elicited a higher phase II gain, irrespective of exercise intensity, and greater oxygen deficit during moderate intensity exercise. These findings confirm our experimental hypothesis and are consistent with the evidence for an impaired skeletal muscle oxidative metabolism in obese children (Gidding et al. 2004; Salvadego et al. 2010).

In the current study, the slower phase II τ observed for the obese children during treadmill running confirms recent findings from cycle ergometry (Potter et al. 2013;

Salvadego et al. 2010). Potter and colleagues have demonstrated significantly slower phase II τ and MRT in obese 11-12 year old children compared to age-matched normal weight controls (Potter et al. 2013). Similar findings were observed with 15-17 year old obese adolescents (Salvadego et al. 2010). It is interesting to note that the findings of our study with 8 to 10 year old children, in conjunction with the findings of Potter et al. (2013), suggest that the deleterious effects of excess bodyweight are already evident in pre-pubertal children.

In addition to the slower $\dot{V}O_2$ kinetics and greater phase II amplitude that was observed, obese children also exhibited a greater O_2 deficit; a difference that persisted once body mass was accounted for. A larger O_2 deficit is associated with a greater reliance on substrate level phosphorylation to meet the increased metabolic demand at the onset of exercise, thereby engendering a greater metabolic perturbation. This series of events has conventionally been implicated in a reduced exercise tolerance (e.g. see reviews: Grassi 2006; Whipp and Rossiter 2005). However, it has recently been suggested that slower $\dot{V}O_2$ kinetics may rather be an "epiphenomenon" of a lower metabolic stability (Grassi et al. 2011). Accordingly, during treadmill exercise, it may be obese children's lower metabolic stability which is the primary determinant to their reduced exercise tolerance (Grassi et al. 2011).

The rate-limiting determinant of the $\dot{V}O_2$ kinetic response continues to be a contentious issue, with oxygen delivery and oxygen utilisation both proposed as putative mediators (Grassi 2005; Poole et al. 2008). More recently it has been suggested that these two theories co-exist on a continuum, distinguished by a "tipping point" in muscle oxygen delivery (for further information see Poole and Jones 2005; Poole et al. 2008). Specifically, there is compelling evidence to suggest that in young healthy individuals, $\dot{V}O_2$ kinetics are subject to mitochondrial control rather than limited by the bulk muscle oxygen delivery (Grassi 2005; Grassi 2006; Poole and Jones 2012; Rossiter 2011). Factors 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) have all been proposed as being partially responsible for the delayed activation of oxidative phosphorylation at the onset of exercise (Grassi 2005). However, some pathologies (e.g. diabetes, heart failure) and exercise conditions (e.g. inspired hypoxia/hyperoxia) may change the microvascular O_2 partial pressure of a muscle fibre or the fibre type recruitment profile which in-turn may modify the balance of exercise with respect to the "tipping point" (Poole et al. 2008).

In this regard, it is perhaps pertinent to note the greater proportion of type II muscle fibres previously reported in obese participants (Kriketos et al. 1997). Type II muscle fibres have a lower oxidative but a higher glycolytic capacity (Crow and Kushmerick 1982; Krustrup et al. 2008) and have been shown to be less efficient and fatigue resistant than type I muscle fibres (Coyle et al. 1992). Type I and type II muscle fibres demonstrate clear differences in their regulation of the O_2 delivery/utilisation balance, with type II fibres engendering lower microvascular O_2 partial pressures, both across the transient phase and during steady state exercise (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 et al. 2008). While it is unclear whether these findings in adults are relevant for children, a greater reliance on type II muscle fibres might be anticipated to result in an overall slowing of the dynamic $\dot{V}O_2$ response in this paediatric population. Incidentally, we might expect the obese children to demonstrate a greater (slower developing) $\dot{V}O_2$ slow component. In fact, what we observed was a similar $\dot{V}O_2$ slow component amplitude between the obese and normal weight children. It is plausible that the 6 minutes of steady state exercise may not have been sufficient to allow the slow component to fully manifest in the obese children, thus artificially causing it to appear diminished.

A novel aspect of the current study was the use of treadmill ergometry for the determination of $\dot{V}O_2$ kinetics. Previous studies involving paediatric populations have relied upon cycle (Cleuziou et al. 2002; Obert et al. 2000; Potter et al. 2013; Winlove et al. 2010) or arm crank ergometry (Winlove et al. 2010). More specifically, studies investigating the influence of weight status on the dynamic $\dot{V}O_2$ response in children have exclusively utilised cycle ergometry (Cooper et al. 1990; Loftin et al. 2005; Potter et al. 2013; Salvadego et al. 2010; Unnithan et al. 2007). Whilst each ergometer undoubtedly has its relative merits, the treadmill has the significant advantage that it more closely resembles the habitual physical activity of children. Furthermore, with regards to the comparison of children of differing bodyweights (obese cf. normal weight), it is also pertinent that treadmill exercise requires individuals to transport their total body mass. Thus, differences in $\dot{V}O_2$ responses of children of differing body mass between the current study and past research (Cooper et al. 1990; Loftin et al. 2005; Unnithan et al. 2007) may in part be explained by the differences in exercise modality (weight bearing cf. non-weight bearing, respectively) or biomechanical parameters

(i.e. gait, mechanical work) which may become evident during treadmill exercise (Lee and Hidler 2008). Further consideration within this area is warranted, however.

With the exception of the $\dot{V}O_2$ slow component, which was significantly smaller (but similar irrespective of weight status) in the present study, the parameters of the $\dot{V}O_2$ kinetic response are similar to those reported previously during cycle ergometry for normal weight children (Cleuziou et al. 2002; Obert et al. 2000; Winlove et al. 2010). This is in keeping with past research in adults (Carter et al. 2000). Given the debate that continues to surround the aetiology of the $\dot{V}O_2$ slow component, it is difficult to provide an explanation for the smaller $\dot{V}O_2$ slow component during treadmill exercise. In adults, it has been suggested that it may be related to a lower intramuscular tension development but higher eccentric exercise component during running (Carter et al. 2000), but the applicability of these suggestions to the present pre-pubertal population remains to be elucidated.

Four key parameters have been identified for the assessment of an individual's aerobic fitness: $\dot{V}O_2$peak, the GET, exercise economy and $\dot{V}O_2$ kinetics (Whipp et al. 1981). This is the first study that has simultaneously assessed all of these parameters in obese and normal weight children. The present findings suggest that the influence of body mass is manifest in the relative and temporal parameters (phase II gain and phase II τ) but not in the absolute magnitude of the $\dot{V}O_2$ response (peak $\dot{V}O_2$, GET, slow component amplitude). These findings thus highlight the importance of not relying solely on the assessment of $\dot{V}O_2$peak, as has been common practice in paediatric exercise science (Armstrong and Fawkner 2007; Potter et al. 2013).

It is prudent to acknowledge certain limitations with the study design; i) The inclusion of a $\dot{V}O_2$peak validation test would have been more scientifically valid to confirm whether the $\dot{V}O_2$peak values obtained at termination of the GXT were true peak values for this study population (Barker et al. 2009; Day et al. 2003; Poole et al. 2008). ii) It is suggested that several repetitions of exercise are needed for reliable $\dot{V}O_2$ kinetics analyses (Lamarra et al. 1987). Although the current study only utilised two repeat transitions, this is in keeping with previous research in this area (Cleuziou et al. 2002; Marwood et al. 2010; Obert et al. 2000). iii) Although 40%Δ was chosen to reflect the upper limit of heavy intensity exercise (Carter et al. 2002; Fawkner and Armstrong 2003; Winlove et al. 2010), we are unable to retrospectively determine whether this was above or below critical power.

In conclusion, this study demonstrates that weight status significantly influences the dynamic $\dot{V}O_2$ response at the onset of exercise in pre-pubertal children. Specifically, obese children demonstrate a slower phase II τ and greater phase II gain during both moderate- and heavy intensity exercise. Furthermore, even following appropriate scaling, the slower $\dot{V}O_2$ kinetics were associated with a greater O_2 deficit during moderate intensity exercise, which would likely result in a reduced exercise tolerance. This study also highlighted that the deleterious effects of being obese are already manifest pre-puberty.

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Table 1. Peak physiological responses to treadmill exercise in obese (OB) and normal weight (NW) children

	OB	NW
N	18	19
$\dot{V}O_2$peak ($L \cdot min^{-1}$)	2.64 ± 0.29 *	2.23 ± 0.40
$\dot{V}O_2$peak ($mL \cdot kg^{-1} \cdot min^{-1}$)	52.1 ± 7.3 *	63.6 ± 9.1
$\dot{V}O_2$peak ($mL \cdot kg^{-0.61} \cdot min^{-1}$)	240.3 ± 26.6	254.2 ± 38.3
$\dot{V}O_2$peak ($mL \cdot kg^{LBM} \cdot min^{-1}$)	151.1 ± 19.2	150.1 ± 18.9
Peak HR ($b \cdot min^{-1}$)	200 ± 15	198 ± 12
GET ($L \cdot min^{-1}$)	1.70 ± 0.41 *	1.38 ± 0.29
GET (%)	65 ± 10	62 ± 7

Mean ± SD. $\dot{V}O_2$, oxygen uptake; HR, heart rate; GET, gas exchange threshold; LBM, Lean body mass

* *significant difference relative to age-matched counterpart ($P < 0.05$).*

Table 2. Oxygen uptake kinetics during moderate- and heavy intensity exercise on a treadmill in obese (OB) and normal weight (NW) children

	Moderate Intensity		Heavy Intensity	
	OB	NW	OB	NW
N	18	19	18	19
Phase II time delay (s)	10 ± 9	6 ± 11	14 ± 6	15 ± 3 $^{\#}$
Phase II τ (s)	30 ± 13 *	22 ± 7	33 ± 9 *	27 ± 6 $^{\#}$
95% confidence interval (s)	6 ± 2	6 ± 2	4 ± 1	4 ± 2
MRT (s)	35 ± 16 $^{*\#}$	25 ± 10	52 ± 14 $^{\#}$	46 ± 7
Phase II amplitude ($L \cdot min^{-1}$)	0.63 ± 0.08 *	0.44 ± 0.07	1.75 ± 0.26 $^{\#}$	1.35 ± 0.27 $^{\#}$
Phase II amplitude ($mL \cdot kg^{\cdot b} \cdot min^{-1}$)	47.8 ± 5.9 *	42.2 ± 5.8	336.0 ± 48.5 $^{\#}$	303.7 ± 55.6 $^{\#}$
Phase II gain ($mLO_2 \cdot kg^{-1} \cdot km^{-1}$)	156.3 ± 21.2 *	110.7 ± 17.6	212.2 ± 60.6 $^{*\#}$	163.3 ± 23.4 $^{\#}$
Slow component amplitude ($L \cdot min^{-1}$)			0.05 ± 0.02	0.05 ± 0.02
Slow component amplitude (% EE $\dot{V}O_2$)			3 ± 2	4 ± 2
End-exercise HR ($b \cdot min^{-1}$)	120 ± 14	113 ± 8	182 ± 18	176 ± 16 $^{\#}$
End-exercise $\dot{V}O_2$ ($L \cdot min^{-1}$)	0.91 ± 0.09	0.71 ± 0.09	2.07 ± 0.28*	1.71 ± 0.28
End-exercise $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	28.8 ± 7.5*	35.5 ± 6.6	31.7 ± 7.0*	36.8 ± 4.7
End-exercise $\dot{V}_E$ ($L \cdot min^{-1}$)	57.8 ± 13.5*	46.2 ± 9.4	76.6 ± 17.5*#	56.6 ± 9.3#
End-exercise RER	0.93 ± 0.03*	0.90 ± 0.03	0.97 ± 0.04*#	0.95 ± 0.04#

Mean ± SD. MRT, mean response time; EE, end exercise; $\dot{V}O_2$, oxygen uptake; HR, heart rate; RER, respiratory exchange ratio; ^{b,} *moderate phase II amplitude scaling exponent, 0.66; heavy phase II amplitude scaling exponent, 0.42.*

* *Significant difference between groups within an exercise intensity (P < 0.05).*

$^{\#}$ *Significant difference between exercise intensities within group (P < 0.05).*