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PEDIATRIC ORIGINAL ARTICLE

The oxygen uptake kinetic response to moderate intensity exercise in overweight and non-overweight children

CR Potter[1], JK Zakrzewski[2], SB Draper[1] and VB Unnithan[3]

OBJECTIVE: To compare the phase II oxygen uptake time constant ($\tau V'O_2$) and $V'O_2$ mean response time ($V'O_2$MRT) in overweight (OW) and non-OW (NO) children during moderate intensity exercise.

DESIGN: Between subjects where participants completed a maximal ramp exercise test on an electromagnetically braked cycle ergometer to determine peak $V'O_2$ ($V'O_{2peak}$) and gas exchange threshold (GET). Gas exchange was measured breath-by-breath using a mass spectrometer. On subsequent visits, 6 square-wave transitions ($\leqslant 2$ per day) from 0 W to 90% GET were completed. Individual phase II $\tau V'O_2$ and $V'O_2$MRTs were estimated from time aligned average $V'O_2$ traces.

SUBJECTS: Eleven OW (11.8 ± 0.4 years) and 12 NO (11.9 ± 0.4 years) children were recruited to the study. The OW group was significantly heavier (62.9 ± 9.7 vs 39.4 ± 5.8 kg, $P<0.001$), taller (1.58 ± 0.05 vs 1.47 ± 0.07 m, $P<0.001$) and had a higher body mass index (25.8 ± 3.4 vs 18.3 ± 1.8 kg m^{-2}, $P<0.001$).

RESULTS: Both $\tau V'O_2$ (30.2 ± 9.6 vs 22.8 ± 7.1 s, $P<0.05$) and $V'O_2$MRT (43.5 ± 10.7 vs 36.3 ± 5.3 s, $P<0.05$) were significantly slower in OW compared with NO children; absolute $V'O_{2peak}$ was higher in the OW compared with NO group (2.23 ± 0.04 vs 1.74 ± 0.04 l min^{-1}, $P<0.05$); mass relative $V'O_{2peak}$ was lower in OW compared with NO children (35.9 ± 8.3 vs 43.8 ± 6.2 ml kg^{-1} min^{-1}, $P<0.05$); allometrically scaled $V'O_{2peak}$ was similar between OW and NO groups whether relative to body mass$^{0.67}$ (139.8 ± 29.1 vs 147.2 ± 23.9 ml kg^{-67} min^{-1}) or stature^3 (576.0 ± 87.2 vs 544.9 ± 84.9 ml m^{-3} min^{-1}) ($P>0.05$); absolute $V'O_2$ at GET was similar between OW and NO groups (0.94 ± 0.24 vs 0.78 ± 0.27 l min^{-1}, $P>0.05$); GET expressed as percentage of $V'O_{2peak}$ was similar between the groups (42.0 ± 0.1 vs 44.8 ± 0.1%, $P>0.05$).

CONCLUSION: These findings demonstrate impairment in the factors determining $V'O_2$ kinetics in OW children at a relatively young age. Furthermore, assessment of cardiorespiratory fitness using peak exercise values is likely to be misleading and not useful when designing exercise programmes for OW children.

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INTRODUCTION

Various inter-related factors have contributed to the large multi-national increase in numbers of overweight (OW) and obese children.[1] Despite this, it is likely that a complex interplay between excessive energy intake and insufficient energy expenditure is at the root of the problem. Exercise reduces body fat by creating a negative energy balance[2] and, independent of changes in body composition, improves various health markers in children.[3] A clear understanding of cardiorespiratory fitness in OW children is important so that exercise programmes can be tailored to the needs of this population; an impaired cardiorespiratory response to exercise would highlight the need to improve cardiorespiratory fitness, rather than focusing on weight loss alone.

OW and obesity are often associated with reduced exercise tolerance in children.[4] Studies assessing cardiorespiratory exercise responses suggest that OW status is not necessarily associated with cardiorespiratory deconditioning and the decline in functional fitness is primarily attributable to the inert load created by excess body fat,[5] whereas others suggest that a degree of cardiorespiratory deconditioning exists in the OW child.[4] The majority of these studies have focused on maximal or steady-state respiratory responses at submaximal work rates.[5,6] However, children's activity patterns are characterised by short bouts of submaximal non-steady-state activity[7] and are better described by oxygen uptake ($V'O_2$) kinetics. It has been suggested that the phase II time constant of $V'O_2$ ($\tau V'O_2$) provides the best overall index of cardiorespiratory function,[8] reflects the control of the physiological adaptations that occur to meet the increased metabolic demand[9] and provides a non-invasive tool to investigate skeletal muscle oxidative function.[10] Furthermore, because $\tau V'O_2$ is not expressed in relation to body mass, it is a fundamental parameter of aerobic function[11] that facilitates meaningful comparisons between individuals and groups without the confounding effects of mass relative expression.

Slow $V'O_2$ kinetics increases the requirement for anaerobic sources of energy, leading to an early onset of fatigue, and is a marker of impaired exercise tolerance.[8] This is particularly relevant for children because the potential for anaerobic energy provision seems to increase with maturation.[12] For example, children exhibit lower blood lactate levels for a given exercise demand when compared with adults.[12] Children with slow $V'O_2$ kinetics are likely to have reduced exercise tolerance compared with older children or adults with similarly slow kinetics because they have less potential for sustaining exercise anaerobically. Children also have higher levels of oxidative enzymes, such as succinic dehydrogenase and isocitirc dehydrogenase,[13] and a lower

[1]School of Sport and Exercise Sciences, Oxstalls Campus, University of Gloucestershire, Gloucestershire, UK; [2]Department for Health, University of Bath, Bath, UK and [3]Centre for Sport, Health and Exercise Research, Faculty of Health, Staffordshire University, UK. Correspondence: Dr CR Potter, School of Sport and Exercise Sciences, Oxstalls Campus, University of Gloucestershire, Oxstalls Lane, Gloucester GL2 9HW, UK.
E-mail: cpotter@glos.ac.uk
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phosphofructokinase: isocitirc dehydrogenase ratio.[14] Any difference in moderate intensity $V'O_2$ kinetics between OW and non-OW (NO) children would be of particular interest as the immature metabolism seems preferentially disposed to oxidative energy provision.

Despite the usefulness of $V'O_2$ kinetics as an indicator of health and exercise tolerance, few studies have assessed the relationship between weight status and $V'O_2$ kinetics in children. Although the majority of these studies have demonstrated no significant effect of weight status on $V'O_2$ kinetics,[15,16,17] a recent study reported slower $V'O_2$ kinetics across a range of exercise intensities in obese compared with non-obese subjects aged 16–17 years.[18] The findings in this area are not only inconclusive, but also potentially affected by methodological limitations, including an inadequate number of exercise transitions,[16,17,18] inappropriate modelling techniques[15,16] and small study samples.[17] Of the studies that have appropriately modelled the $V'O_2$ kinetic response, they either calculated the mean response time (MRT) using a single exercise transition[17] or used only two exercise transitions to estimate $\tau V'O_2$.[18] In addition, Salvadego *et al.*[18] averaged data every 10 s for kinetics analysis, considerably reducing the number of data points available for modelling (presumably done because of the noisy nature of the second-to-second data from two transitions). The confidence in $\tau V'O_2$ is dependent on noise, $V'O_2$ amplitude and the number of data points used for modelling.[19] Unfortunately in children, noise is high while amplitude is small,[19] both of which combine to drastically reduce the confidence in $\tau V'O_2$ when small numbers of transitions are used. Although the findings of Salvadego *et al.*[18] can be criticised for using few transitions and modelling relatively few data points, nonetheless, differences in $V'O_2$ kinetics between obese and non-obese adolescents were reported. What remains to be determined is whether such a difference exists in younger children.

Consequently, there is a need for well-designed studies to assess the $V'O_2$ kinetic response to exercise in OW and NO children before adolescence. The aim of the present study was to compare the $\tau V'O_2$ and $V'O_2$MRT in OW and NO children aged between 11 and 12 years during moderate intensity exercise.

MATERIALS AND METHODS

Participants

Eleven OW (five girls and six boys) and 12 NO (seven girls and five boys) aged 11–12 years volunteered to participate in the study. Participants were recruited from schools in Gloucestershire, UK. OW status was defined using age- and sex-specific body mass index (BMI) reference points.[20] Written informed consent was obtained from the parents and written assent from the participants. The children were screened using a health history questionnaire; exclusion criteria included: known congenital heart disease, musculoskeletal problems, uncontrolled exercise-induced asthma, diabetes and epilepsy. Stature was measured using a stadiometer (Holtain, Holtain Limited, Dyfed, UK) to the nearest 0.01 m and body mass using an electronic scale (Seca Model 888, Hamburg, Germany) to the nearest 0.1-kg beam. BMI was calculated as body mass (kg) divided by stature squared (m^2). All children were active but not engaged in any systematic exercise training. Ethical approval for the study was obtained from the University of Gloucestershire Research Ethics Committee.

Study design

Participants visited the laboratory a minimum of four times. During the first visit, subjects completed a pre-exercise questionnaire to confirm they were in a postprandial state ($\sim$3 h), had refrained from vigorous physical activity ($\geqslant$12 h) and had not consumed caffeinated products (12 h). Before exercise testing, subjects were familiarised with the exercise test protocol, breathing valve apparatus and cycle ergometer and instructed to maintain pedalling cadence of 60 rev min^{-1} for all tests. The seat and handlebar height were adapted for each child and maintained during subsequent tests. Following habituation, participants completed a maximal ramp ($V'O_{2peak}$) exercise test; on subsequent visits ($\geqslant$4 days), participants completed identical square-wave exercise tests at moderate intensity.

Maximal exercise test

Subjects performed a ramp test to voluntary exhaustion to determine (1) $V'O_{2peak}$ and (2) the gas exchange threshold (GET). Following a 2-min warm-up of unloaded pedalling, the test began at a power output of 30 W and increased by 12 W min^{-1} to attain a test that lasted a maximum of 15 min in duration. The children were encouraged to continue until voluntary exhaustion. In the absence of a $V'O_2$ plateau, $V'O_{2peak}$ was recorded as the highest 15 s moving average $V'O_2$ where at least two of the secondary criteria were met. Secondary criteria for the attainment of $V'O_{2peak}$ were heart rate levelling off at $\sim$200 beats min^{-1} and a respiratory exchange ratio of $\geqslant$1.00, in addition to the subject demonstrating clear subjective symptoms of fatigue.[12] GET was determined using the V-slope method.[21] Data were smoothed using a 15-s sequential average, systematically dissecting the $V'CO_2/V'O_2$ data and plotting linear regression lines using all data either side of that point.

Square-wave exercise test

Square-wave exercise tests consisted of 3-min unloaded cycling (0 W) followed by 6-min cycling at a work rate equivalent to 90% of individual GET. A maximum of two transitions were completed on any 1 day and $V'O_2$, $V'CO_2$, V'_E and heart rate had returned to resting values before a transition was repeated.

Apparatus

All exercise tests were performed on an electromagnetically braked cycle ergometer (Excalibur Sport, Lode, The Netherlands). Gas exchange was measured on a breath-by-breath basis using a quadrupole mass spectrometer (MSX671, Morgan Medical, Rainham, UK) from a sample drawn continuously from the mouthpiece at 1 ml s^{-1}. The mass spectrometer was calibrated using a gas mixture of known composition (Linde Gas Ltd, Stoke-On-Trent, UK). Expired air volume was measured using a turbine flow metre (Interface Associates, Laguna Niguel, CA, USA) with a dead space volume of 90 ml. Volume calibration was achieved using a multiflow 3-l volume calibration syringe (Pulmonary Data Services Instrumentation, Louisville, CO, USA) over a range of flow speeds. Heart rate was monitored throughout exercise tests using short-range telemetry (Polar Vantage, Polar, Kempele, Finland). All calibration procedures were carried out before each exercise test.

Mathematical modelling

Breath-by-breath data for the phase 1 duration criteria (end-tidal partial pressure of O_2 ($ETPO_2$), end-tidal partial pressure of CO_2 ($ETPCO_2$) and respiratory exchange ratio) and for $V'O_2$ from each of the six transitions were interpolated into 1-s intervals, time aligned and averaged for each subject. The end of phase 1 was determined visually from the averaged response profile, as the time when a decrease $ETPO_2$ with a simultaneous increase in $ETPCO_2$ and a sharp decrease in the respiratory exchange ratio occurred.[22] The averaged $V'O_2$ response profile was used to derive the parameters of the $V'O_2$ kinetic response.

The phase II $\tau V'O_2$ for each individual was estimated by fitting an exponential equation (equation 1) to the averaged $V'O_2$ response profile for each individual using least squares nonlinear regression analysis. This included a delay term (δ) and was applied to a fitting window constrained to start after the end of phase I. The $V'O_2$MRT was calculated by fitting a single exponential to the entire response ($t > 0$ s) using equation 2. Baseline $V'O_2$ (BL $V'O_2$) was calculated as the average $V'O_2$ during the final 60 s of unloaded pedalling.

$$\Delta V'O_{2(t)} = \Delta V'O_{2(ss)} \bullet (1 - (e^{-(t-\delta)/\tau})) \quad (1)$$

$$\Delta V'O_{2(t)} = \Delta V'O_{2(ss)} \bullet (1 - (e^{-t/\tau})) \quad (2)$$

$\Delta V'O_{2(t)}$—the increase of $V'O_2$ at time t above the 0 watt baseline; $\Delta V'O_{2(ss)}$—the increase of $V'O_2$ at steady state, t—time, e—natural logarithm, δ—the delay term; τ—time constant.

Normalisation of maximal data

To compare $V'O_{2peak}$ between groups, comparisons were made in absolute terms (l min^{-1}), in ratio with body mass (ml kg^{-1} min^{-1}), to the 0.67 power of body mass (ml $kg^{-0.67}$ min^{-1}) (see reference Armstrong and Welsman[12] for further details of allometric scaling methods) and to stature cubed (ml m^{-3} min^{-1}) in accordance with Unnithan *et al.*[17]

Statistical analyses

As Fawkner *et al.*[23] demonstrated no significant sex differences in children's $V'O_2$ kinetics during moderate intensity exercise, boys' and girls' data were combined. Statistical analyses and data modelling were completed using the Statistical Package for the Social Science (SPSS) software version 14.0 for Windows (SPSS Inc., Chicago, IL, USA). Shapiro-Wilk and Levene's tests were used to verify normal distribution and homogeneity of variance, respectively. The assumptions of parametric data were met and Student's independent *t*-tests were used to compare mean OW and NO values for participant characteristics, maximal exercise test responses and the $V'O_2$ kinetic response parameters. Values are expressed as mean ± s.d. and given to three significant figures. Statistical significance was accepted at $P \leqslant 0.05$.

RESULTS

Participant characteristics

Participant characteristics are displayed in Table 1. BMI, body mass and stature were greater ($P < 0.001$) in the OW compared with NO children, whereas age was similar between the two groups ($P > 0.05$).

Maximal exercise test responses

Absolute $V'O_{2peak}$ ($l\,min^{-1}$) was higher in the OW compared with NO group ($P < 0.05$). When expressed in ratio with body mass, $V'O_{2peak}$ ($ml\,kg^{-1}\,min^{-1}$) was lower in the OW compared with NO children ($P < 0.05$). There was no significant difference between the groups when $V'O_{2peak}$ was expressed to the 0.67 power of body mass ($ml\,kg^{-0.67}\,min^{-1}$) or when expressed in relation to stature cubed ($ml\,m^{-3}\,min^{-1}$) ($P > 0.05$). The GET was successfully determined in all participants. Absolute $V'O_2$ at GET ($l\,min^{-1}$) was similar between OW and NO groups ($P > 0.05$). The $V'O_2$ at GET expressed relative to body mass ($ml\,kg^{-1}\,min^{-1}$) was lower in OW compared with NO children ($P < 0.05$). There was no difference in GET as a percentage of $V'O_{2peak}$ between OW and NO children ($P > 0.05$). Values are presented in Table 2.

Kinetic responses to square-wave exercise tests

The $V'O_2$ kinetic response parameters are displayed in Table 3. Phase I duration was similar between OW and NO children ($P > 0.05$). Baseline $V'O_2$ ($l\,min^{-1}$) was higher in the OW compared with NO group ($P < 0.01$). The $\tau V'O_2$ ($P < 0.05$) and $V'O_2$MRT ($P < 0.05$) were slower in OW compared with NO children. The $V'O_2$ response profiles and model fits for the estimation of $\tau V'O_2$ for representative OW and NO children are shown in Figures 1 and 2, respectively.

DISCUSSION

The main finding of the present study was that the $V'O_2$ kinetic response to moderate intensity exercise was slower in OW 11- to 12-year-old children compared with NO children, as reflected by both the shorter $\tau V'O_2$ and $V'O_2$MRT in the OW group compared to the NO children, in spite of the OW group having a higher absolute peak aerobic power.

Table 1. Subject characteristics

	OW (n = 11) (6 M, 5 F)	*NO (n = 12) (5 M, 7 F)*
Age (years)	11.8 ± 0.4	11.9 ± 0.4
Body mass (kg)	62.9 ± 9.7	39.4 ± 5.8*
Stature (m)	1.58 ± 0.05	1.47 ± 0.07*
BMI ($kg\,m^{-2}$)	25.8 ± 3.4	18.3 ± 1.8*

Abbreviations: BMI, body mass index; F, female; M, male; NO, non-overweight; OW, overweight. Values are mean ± s.d., expressed to three significant figures. *Denotes significant difference ($P \leqslant 0.001$).

Our findings are in agreement with a recent study that reported slower $V'O_2$ kinetics across a range of workloads in obese compared with non-obese adolescents.[18] This was despite that study using only two transitions and intra-test averaging, which yielded few non-steady-state data points for modelling.

Table 2. Maximal progressive exercise test responses for OW and NO subjects

	OW (n = 11) (6 M, 5 F)	*NO (n = 12) (5 M, 7 F)*
$V'O_{2peak}$ ($l\,min^{-1}$)	2.23 ± 0.41	1.74 ± 0.40*
$V'O_{2peak}$ ($ml\,kg^{-1}\,min^{-1}$)	35.9 ± 8.3	43.8 ± 6.2*
$V'O_{2peak}$ ($ml\,kg^{-67}\,min^{-1}$)	139.8 ± 29.1	147.2 ± 23.9
$V'O_{2peak}$ ($ml\,m^{-3}\,min^{-1}$)	576.0 ± 87.2	544.9 ± 84.9
$V'O_2$ at GET ($l\,min^{-1}$)	0.94 ± 0.24	0.78 ± 0.27
$V'O_2$ at GET ($ml\,kg^{-1}\,min^{-1}$)	15.1 ± 4.4	19.7 ± 5.1*
GET % $V'O_{2peak}$	42.0 ± 0.1	44.8 ± 0.1

Abbreviations: F, female; GET % $V'O_{2peak}$, percentage of peak $V'O_2$ at which the gas exchange threshold occurred; M, male; NO, non-overweight; OW, overweight; $V'O_{2peak}$, peak oxygen uptake; $V'O_2$ at GET, $V'O_2$ at the gas exchange threshold. Values are mean ± s.d. *Denotes significant difference ($P \leqslant 0.05$).

Table 3. Kinetic responses for OW and NO subjects

	OW (n = 11) (6 M, 5 F)	*NO (n = 12) (5 M, 7 F)*
Phase I (s)	17.3 ± 4.6	17.4 ± 4.4
[a]$\tau V'O_2$ (s)	30.2 ± 9.6	22.8 ± 7.1*
[a]$V'O_2$amplitude ($l\,min^{-1}$)	0.81 ± 0.20	0.66 ± 0.16
[a]Delay (s)	11.2 ± 2.6	13.0 ± 4.3
[b]$V'O_2$MRT (s)	43.5 ± 10.7	36.3 ± 5.3*
[b]$V'O_2$ amplitude ($l\,min^{-1}$)	0.82 ± 0.20	0.67 ± 0.16
BL $V'O_2$ ($l\,min^{-1}$)	0.62 ± 0.13	0.45 ± 0.08**

Abbreviations: BL $V'O_2$, average $V'O_2$ during final 60 s of unloaded pedalling; Delay, delay time; F, female; M, male; NO, non-overweight; OW, overweight; Phase I, phase I duration as determined according to Mettauer *et al.*[12] criteria; $\tau V'O_2$, time constant for oxygen uptake; $V'O_2$ amplitude, change in mean steady-state oxygen consumption from baseline pedalling to enforced work rate; $V'O_2$MRT, mean response time for $V'O_2$. Values are mean ± s.d., expressed to three significant figures. *Denotes significant difference ($P \leqslant 0.05$). **Denotes significant difference ($P \leqslant 0.01$). [a]Denotes using a single exponential model with delay fit following phase I. [b]Denotes using a single exponential model fit to the entire response.

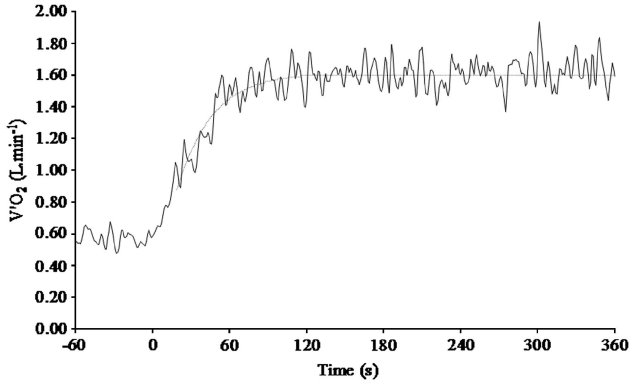

Figure 1. $V'O_2$ response (solid line) and model fit (dotted line) for the estimation of $\tau V'O_2$ for a representative OW subject ($\tau = 27.5$ s; $\delta = 9.5$ s).

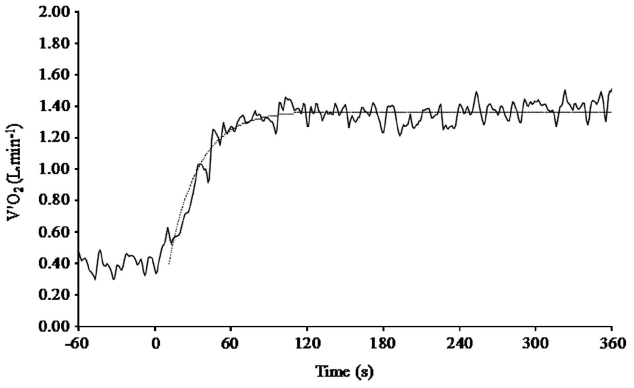

Figure 2. $V'O_2$ response (solid line) and model fit (dotted line) for the estimation of $\tau V'O_2$ for a representative NO subject ($\tau = 23.3$ s; $\delta = 10.9$ s).

The present study extends these findings to OW children aged 11–12 years, suggesting that slower $V'O_2$ kinetics are manifest at a younger age and at a lesser degree of OW status. The findings of the current study are further strengthened by multiple transitions being averaged together prior to modelling. Our findings are partially supported by those of Unnithan *et al.*,[17] who despite reporting a non-significant difference in $V'O_2$MRT between OW (52.6 ± 11.7 s) and NO (45.6 ± 7.4 s) children aged between 11 and 12 years, reported a clear trend for slower kinetics in the OW group (presumably this difference did not attain significance because of the high inter-subject variability). The findings of the current study are at odds with some other previous research investigating $V'O_2$ kinetics in OW and NO children however. Cooper *et al.*[15] reported no significant difference in $\tau V'O_2$ between obese (29 ± 9 s) and non-obese (28 ± 6 s) children. Although subjects performed multiple transitions, inappropriate modelling strategies were used and exercise was initiated from a resting baseline, rather than unloaded pedalling, which is not optimal for the modelling of the response.[24] Loftin *et al.*[16] reported that $\tau V'O_2$ did not differ between 'severely' OW (33.9 ± 22.7 s) and NO (41.5 ± 21.3 s) adolescents. However, subjects only completed one exercise transition and gas exchange responses were recorded every 10 s. The findings presented here also conflict with studies reporting that $V'O_2$ kinetics in OW and NO adults[25] and O_2 deficit in obese and non-obese children[26] are not significantly different.

The $V'O_2$ kinetic response reflects the efficiency of the cardiorespiratory system to adapt to an increased metabolic demand and provides an indication of cardiorespiratory function that is numerically independent of body mass. The evidence presented here demonstrates that the $V'O_2$ kinetic response is slowed in OW children, which implies that factors determining the speed of the $V'O_2$ kinetic response are impaired in this population. The increase in $V'O_2$ during phase I is mainly attributed to the increase in cardiac output resulting in increased pulmonary blood flow.[27] As the phase I response duration was similar between groups (OW 17.3 ± 4.7 s, NO 17.4 ± 4.4 s), it is unlikely that such factors would explain differences in the $V'O_2$ kinetic response between groups. Alternatively, factors determining the speed of the phase II response may be impaired in the OW child. The speed of this response is dependent on a number of factors related to the rate of O_2 delivery to the exercising muscles[28] and the potential of the muscle to utilse O_2.[29] However, breath-by-breath measurements at the mouth are not sensitive enough for the accurate discrimination of such factors. More direct methods are required to investigate these factors, such as magnetic resonance spectroscopy and near-infrared spectroscopy.

We found that OW children had significantly higher peak aerobic power in absolute terms than NO children, and conversely, OW children had significantly lower peak aerobic power when expressed in ratio with body mass. This implies body fat acts as an inert load during exercise and that parameters of peak cardiorespiratory function are decreased when expressed per kg largely due to body fat inflating the denominator. As $V'O_{2peak}$ is strongly correlated with body size,[12] OW children may have similar or higher levels of peak cardiorespiratory power than NO children simply because of their greater body size when absolute values are compared, whereas expression in ratio to body mass may disadvantage OW individuals. To further investigate this issue, we scaled $V'O_{2peak}$ to the 0.67 power of body mass,[12] and to stature cubed[17] as the OW children were also significantly taller than the NO group. This further analysis served to demonstrate the problems associated with using $V'O_{2peak}$ to assess cardiorespiratory function in OW subjects. Scaling $V'O_{2peak}$ to the 0.67 power of body mass indicated similar levels of peak aerobic power between the groups, as did scaling to height cubed, contradicting the kinetics data. The advantage of using the phase II $\tau V'O_2$ to assess cardiorespiratory function is that it facilitates comparison without needing to scale for body size and thus may be a more reliable indicator of cardiorespiratory function than $V'O_{2peak}$. Indeed, if the peak data alone were used as the criteria for assessing cardiorespiratory health in this study, then depending on the method of expression chosen, the OW group may have been considered to exhibit superior levels of cardiorespiratory function to the NO group (absolute $V'O_{2peak}$), inferior ($V'O_{2peak}$ in ratio with body mass), or similar ($V'O_{2peak}$ scaled to the 0.67 power of body mass and relative to height cubed), when the kinetics data are unequivocal that there is cardiorespiratory impairment in the OW subjects.

Many studies indicate that OW children participate in less activity,[30] particularly moderate to vigorous physical activity,[31] and more sedentary behaviour[32] than NO children. Reduced physical activity levels may depress cardiorespiratory fitness in obese individuals[33] and refraining from exercise may reduce fitness further. Therefore, a potential explanation for the slowed $V'O_2$ kinetic response in the OW child may be that cardiorespiratory fitness, as reflected by $V'O_2$ kinetics, acts as a proxy indicator of low physical activity levels. However, the lack of detailed physical activity data in the present study precludes further examination of this point.

The present study was the first to measure both the $\tau V'O_2$ and the $V'O_2$ MRT in 11- to 12-year-old OW and NO children and has improved on the procedures used in previous studies to increase the confidence with which these parameters can be estimated. The exercise protocol used in the present study included a maximal exercise test followed by six transitions from unloaded pedalling to 90% GET. A maximal test followed by a number of identical transitions has been considered the 'gold standard' approach to assessing $V'O_2$ kinetics.[9] This study design facilitated adherence to the moderate intensity exercise domain,[8] as the individual work rates for the square-wave tests were calculated as a standard percentage of the individual GET. A previous study by Salvadego *et al.*[18] did not prescribe work rates according to GET, although their lower exercise intensity—40% $V'O_{2max}$—was reported to be below their subjects' GETs. Both OW and NO subjects in the current study reached GET at a slightly higher mean percentage of $V'O_{2peak}$ than that reported by Salvadego *et al.*[18] A high percentage of GET was selected in the present study to maximise the amplitude of the $V'O_2$ response. We also used unloaded pedalling as a baseline level, rather than rest, which is preferable for modelling the kinetic response.[24] The respiratory responses were measured on a breath-by-breath basis using mass spectrometry, which is most appropriate for assessing the time course of the $V'O_2$ response and can be applied in children with an acceptable degree of validity and reproducibility.[34]

The inclusion of six exercise transitions reduces respiratory noise and increases the confidence with which the kinetic parameters can be estimated.[19] This is particularly important when investigating child populations, as it has been shown that children produce a greater amount of noise than adults.[19] Lastly, phase I duration was individually assessed using physiological variables.[22] Other studies have assumed phase I to be constant between individuals,[15,16,18] an assumption that is likely to be subject to error and does not account for individual differences. Indeed, the average phase I duration for the OW and NO group in the present study was 17.3 ± 4.6 s and 17.4 ± 4.4 s, respectively, and there was high inter-subject variability as indicated by the s.d's.

The findings from the present study also have a number of practical implications relating to exercise regimens in the OW child. The slowed $V'O_2$ kinetic response in the OW child implies a reduced exercise tolerance. A weight loss programme incorporating a combination of exercise and diet may therefore be optimal, with the focus being on both to improve cardiorespiratory fitness and to reduce body mass. In contrast, individual OW children not demonstrating a slowed $V'O_2$ kinetic response to exercise may not be more susceptible to the early onset of fatigue during this type of exercise. Thus, there is no *a priori* reason to impose a medical restriction preventing these individuals from being fully active and weight loss regimens may be tailored with the aim of increasing caloric expenditure, rather than improving cardiorespiratory fitness. This may be achieved using lower intensity exercise, which is preferable, as limiting exercise intensity may improve subject compliance[35] and facilitate the use of greater exercise durations.

We did not report confidence intervals for $\tau V'O_2$ here. The reason for this is the questionable accuracy of confidence intervals generated by commercially available software. Motulsky and Ransnas[36] point out that the nature of the errors in nonlinear functions means that: '...values are based on linearising assumptions and will always underestimate the true uncertainty of any nonlinear equation' (p. 370). Alternative approaches for calculating confidence intervals are beyond the scope of the current study and the reader is referred to Motulsky and Ransnas[36] for further details of their recommendations.

The BMI reference points set by Cole *et al.*[20] used to define OW status have advantages over BMI centiles[37] and the non-invasive nature of this measure is appropriate for children. Although BMI correlates with more direct measures of fatness, it does not differentiate between muscle and fat. However, in a study using a large sample, Mei *et al.*[38] found that BMI performed well in identifying OW status when compared with sum of triceps and subscapular skinfold thicknesses and dual-energy X-ray absorptiometry. Indeed, Daniels[39] described BMI as: '…the most useful method for assessing adiposity in the clinical setting' in children and adolescents (p. S39).

Our study was limited to the moderate intensity domain where the kinetic response is appropriately modelled by a single exponential plus delay model.[24] The nature of the $V'O_2$ kinetic response in OW young children at higher exercise intensities is yet to be examined in detail. Well-designed studies are needed to investigate whether the difference identified here during moderate intensity exercise between OW and NO subjects persists into higher exercise intensities. This will be particularly challenging as $V'O_2$ kinetics are more complex above the GET, consisting of both a fast primary component and a slowly emerging secondary component.[8] The total response amplitude in young children is small, making differentiation of primary and secondary response components technically challenging.

A further limitation of the present study was that we did not precisely quantify the physical activity levels of our subjects. This meant it was not possible to investigate the relationship between physical activity and moderate intensity $V'O_2$ kinetics. There is strong and consistent evidence detailing the negative relationship between levels of physical activity and OW status.[40] What is not clear is whether being OW affects $V'O_2$ kinetics independently of physical activity levels. Future studies may wish to try to unpick this relationship, to determine whether slower $V'O_2$ kinetics in OW children can be attributed to low levels of physical activity or whether the aetiology is more complex.

In conclusion, this study demonstrates that OW children aged 11–12 years exhibit slower $V'O_2$ kinetic responses compared to NO children during moderate intensity cycling, in spite of the OW group having a higher absolute peak aerobic power. These findings give cause for concern because they demonstrate impairment in the factors determining $V'O_2$ kinetics in OW children at a relatively young age and at a stage of maturity where children are particularly suited to aerobic metabolism. Furthermore, the findings suggest that assessment of cardiorespiratory fitness using peak exercise data is likely to be misleading and not useful when designing exercise programmes for OW children.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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