COMPARISON OF OXYGEN UPTAKE KINETICS AND OXYGEN DEFICIT IN SEVERELY OVERWEIGHT AND NORMAL WEIGHT ADOLESCENT FEMALES

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Received: 27 June 2005 / Accepted: 29 August 2005 / Published (online): 01 December 2005

ABSTRACT

The purpose of this study was to determine if differences in oxygen uptake kinetics and oxygen deficit existed between normal weight and severely overweight adolescent girls. Subjects included 10 normal weight and 8 severely overweight girls. The participants performed a leg cycling VO2 peak test and a constant load leg cycling test at 80% of the ventilatory threshold (T-vent). In the constant workload test O2 kinetics as indicated by Phase I (VO2 L at 20 sec) and Phase II time constants (τ) were determined. Also, the O2 deficit (VO2 L) was measured. As expected significant differences were noted in body composition and VO2 peak relative to mass with normal weight body mass averaging 55.3 ± 7.0 kg, severely overweight 90.5 ± 18.0 kg, % fat normal weight 27.3 ± 3.9%, severely overweight 49.7 ± 4.9% and VO2 peak (ml·kg⁻¹·min⁻¹) normal weight 32.0 ± 2.7 and severely overweight 22.0 ± 5.3. VO2 peak (l·min⁻¹) and T-vent (%VO2 max) were similar between groups. Results revealed similar O2 kinetic responses between groups; phase I kinetics normal weight 0.72 ± 0.15 L; severely overweight 0.75 ± 0.13L, phase II (τ) normal weight 41.5 ± 21.3 sec; severely overweight 33.9 ± 22.7 sec. However, the O2 deficit was significantly higher in the severely overweight (0.75 ± 0.15L) when compared to the normal weight group (0.34 ± 0.13L). Correlations ranged from r = -0.15 to 0.51 between VO2 peak (L·min⁻¹) or fat weight and phase I, τ and O2 deficit. These data generally support previous research concerning the independence of O2 uptake response and body size.

KEY WORDS: O2 kinetics, O2 deficit, severely overweight, female, youth.

INTRODUCTION

Volume of oxygen uptake (VO2) is an indirect measure of cellular respiration and energy expenditure and fundamentally is the product of cardiac output and the uptake of oxygen at the cellular level (a-vO2 diff). The abrupt rise in VO2 when moving from a resting to a dynamic exercise steady-state has been identified as oxygen uptake kinetics. Typically, VO2 will continue to increase for three to five minutes before steady-state occurs (Poole et al., 1991). Oxygen uptake kinetic research has primarily focused on the kinetic response of apparently healthy adults and children (Armon et al., 1991; Cooper et al., 1984; 1985; Poole et al., 1991). In comparative studies, some researches have found children to reach steady-state faster than adults (Armon et al., 1991) although Hebestreit et al. (1998) observed similar responses. Williams et al. (2001) found similar τ responses below the lactate...
threshold and different responses above the lactate threshold when comparing boys and men. Limited research though has focused on the $O_2$ kinetic response of severely overweight children (Cooper, et al., 1990), specifically phase I and II kinetics and the $O_2$ deficit, even though childhood obesity has been increasing in the world over the past decade (Flegal, 1999; James et al., 2001).

The oxygen uptake kinetic response can be defined by three phases or stages. Phase I can be defined as the first twenty seconds of the metabolic response to exercise. Gaesser and Poole (1996) define phase I as increased VO$_2$ primarily due to augmented cardiac output and pulmonary blood flow. The phase II kinetic time constant ($\tau$) response is the time to reach 63% of VO$_2$ steady-state and is characterized by an exponential rise in VO$_2$ just after the beginning of phase I (Barstow et al., 1994). Increased venous return from the exercising muscle as well as continued pulmonary blood flow denote phase II kinetics. Gaesser and Poole (1996) also suggested that the phase II kinetic response is relatively constant in the transition from rest to steady-state during exercise of light to moderate intensity (below the lactate threshold). Moreover, increased levels of physical fitness (VO$_2$ max) may lead to a faster $\tau$. Also, $\tau$ may be delayed as a result of increased exercise intensity and probably facilitates an oxygen drift (Gaesser and Poole, 1996).

The oxygen deficit is the difference between the total energy cost of work, assuming steady-state throughout the entire exercise bout, and the measured portion of the total energy expenditure that was met during the exercise period by aerobic energy production (McArdle et al., 2001). The $O_2$ deficit can be directly linked to steady-state VO$_2$. Therefore, if steady-state is attained more rapidly a lower oxygen deficit would be noted.

Typically, obese individuals including children are assumed to be unfit. However research by Cooper, et al. (1990) found that obesity was not an indicator of poor fitness. Moreover, they suggested that cardiorespiratory testing could be utilized to investigate serious physiological impairment and to individualize treatment in the severely overweight child.

The purpose of this study was to determine if severely overweight adolescent girls respond differently than normal weight adolescent girls in regard to $O_2$ kinetics as well as $O_2$ deficit below the ventilatory threshold (T-vent). Also the association of fitness (VO$_2$ peak L·min$^{-1}$), fatness (fat mass kg) and $O_2$ kinetics and $O_2$ deficit were examined.

**METHODS**

**Subjects**

Ten normal weight and eight severely overweight girls, 11 – 15 years of age, volunteered to participate. The normal weight girls were recruited from a local Parochial school and the severely overweight participants from the Committed to Kids program (prior to participation) at Louisiana State University Health Sciences Center. The study was approved by the respective university committees for the use of human subjects. Informed consent was completed by the parents and the girls completed an assent form.

**Apparatus**

A Preference HRT-2000i cycle ergometer was used for all exercise testing. A Sensormedics, model 2900c or a Parvomedics TrueOne 2400 metabolic cart was used to measure oxygen uptake, carbon dioxide, and pulmonary ventilation. Prior to testing, the metabolic system was calibrated according to manufacturers’ specifications. A Polar heart rate monitor was used to measure heart rate throughout testing and Dual Energy X-Ray Absorptiometry (DEX) (Lunar-GE DPX) was utilized to measure body composition. Body mass index (BMI) was expressed as body mass kg·m$^{-2}$. The BMI percentiles for seven of the eight overweight girls was >95% based on age. The group was therefore classified as severely overweight.

**Procedures**

Each subject reported to the laboratory for two separate metabolic tests. Day one included a VO$_2$ peak test and body composition assessment. Each subject was given a period of time for familiarization with the cycle ergometer. After familiarization, resting data were collected for two minutes. The peak test began at an initial power output of 20 watts. The subjects then followed a ramp protocol for achieving the VO$_2$ peak data. The resistance of the cycle ergometer was increased each minute in 10 watt (W) increments until volitional fatigue. The cycling was maintained at a 60 rpm rate. The resistance was set in increments of 10W so that fatigue of the severely overweight children would not hinder their VO$_2$ peak data. The ventilatory threshold was calculated by the V-slope method (Beaver et al., 1986). The second day of testing (at least 3 days following the peak test) included the constant work rate cycling test at 80% of T-vent. For this test, metabolic parameters were recorded at 10 second intervals for the test duration. The phase I $O_2$ kinetic response was the VO$_2$ (L·min$^{-1}$) at 20 seconds after exercise began. The phase II ($\tau$) response was determined from the following equation: $\Delta$ VO$_{2ss}$ (t) = $\Delta$VO$_{2ss}$ x (1 - e$^{-t/\tau}$), where $\Delta$ VO$_{2ss}$(t); was the increase in VO$_2$ above baseline.
Table 1. Physical characteristics of the participants. Data are means (±SD).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal Weight</th>
<th>Severely overweight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 10)</td>
<td>(n = 8)</td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>14.4 (.3)</td>
<td>16.3 (2.8)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.48 (.04)</td>
<td>1.49 (.09)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>55.3 (7.0)</td>
<td>90.5 (18.0) *</td>
</tr>
<tr>
<td>BMI (kg·m⁻²)</td>
<td>25.2 (2.5)</td>
<td>41.2 (9.2) *</td>
</tr>
<tr>
<td>Fat Weight (kg)</td>
<td>14.4 (3.3)</td>
<td>39.5 (8.2) *</td>
</tr>
<tr>
<td>% Fat</td>
<td>27.3 (3.9)</td>
<td>47.9 (4.9) *</td>
</tr>
<tr>
<td>Fat-Free Body Mass (kg)</td>
<td>40.2 (4.5)</td>
<td>45.5 (6.2)</td>
</tr>
</tbody>
</table>

* p < 0.05.

Table 2. Peak exercise responses. Data are means (±SD).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal Weight</th>
<th>Severely overweight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 10)</td>
<td>(n = 8)</td>
<td></td>
</tr>
<tr>
<td>VO₂ (L·min⁻¹)</td>
<td>1.91 (.39)</td>
<td>1.91 (.24)</td>
</tr>
<tr>
<td>VO₂ (ml·kg⁻¹·min⁻¹)</td>
<td>32.0 (2.7)</td>
<td>22.0 (5.3) *</td>
</tr>
<tr>
<td>VCO₂ (L·min⁻¹)</td>
<td>2.26 (.39)</td>
<td>2.13 (.32)</td>
</tr>
<tr>
<td>RER</td>
<td>1.18 (.06)</td>
<td>1.12 (.10)</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>192.4 (9.4)</td>
<td>183.0 (12.8)</td>
</tr>
<tr>
<td>T-vent (% VO₂ peak)</td>
<td>62.8 (9.6)</td>
<td>61.7 (7.6)</td>
</tr>
<tr>
<td>VE_BTPS (L·min⁻¹)</td>
<td>81.9 (13.1)</td>
<td>73.3 (13.0)</td>
</tr>
<tr>
<td>Power Output (W)</td>
<td>110.0 (16.3)</td>
<td>93.8 (31.5)</td>
</tr>
</tbody>
</table>

* p < 0.05.

VO₂ at any time (t); Δ VO₂ss was the difference between phase I VO₂ at the 20-second mark and steady-state VO₂ (phase III); e was natural logarithm; and τ was the time to reach 63% of Δ VO₂ss (Cooper et al., 1985). Oxygen uptake response in the transition from rest to steady-state exercise in a severely obese and normal weight adolescent female was presented in Figure 1.

The O₂ deficit was calculated by subtracting the total energy cost (VO₂ L) of the constant workload test from the energy cost of assumed steady-state VO₂ for the test duration (McArdle et al., 2001). Since steady-state is typically not reached for three to five minutes (Gaesser and Poole, 1996) the O₂ deficit indicates a portion of the energy was supplied by anaerobic sources.

Prior to the constant workload test, resting data were collected for ten minutes with the last two minutes used for analysis. Following rest the constant work test began and continued for six minutes to steady-state. Subjects were instructed not to eat or exercise for 4 hours preceding each exercise test.

Statistics
Independent t-tests were used to compare group responses. Product moment correlation coefficients were used to determine association of variables. The SPSS statistical (version 11.0) program was utilized and the alpha level for all tests was set at the p < 0.05.

RESULTS

Physical characteristics for the subjects can be found in Table 1. As noted, body mass, fat weight, % fat and BMI were significantly higher (p < 0.05) in the severely overweight as compared to the normal weight girls. Peak responses are reported in Table 2. VO₂ peak expressed in absolute units were similar between groups as the severely overweight and normal weight girls averaged 1.91 L·min⁻¹. Since the severely overweight girls body mass was about 40 kg higher than the normal weight girls, the VO₂ peak relative to body mass were significantly lower in the severely overweight girls. However T-vent, expressed as a percentage of VO₂ peak were similar between groups. Table 3 shows the oxygen uptake kinetic and oxygen deficit group comparisons. Phase I and τ kinetic responses were similar between groups while the O₂ deficit was significantly higher in the severely overweight (0.75 L) as compared to the normal weight group (0.34 L). In Table 4 bivariate correlations between the oxygen uptake kinetic responses or oxygen deficit and VO₂ peak or fat weight (kg) are reported. As indicated, correlations ranged from – 0.15 to 0.51.

DISCUSSION

Comparing the initial exercise response of a constant workload submaximal test in children to other
individuals has most typically resulted in comparing children to adults (Armon et al., 1991; Fawkner et al., 2002; Hebestreit et al., 1998; Williams et al., 2001; Zanconato et al. 1991) or children to older youth (Cooper et al., 1985). Limited research effort has examined diseased (Mocellin et al., 1999) or severely overweight youth (Cooper et al., 1990).

Table 4. Bivariate correlation coefficients relating VO2 peak or fat weight with O2 kinetics and O2 deficit.

<table>
<thead>
<tr>
<th>Variable</th>
<th>VO2 peak (L·min⁻¹)</th>
<th>Fat Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase I</td>
<td>.06</td>
<td>.08</td>
</tr>
<tr>
<td>Phase II (τ)</td>
<td>.26</td>
<td>-.15</td>
</tr>
<tr>
<td>O2 deficit (L)</td>
<td>.51</td>
<td>.35</td>
</tr>
</tbody>
</table>

The protocol used in the current study was adopted from a series of studies by Cooper and colleagues (Armon et al., 1991; Cooper et al., 1985; Zanconato et al. 1991). We observed no differences (p < 0.05) in phase I or τ kinetic responses when comparing severely overweight female youth to normal weight female youth. In the work of Cooper and colleagues the investigators compared the O2 kinetic response in children to youth or adults while exercising between 50 to 80% of the ventilatory (anaerobic) threshold. Cooper et al. (1985) compared oxygen uptake and heart rate kinetics in young children (average age 8.6 yrs) to older youth (17.4 yrs). The participants performed cycle ergometry at intensities equaling 75 % of the ventilatory threshold. Phase I O2 kinetics was expressed as a percentage of VO2 peak. Results indicated that the older youth worked at a higher percentage of max (63.5%) as compared to the children (42.5%). Phase II responses were similar between groups and Cooper et al. (1985) suggested that the kinetics of VO2 were independent of body size and age during growth when exercising at intensities below the ventilatory threshold. In two related studies, Armon et al., (1991) and Zanconato et al. (1991) compared the O2 kinetic response in children to adults. Armon et al. observed different τ with the children adjusting more quickly to high intensity constant work test than the adults. The average O2 cost (ml O2 · min⁻¹·W⁻¹) was also greater in the children as compared to the adults. Zanconato et al. (1991) examined the O2 cost (integral of VO2 above baseline) during exercise (1 min bouts) and recovery in children and adults. The O2 cost was independent of work intensity and was higher in children only during exercise above the ventilatory (anaerobic) threshold.

In more recent work, Fawkner et al. (2002) observed that children had a faster τ response than adults. Moreover, no gender differences were evident in either the children or adults. Hebestreit et
al., (1998) and Williams et al. (2001) found no differences in \( \tau \) when comparing boys to men when exercising at either 50% of VO2 peak (Hebestreit 1998) or 80% of VO2 at lactate threshold (Williams et al. 2001). However, during heavier work (50% of the diff between VO2 at lactate threshold and VO2 max) \( \tau \) was faster in the boys. Fawkner and Armstrong (2004) observed no statistical differences when using a mono-exponential model as compared to double-exponential model in finding \( \tau \) during moderate intensity constant load work tests in children. However, \( \tau \) occurred earlier for the boys when compared to the girls. The second component of the double-exponential model is typically identified as the slow component of VO2. Thus, the slow component of VO2 was not confounding the model determination. Typically, the slow component is observed in workloads above the lactate threshold (Fawkner and Armstrong, 2003; Gaesser and Poole, 1996).

In earlier research, Sady, et al. (1983) found no child – adult differences in the \( \frac{1}{2} \) time response to steady-state. Some of the discrepancies in findings are probably due to different methods employed to observe initial responses to constant work tests. For example, Reybrouck et al. (2003) observed that increasing treadmill elevation (intensity) resulted in slower O2 kinetic responses. Moreover, Hebestreit et al. (1998) suggested eliminating phase I from the determination of \( \tau \).

In the only study that we have found that examined severely obese youth, Cooper et al. (1990) studied O2 uptake kinetics in youth (13.4 ± 2.3 yrs) who were greater than 160% of their ideal body mass. The values were compared to predicted normal weight standards. There was no significant difference between groups in relation to VO2 max (L·min\(^{-1}\)) or T-vent (L·min\(^{-1}\)). The \( \tau \) response in the obese youth (29 ± 9 sec) was similar to the normal weight controls (28 ± 6 sec), as was the VO2 max (L·min\(^{-1}\)) and T-vent (L·min\(^{-1}\)). As mentioned, in the current study no \( \tau \) differences were noted, however the values obtained (severely overweight 33.9 ± 22.7 sec, normal weight 41.5 ± 21 sec) were higher than the values of Cooper et al. (1990), but were within the expected range (Armon et al., 1991; Cooper et al., 1985; Fawkner et al., 2002; Hebesteit et al., 1998; Williams et al., 2001).

Cooper et al. (1990) also observed that the kinetic response of VCO2 and \( V_e \) were prolonged in the obese group. Cooper et al. (1990) summarized that the subjects did not differ from the normal weight children on the basis of cardiorespiratory fitness (VO2 max L·min\(^{-1}\)) and the de-conditioning in the severely overweight child due to excessive body mass is a fallacy. The results of the present study agree with Cooper et al. (1990) in which there were no significant differences between the absolute VO2 (L·min\(^{-1}\)) values for VO2 peak or for T-vent (% of VO2 peak) between the severely overweight and normal weight subjects.

As noted earlier, O2 kinetic responses were similar for the severely overweight and normal weight girls. On the other hand, the severely overweight group had significant larger O2 deficit values than the normal weight group. We speculate that the larger values found in the severely overweight group may have been due to a greater percentage of the energy derived from anaerobic sources (Medbo et al., 1988, Renoux et al., 1999). This is only speculation as anaerobic indicators such as lactate production were not measured in the current study. Also, since the constant workloads employed were 20% below T-vent, we suggest that the slow component of VO2 was not confounding the O2 deficit since the slow component typically occurs above the lactate threshold (Gaesser and Poole, 1996; Fawner and Armstrong, 2003).

As noted in Table 4, weak correlation’s (r’s ranging from - 0.15 to 0.26) were found between phase I, \( \tau \) with VO2 peak or fat weight. These results suggest that fitness or fatness level had limited effect on O2 kinetic responses. Cooper et al. (1985) found similar results for phase I kinetics (r = 0.36) with mass or stature. However, in young heart patients, Mocellin et al., (1999) reported an r of -0.59 between VO2 max and the \( \frac{1}{2} \) time to VO2 steady-state. Fawkner et al. (2002) found no significant correlations for children, although in adult males phase II kinetics were related to VO2 max expressed in L·min\(^{-1}\), \( r = -0.62, -0.81 \text{ ml·kg}^{-1}·\text{min}^{-1} \) and -0.82 ml·kg\(^{-1}·\text{min}^{-1} \). On the other hand, the O2 deficit was moderately related to fitness \( (r = 0.51) \) or fatness \( (r = 0.35) \). Consequently as the peak VO2 (L·min\(^{-1}\)) or fat weight (kg) increased, the O2 deficit increased moderately.

**CONCLUSIONS**

In conclusion, we found severely overweight female youth to respond to a constant workload test below T-vent in a similar fashion as normal weight female youth in regard to the O2 kinetic response. These findings support the work of others. However, significant differences were found between groups for the O2 deficit and suggest that a greater anaerobic contribution may have been evident during the constant workload test in the severely overweight group. Research is needed to further explore the anaerobic contribution to the first few minutes of exercise stress in severely overweight youth.
REFERENCES


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Exercise physiology, acute and chronic response of normal weight and obese youth to physical activity, physiologic response of endurance athletes.

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KEY POINTS

- VO$_2$ (L·min$^{-1}$) similar between the severely overweight and normal weight female youth
- Phase I and II O$_2$ kinetic responses similar between severely overweight and normal weight female youth
- O$_2$ deficit was significantly greater in the severely overweight participants

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