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Sex Differences in Pulmonary Oxygen Uptake Kinetics in Obese Adolescents

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Abstract

Objective—To determine if sex differences exist in the pulmonary oxygen uptake (VO_2) uptake on-kinetic response to moderate exercise in obese adolescents. Additionally, we examined if a relationship exists between the VO_2 on-transient response to moderate intensity exercise, steady state VO_2, and peak VO_2 between obese male and female adolescents.

Study design—Male (n=12) and female (n=28) adolescents completed a graded exercise test to exhaustion on a treadmill. Data from the initial 4-min of treadmill walking were used to determine the time constant.

Results—The time constant was significantly different (*P*=0.001) between obese male and female adolescents (15.17±8.45 s vs. 23.07±8.91 s, respectively). No significant relationships were observed between the time constant and variables of interest in either sex.

Conclusions—Sex differences exist in VO_2 uptake on-kinetics during moderate exercise in obese adolescents, indicating an enhanced potential for males to deliver and/or utilize oxygen. It may be advantageous for females to engage in a longer warm-up period prior to initiation of an exercise regimen, preventing an early termination of the exercise session.

Keywords

Aerobic Fitness/VO2 Max; Assessing Physiological Demands of Physical Activity; Gas Exchange Kinetics in Laboratory and Field

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The study of the physiological mechanisms responsible for the oxygen consumption (VO_2) response to exercise is important in the context of understanding one's health, aerobic performance capabilities and the metabolic activity of muscle (1–3). Oxygen uptake increases when external work is imposed although the rise in VO_2 is not immediate and thus does not initially reflect the level expected for a specific workload at the initiation of exercise. Pulmonary VO_2 on-kinetics reflects the rate change in VO_2 during exercise; specifically the time needed for the cardiopulmonary system to deliver and skeletal muscle to consume the increased level of oxygen needed for aerobic metabolism (4, 5). A key portion and subsequent derived measure of VO_2 on-kinetic response is the phase II time constant ($\tau 2$), representing the time taken to reach 63% of steady state VO_2. In essence, individuals with a better health status and who participate in a regular aerobic exercise training program have a faster $\tau 2$. Comparatively, individuals with a poorer health status and/or lead a sedentary lifestyle have a slower $\tau 2$. In fact, $\tau 2$ has proven to be a valuable tool in providing information related to an individual's ability to tolerate physical activity (2, 3).

$\tau 2$ becomes progressively longer from adolescence into adulthood (6, 7), suggesting a maturation effect that significantly prolongs the moderate-intensity VO_2 on-kinetic response in adults (6, 8, 9). Although there is no support for greater oxygen delivery capacity in adolescents, there is support for enhanced oxidative enzymatic activity (10, 11) in adolescents when compared with adults. Studies that have attempted to provide an explanation of the enhanced muscle enzymatic activity, as well as fiber type distribution, between adults and adolescents have solely focused on normal weight male adolescents (11, 12). Moreover, it has recently been suggested that overweight children have an impaired exercise capacity when compared with their normal weight counterparts (13). Several studies have evaluated VO_2 on-kinetics between obese and non-obese children and adolescents. Earlier studies suggested that increased adiposity is not indicative of delayed VO_2 on-kinetics during submaximal exercise (12, 14, 15). However, more recent findings have suggested that obese children and adolescents display a markedly slower $\tau 2$ during both moderate and high intensity exercise when compared with their normal weight counterparts (16, 17).

Only one study has evaluated sex differences in VO_2 on-kinetics in adolescents (6), demonstrating non-significant sex differences between lean male and female adolescents. Interestingly, their results display moderate effect sizes (0.49), indicating that a larger sample size may have allowed for greater detection of differences between boys and girls. More importantly, studies have shown equivocal results regarding the impact increased adiposity may have on mitochondrial function in obese adolescents (18, 19).

The aim of this study was to investigate the VO_2 on-kinetic response to exercise performed below the ventilatory threshold (i.e. moderate intensity) in obese male and female adolescents. Additionally, we aimed to examine if a relationship exists between the VO_2 on-kinetic response to moderate intensity exercise and both steady state VO_2 and peak oxygen consumption (VO_{2peak}) in obese male and female adolescents.

METHODS

Obese male and female adolescents between 11 and 16 years of age [Body Mass Index (BMI) ≥ 85th percentile for age and sex according to the 2000 CDC Growth Charts] were recruited to participate in this study. Study procedures were explained and parents provided written, informed consent, and adolescents provided written assent prior to participation. A complete medical history, physical examination, and evaluation for participation in exercise testing were conducted by a physician. The physical examination included a standardized assessment of pubertal development via Tanner staging. To control for pubertal influence on VO_2 on-kinetics, the study was limited to adolescent males with a Tanner stage of at least 2 and females who had experienced menarche (20). All procedures were approved by the VCU Institutional Review Board.

Following an overnight fast, each adolescent underwent anthropometric measurement and fasting blood glucose assessment. Anthropometric measurements included height (to the nearest 0.5 cm), weight (to the nearest 0.25 kg), and body composition via whole body dual energy Xray absorptiometry (DXA, Hologic 4500a/Discovery scanner). Adolescents diagnosed with type 2 diabetes exhibit delayed VO_2 on-kinetics (14). Therefore, this study was limited to participants who did not have impaired fasting glucose (>100 mg/dL) (21).

Participants were asked to refrain from exercise 24 hours prior to the exercise test and arrived at least 4 hours postprandial. Peak oxygen consumption and VO_2 on-kinetics were determined using a maximal graded exercise test to exhaustion on a treadmill (Trackmaster TMX425C, Full Vision, Inc., Newton KS). Previous research has shown a high degree of reliability in using a single bout of exercise on a treadmill to measure VO_2 on-kinetics (22). Additionally, the utilization of a treadmill requires a subject to move their own weight, potentially affecting the cardiovascular and metabolic responses to exercise and exercise intolerance. Oxygen consumption, obtained through breath-by-breath gas exchange variables, was measured using a VMAX Sepctra Sensormedics gas analyzer (Sensormedics Corp., Yorba Linda, CA). Heart rate (HR) responses were recorded at each minute during the test via heart rate monitor (Model E600, Polar Electro, Lake Success, NY) and ratings of perceived exertion (RPE; 6–20 Borg Scale) were documented near the end of each stage.

Following a 3-min rest period of standing gas exchange, subjects began a step transition into a 4-minute stage at 2.5 mph and 0% grade. The progressive protocol continued with a 2-min stage at 3 mph at 0% grade. Subsequent 2-minute stages were held constant at 3.0 mph while grade was increased to 2%, 5%, 8%, 11%, 14%, and 17.0%. Subjects were verbally encouraged to give maximal effort during the test until volitional exhaustion was achieved. The attainment of VO_{2peak} was determined by participants satisfying at least two of the following criteria: (1) a respiratory exchange ratio (RER) ≥ 1.00; (2) a maximum HR ≥ 90% of age predicated maximum HR; and (3) RPE ≥ 18. Peak oxygen consumption was taken at the highest recorded 20s average during the maximal exercise test (23). Following the exercise test, ventilatory threshold (VT) was determined non-invasively by visual inspection using the V-slope method, which has shown good inter-observer agreement between and across exercise protocols (24). Ventilatory threshold was defined as the inflection point in which carbon dioxide production begins to rise at a more rapid rate than VO_2 (25).

Data from the initial 4-min stage was used for the exercise transition to assess VO_2 on-kinetics. A single bout of submaximal exercise on a treadmill has provided a high degree of reliability in measures of VO_2 on-kinetics (22). However, exercise eliciting a response above the VT poses a likelihood of a secondary rise in VO_2 on-kinetics that may alter the reliability of the τ2 (3). Therefore, to determine an intensity similar to that used in a previous investigation evaluating sex-based differences in VO_2 on-kinetics during transition from rest to moderate intensity exercise, data analysis within this study was limited to subjects with an initial stage VO_2 ($mLO_2 \cdot kg^{-1} \cdot min^{-1}$) less than 60% of VO_{2peak} and within 75–95% of their VT (6).

To determine VO_2 on-kinetics, O_2 uptake during the last 2-min of rest and throughout the first stage of the exercise test was averaged over 10 second intervals to reduce noise and enhance the underlying physiological response characteristics (22). Oxygen uptake at time zero was defined using the 2-min averaged resting data. The initial 20 seconds of exercise were not included in the kinetic analysis given the cardiodynamic effects of Phase 1. The remaining data set was fitted to a mono exponential curve with a delay relative to the onset of exercise of the form:

$$VO_2\,(t) = VO_{2\,(resting)} + VO_{2\,(amplitude)}\,[1 - e^{-(t/\,\tau VO_2)}]$$

where $VO_{2\,(t)}$ is O_2 uptake at any time *t*, $VO_{2\,(resting)}$ is the mean O_2 uptake measured during rest, $VO_{2\,(amplitude)}$ is the increase in O_2 uptake above rest (average of the last two minutes of exercise), *e* is the base of the natural logarithm, and τVO_2 is the τ2 or the fundamental component of the increase in VO_2 above baseline reported in seconds (2, 3, 26).

Statistical Analyses

Independent samples t-tests were used to investigate differences in anthropometric and exercise responses between the two groups. Additionally, correlation coefficients were used to investigate potential relationships between VO_2 on-kinetics and submaximal and maximal VO_2 variables. Furthermore, to account for potential differences in physical maturity between male and female participants, analyses were repeated using nine male-female adolescent pairs who were matched for Tanner staging. Statistical significance was set at $P \leq 0.05$ for all analyses.

RESULTS

The participants' physical characteristics and responses to the graded exercise test are presented in Table I. Group samples sizes were unequal due to attrition of recruited participants by not meeting inclusion criteria. Equal variances were observed in both groups, therefore results of the independent samples t-tests analyses were provided to indicate observed differences between the obese adolescent males and females. No significant differences were seen in age, BMI, and body composition variables between the two groups. Male adolescents displayed a significantly higher VO_{2peak} ($P = 0.030$, $d = 0.77$) and faster τ2 ($P = 0.013$, $d = 0.91$) than females. End stage VO_2 was approximately 87% of VT among both groups ($P = 0.745$). Resting VO_2 and the intensity of Stage 1 at the end of the 4-min stage, expressed as absolute VO_2, VO_2 per lean mass, and the percentage of VO_{2peak} were

not significantly different between the two groups ($P \geq 0.133$). A subgroup of age and Tanner matched male and female subjects was analyzed for comparison of physical characteristics and responses to the graded exercise test and are presented in Table II. Independent sample t-tests of the subgroup after Tanner stage matching continued to demonstrate significantly faster $\tau 2$ ($P = 0.038$, $d = 1.06$) in obese males compared with obese females.

A Pearson product moment correlation was used to asses any potential relationships between the $\tau 2$ and both maximal VO_2, expressed in terms of per body weight and per lean mass, and moderate (Stage 1) VO_2, expressed in absolute terms, as well as per lean mass and per body weight. No significant relationships were observed between the $\tau 2$ and the VO_2 variables of interest in either sex.

DISCUSSION

To date, only one study has evaluated sex differences in VO_2 on-kinetics in adolescents, demonstrating non-significant sex differences between lean male and female adolescents(6). Although the results of the current study are not in agreement with those of Fawkner et al. (6), their results display moderate effect sizes (0.49) in evaluating sex-based differences in VO_2 on-kinetics. Thus, the larger sample size presented in the current study may have allowed for greater detection of differences between boys and girls.

Previous studies investigating VO_2 on-kinetics in obese children and adolescents have suggested non-significant differences compared with their lean counterparts (13, 14). Interestingly, relative VO_{2peak} was shown to be significantly lower in the overweight and obese children and adolescents, leading investigators to suggest that increased adiposity was not indicative of poor submaximal exercise capacity in children or adolescents (12, 13). However, neither study considered sex differences, which in light of the results of the current study, may have confounded earlier reports in which study samples have been made up of between 40–46% female subjects. Our VO_2 on-kinetic differences observed during submaximal exercise between obese female and male adolescents was further supported by a similar energy requirement for the given workload. Within the current study, there were no significant differences in absolute VO_2, VO_2 per lean mass, and the percentage of VO_{2peak} at the end of the 4-minute submaximal Stage 1 workload.

In the current study, we did not find a significant relationship between VO_{2peak} and the $\tau 2$ among either group. It is plausible that the on-kinetic response is primarily influenced by the ability of skeletal muscle to create energy aerobically (26, 27) rather than the capacity of O_2 delivery. In fact, the primary increase in VO_2 on-kinetics after the onset of exercise has been shown to closely reflect the kinetics of aerobic energy production within skeletal muscle (11, 23). This is further supported in research performed by Grassi et al (28, 29), who demonstrated that VO_2 on-kinetics during moderate intensity contraction in the isolated canine gastrocnemius was not enhanced during elevated peripheral oxygen diffusion nor O_2 delivery, by means of muscle pump perfusion to levels associated with steady state exercise. In adults, it has been shown that cardiac output and bulk muscle blood flow are faster than the VO_2 on-kinetic response (30), indicating that oxygen delivery is adequate to support

oxygen demand of the muscle during the transition from rest to exercise. Furthermore, in the current study we did not find significant relationships ($P > 0.340$) between submaximal VO_2 variables, in both relative and absolute measurements, to submaximal phase II on-kinetics. Although cardiac output was not measured within the current study, our findings in obese male and female adolescents support previous investigations that suggest O_2 delivery may not be the limiting factor during the transition from rest to aerobic exercise (28, 30).

Assuming O_2 delivery does not represent a limiting factor in the on-kinetic response, one explanation for the observed sex differences is that obese males may exhibit greater aerobic enzyme activation compared with obese females, allowing for more efficient oxidative energy production in skeletal muscle, and thus, a faster on-kinetic response (28, 30). Leclair et al. (10) examined levels of deoxygenated hemoglobin via near-infrared spectroscopy and found that faster O_2 extraction at the onset of exercise occurs in children compared with adults, thus supporting the notion of enhanced muscle oxidative enzyme activity in children (12, 31). To date, no studies have examined potential sex differences in aerobic enzyme profiles in either lean or obese children or adolescents.

Previous studies have reported the presence of overweight and obesity to be higher among early-maturing girls and lower in early-maturing boys compared with control adolescents (32, 33). Despite evidence associating early pubertal development and obesity in females, such studies do not confirm a direct cause between adiposity and maturational events (i.e. menarche) (33). However, Frisch et al suggested that subcutaneous fat may double as a secondary hormonal gland, influencing release and synthesis of sex hormones such as estrogen thus promoting menarche (34). The hormonal changes associated with early sexual maturation in females may result in the observed VO_2 on-kinetic differences within our population of obese male and female adolescents. Given our findings, future research should be directed toward examining the impact of a prolonged warm up period for obese female adolescents during aerobic exercise training. Specifically, does this approach lead to improved tolerance to exercise training, caloric expenditure and weight loss? From a basic science perspective, the direct cause of delay in VO_2 on-kinetics among obese female adolescents compared with obese males remains unclear. Designing a research protocol which examines skeletal muscle enzymes and fiber type profiles of obese adolescents may provide greater insight into the metabolic control of muscle cells. Also, future research is warranted in understanding the potential role of sex steroids that may impact the dynamic O_2 uptake response to exercise.

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Abbreviations

$\tau 2$	phase II time constant

VO_2	oxygen consumption
BMI	body mass index
DXA	duel energy x-ray absorptiometry
$VO_{2\ peak}$	peak oxygen consumption

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Table 1

Differences in Anthropometric, Body Composition, and Cardiorespiratory Fitness Estimates Among Obese Adolescents (n=40) According to Sex.

Variable	Males (N=12)	Females (N=28)	P value
Age (years)	13.13 ± 1.19	14.20 ± 1.79	0.065
Body mass (kg)	99.99 ± 21.20	97.28 ± 16.72	0.667
BMI (kg/m$_2$)	35.98 ± 5.41	36.53 ± 4.50	0.740
%Body Fat (DXA)	41.43 ± 5.32	42.84 ± 3.91	0.378
Lean Mass (kg)	55.17 ± 6.75	55.24 ± 8.01	0.981
Fat Mass (kg)	39.39 ± 8.01	42.03 ± 9.81	0.433
Tanner stage	3.00 ± 0.73	3.89 ± 0.68	0.001
VO_{2peak} (L·min^{-1})	2.72 ± 0.41	2.42 ± 0.36	0.030
τ2 (seconds)	15.17 ± 8.45	23.07 ± 8.91	0.013
Resting VO_2 (L·min^{-1})	0.41 ± 0.06	0.38 ± 0.06	0.161
Stage 1 VO_2 (L·min^{-1})	1.30 ± 0.25	1.22 ± 0.17	0.262
Stage 1 $VO_{2\ LEAN}$ (L·kg of Lean^{-1}·min^{-1})	22.67 ± 1.92	22.38 ± 2.90	0.762
Stage 1 %Max VO_{2peak}	48.12 ± 6.22	50.90 ± 4.80	0.133
Ventilatory Threshold (L·min^{-1})	1.49 ± 0.29	1.41 ± 0.19	0.309
Stage 1 %Max Ventilatory Threshold	87.64 ± 3.75	87.06 ± 5.61	0.745

Mean ± S.D.; Statistical significance was set at $P \leq 0.05$; BMI, Body Mass Index; DXA, Dual Energy X-ray Absorptiometry; VO_{2peak}, peak oxygen consumption; τ2, Phase II Time Constant; VO_2, oxygen consumption; $VO_{2\ LEAN}$, oxygen consumption per lean mass.

Table 2

Differences in Anthropometric, Body Composition, and Cardiorespiratory Fitness Estimates in Male-Female Subjects Matched According to Tanner Staging of Pubertal Development

Variable	Males (N=9)	Females (N=9)	P value
Age (years)	13.22 ± 1.36	13.28 ± 1.16	0.917
Body mass (kg)	99.26 ± 24.46	99.76 ± 13.45	0.958
BMI (kg/m^2)	34.29 ± 5.00	38.44 ± 4.36	0.079
%Body Fat (DXA)	39.26 ± 3.60	42.97 ± 3.69	0.054
Lean Mass (kg)	55.79 ± 7.96	56.88 ± 8.26	0.786
Fat Mass (kg)	35.92 ± 4.35	42.87 ± 7.07	0.030
Tanner stage	3.33 ± 0.50	3.33 ± 0.50	1.000
VO_{2peak} (L·min^{-1})	2.78 ± 0.44	2.60 ± 0.29	0.337
τ2 (seconds)	15.57 ± 9.69	27.02 ± 11.72	0.038
Resting VO_2 (L·min^{-1})	0.43 ± 0.06	0.38 ± 0.08	0.168
Stage 1 VO_2 (L·min^{-1})	1.33 ± 0.27	1.32 ± 0.12	0.880
Stage 1 $VO_{2\ LEAN}$ (L·kg of Lean·min^{-1})	22.61 ± 0.97	23.57 ± 3.58	0.477
Stage 1 %Max VO_{2peak}	48.03 ± 5.64	50.87 ± 3.80	0.229
Stage 1 Ventilatory Threshold (L·min^{-1})	1.52 ± 0.31	1.49 ± 0.13	0.759
Stage 1 %Max Ventilatory Threshold	87.58 ± 4.30	88.66 ± 4.58	0.615

Mean ± S.D.; Statistical significance was set at $P \leq 0.05$; BMI, Body Mass Index; DXA, Dual Energy X-ray Absorptiometry; VO_{2peak}, peak oxygen consumption; τ2, Phase II Time Constant; VO_2, oxygen consumption; $VO_{2\ LEAN}$, oxygen consumption per lean mass.