Comparison of the Sitting-to-Standing Cycle Ergometry versus Treadmill Approach to Graded Exercise Testing on Cardiorespiratory Values in Overweight People with Mild Asthma

William B. Kist1,2, Katy Burgess2, Sharon E. Kist3, Megan Glasheen1, Elizabeth Delp1, Joanne Kraenzle-Schneider4

1Basic and Pharmaceutical Sciences, St. Louis College of Pharmacy, St. Louis MO 63110, 2Health, Kinesiology, and Recreation, Southern Arkansas University, Magnolia, AR 71753, 3Goldfarb School of Nursing at Barnes Jewish Hospital, St. Louis, MO 63110, 4St. Louis University School of Nursing, St. Louis, MO 63104

Abstract

Kist WB, Burgess K, Kist SE, Glasheen M, Delp E, Kraenzle-Schneider J. Comparison of the Sitting-to-Standing Cycle Ergometry versus Treadmill Approach to Graded Exercise Testing on Cardiorespiratory Values in Overweight People with Mild Asthma. JEPonline 2014;17(4):51-71. The purpose of this study was to: (a) determine if standing on a cycle ergometer (Stand CE) increases VO2 max to the equivalent of values obtained by a treadmill (TM); and (b) describe the combined effects of being overweight and having asthma on exercise cardiorespiratory variables. Subjects were overweight people with asthma (OWAS, n = 10) and those of normal weight and normal lungs (NWNL, n = 15). Groups differed (P<0.05) on BMI (OWAS = 27.8 ± 4.5 kg·m⁻², NWNL = 24.8 ± 3.5), FVC (OWAS = 4.0 ± 0.7 L, NWNL = 4.6 ± 0.9), and FEV1 (OWAS = 3.3 ± 0.4 L, NWNL= 3.7 ± 0.7). There were differences by group, but not trial on VO2 max (OWAS: TM = 31.9 ± 6.5 mL·kg⁻¹·min⁻¹, Stand CE = 29.0 ± 6.8, NWNL: TM = 40.2 ± 4.3, Stand CE = 35.9 ± 5.2). This study showed that Stand CE and TM VO2 max values were not statistically different, and that asthma and being overweight had little influence on the exercise cardiorespiratory variables. Differences, when present were due to the OWAS being more deconditioned.

Key Words: Oxygen Consumption, Metabolic, Ventilation, Mode
INTRODUCTION

Graded exercise testing (GXT) using either a treadmill (TM) or a cycle ergometer (CE) is commonly used to assess healthy individuals and people with diseases (2,5,9,19,26,33). Each GXT modality has advantages and disadvantages (1,2,5,26). However, because maximal oxygen consumption (VO₂ max) obtained by TM-GXT is generally of greater magnitude (up to ~25%) (26) than CE-GXT, TM-GXT is generally considered the gold-standard (2,5,26,33). Most CE-GXT, especially in non-athletic populations (i.e., clinical GXT), have been performed with the subjects seated throughout the GXT (Sit CE) (26,33,50). In an effort to take advantage of the safety of GXT, and to increase CE-VO₂ max, we recently demonstrated, using our novel sit-to-stand CE-GXT (Stand CE) in recreationally-trained healthy individuals, that there was no statistical difference in VO₂ max between TM and Stand CE (26). Our Stand CE has not been tested in a diseased population, such as people with asthma.

Atopic asthma (i.e., IgE-mediated) is characterized by chronic airway inflammation and intermittent bronchoconstriction in response to a variety of stimuli that results in airway obstruction (14). In the USA, an estimated 20 million adults have asthma (3,10,46). Annually, there are 15 million medical visits for asthma treatment, and asthma accounts for 1.1 deaths per 100,000 people. The incidence of asthma is increasing in adults concurrently with increases in body mass index (BMI) (47). The exact mechanism(s) for the asthma and BMI relationship remains unclear (10,40,43,45).

Obesity in the USA is epidemic (39). More than 35% of adults are obese and over 30% are overweight. The classification/distinction between being overweight versus obese is arbitrary, but commonly based upon BMI (overweight >25.0 and ≤29.9 kg·m⁻², obese ≥30 kg·m⁻²) (2,20,40). The healthcare costs of obesity approximately $150 billion a year (39). The prevalence of being overweight or obese in asthma is almost double that of the general population. Being obese is an independent risk factor for asthma (8,30,44,45,52), while being overweight is a risk factor for acquiring a new diagnosis of asthma (8). Obesity, like asthma, has inflammatory features (43).

Although the reason is uncertain, evidence suggests that overweight and obese individuals have increased numbers of eosinophils in the airways that likely cause local inflammation and increased plasma levels of adipokines (from adipocytes), which likely cause inflammation at distal sites such as the bronchioles (15,29,40,43-45). Leptin, an adipokine, has been shown to have receptors in the airways that might be involved in airway inflammation (8). In addition to inflammation, obesity (especially) and being overweight can have negative effects on the pulmonary system that include altered lung volumes, increased work of breathing, and decreased pulmonary compliance (40,50).

Because 85% of people with asthma have experienced exercise induced bronchospasm (EIB), it has been hypothesized that avoidance of exercise may lead to an increased BMI, which may lead to greater airway inflammation, perhaps establishing a cycle of physical deterioration (40). Although this hypothesis is not universally accepted (10,40), it is suspected that even a mildly increased BMI in people with asthma may adversely affect lung function and impair exercise (40,44,53). It has been noted (16) that there are few studies that have evaluated gas exchange during GXT in people with mild asthma, and those studies are confounded by small numbers of subjects, use of children as participants, and short duration and low intensity GXT protocols (35). Amazingly, even the complexity of airflow pattern in asthma with changes in airway obstruction remain mostly unknown (48), especially during exercise. Also, little is known about the combined effects of being overweight and having mild stable asthma on cardiorespiratory variables during exercise (13,17,26).
The primary purpose of this pilot study was to determine if Stand CE could generate cardiorespiratory values, especially VO₂ max, equivalent to TM-VO₂ max values in overweight people with asthma. A secondary purpose was to describe if the combined effects of being overweight and having asthma would affect submaximal and maximal exercise cardiorespiratory variables differently compared to normal weight individuals with normal lungs. Any cardiorespiratory effects, if present, should be manifested through ventilation (50) and/or oxygen kinetics' (23) mechanisms. A third purpose was to establish effect size, for a subsequent larger study, on the effects of asthma and being overweight on cardiorespiratory variables with exercise.

METHODS

Subjects
The University's Institutional Review Board prospectively approved this pilot study. Written informed consent was obtained. Thirteen overweight people with asthma (OWAS, ~50% female) and 15 normal weight people with normal lung function (NWNL, ~50% female) were screened using the physical activity readiness questionnaire and primary investigator-created medical and fitness questionnaires (2,26,33). Normal weight and normal lung participants were “apparently healthy” (2). People with asthma provided evidence of having been diagnosed with asthma by a physician, and were healthy except for having atopic asthma and being overweight. People with asthma were stable as their medication regimen included only the daily use of a steroid metered dose inhaler with occasional use of a short-acting beta-2 agonist (rescue) metered dose inhaler (38). All subjects were familiar with bicycling and jogging/running although they were sedentary (no training for previous 12 months). During all GXT, crucial safety guidelines were followed (4,36,50).

Pre-Exercise Measurements
Spirometry was performed prior to GXT following the American Thoracic Society guidelines (27). Subjects were required to demonstrate a forced expiratory volume in one second (FEV1.0) value greater than 70% of predicted (Knudson’s regression equations, 1983) (49) and a FEV1.0 to forced vital capacity (FEV1.0/FVC) ratio greater than 70% prior to GXT. If OWAS subjects could not attain these values, they would take two inspirations of their rescue metered dose inhaler, wait 15 min, and then repeat spirometry to assure their airway function criteria were met prior to GXT. If these criteria could not be met, subjects were scheduled for an alternate day of testing. If spirometry criteria were not met on the second occasion, subjects would be dropped from the study. Percent body fat (% fat) was calculated from skinfold measurements obtained from the upper, middle, and lower areas of the body, employing a common 3-site protocol and regression formulas (2). Height and weight were measured using a “medical” scale and BMI was calculated (2,6).

Study Design and Exercise Trials
We used a split-plot design, groups (between subjects) by GXT trials, with repeated measures (within subjects) on trials (24). Graded exercise testing trials (TM, Sit CE, Stand CE) were conducted in counterbalanced sequence (randomly chosen). For the TM trial, we used a programmable TM (Quinton ST-55 treadmill, Cardiac Science Corp., Seattle, WA) adhering to the Bruce protocol (2,37). For the two CE trials, we used a mechanically-braked CE (Monark 828E, Vansbro, Sweden) following our previously described protocol (33) that was metabolic equivalent (MET) matched to the Bruce TM protocol.

Before the CE trials, the subjects were acclimated to the CE, which included a brief period of low-intensity cycling and practice in standing up while pedaling. During the Sit CE trial, the subjects
remained seated throughout the GXT until their respiratory exchange ratio (RER, carbon dioxide production/oxygen consumption) was 1.0 and, then, increased their pedaling rate to 70 rev·min\(^{-1}\) for remainder of the trial. The pedaling protocol for the Stand CE trial was identical to the Sit CE trial except that when the RER was 1.0, the subjects stood up and pedaled at 70 rev·min\(^{-1}\) for the remainder of the trial.

Additionally, at the instant of standing up, the workload on the CE was increased from its current setting to 50% of the estimated workload setting (workload = body weight in kg * 0.075) that would be used to perform the Wingate anaerobic CE test (6). This workload increase, which represents an altered testing strategy/approach compared to our earlier work, was intended to increase VO\(_{2}\) max values to those obtained by TM-GXT (26).

To study oxygen kinetics (23) under steady-state submaximal conditions, the subjects exercised on a TM (fourth trial, always after the other three trials) at 40%, 60%, and 80% of their TM-VO\(_{2}\) max value for 10 min at each workload/intensity. That is, we used the speed and grade where the subjects achieved their 40%, 60%, and 80% TM-VO\(_{2}\) max value. Therefore, the subjects with low VO\(_{2}\) max values exercised at a lower absolute workload than those who had higher VO\(_{2}\) max values during the steady-state trial. Post-exercise spirometry was conducted following all trials (49,50), which generally consisted of a Tuesday-Thursday-Tuesday-Thursday sequence (2,33).

For all trials cardiorespiratory variables: ventilation variables (e.g., minute ventilation, V\(_{E}\); tidal volume, VT; respiratory rate; RR, VCO\(_{2}\); carbon dioxide production; PETCO\(_{2}\), peak end tidal carbon dioxide tension; PETO\(_{2}\), and peak end tidal oxygen consumption), and oxygen kinetics-related variables (e.g., oxygen consumption, VO\(_{2}\); VO\(_{2}\) max; and heart rate, HR) were measured before and during exercise. Related cardiorespiratory variables were calculated from the previously noted measures [e.g., O\(_{2}\) pulse, VO\(_{2}\)/HR; RER, ventilation equivalents for carbon dioxide (V\(_{E}/VCO_{2}\)) and oxygen (V\(_{E}/VO_{2}\))].

The so-called “anaerobic threshold” (AT) (7, 50) was determined by the primary investigator using agreement of two methods: “V-slope plot” (VCO\(_{2}\) = ordinate vs. VO\(_{2}\) abscissa), and the nadir of the V\(_{E}/VO_{2}\) during the period of “isocapnic buffering” (plateau) of V\(_{E}/VCO_{2}\) (50). Cardiorespiratory measurements were obtained using a metabolic measurement system (Medical Graphics Corporation CPX-D breath by breath system, St. Paul, MN) in “real time” using breath-by-breath methodology, but averaged for 30 sec for reports (4,37,50). During trials, 12-lead EKG (Quinton Q4500 12-lead EKG system, Cardiac Science Corp. Seattle WA) was monitored for subject safety and to obtain HR measurements (2,36,37).

We calibrated (or validated) all measurement equipment intermittently throughout the study. The metabolic system analyzers (oxygen, carbon dioxide, and volume/flow) were calibrated immediately prior to trials (33). Exercise-related normal values (predicted values and equations) used during GXT come from the work of Wasserman (50).

**Statistical Analyses**

Prior to statistical analyses, the data were screened for normality, homogeneity of variance, and univariate and multivariate outliers (24) using SPSS (version 20, IBM-SPSS, Chicago, IL). Unfortunately, as a consequence of screening, three of the original thirteen OWAS subjects were excluded for being multivariate outliers on crucial variables. That is, the three subjects lost by screening were outliers on both spirometry and body composition/anthropometry values. Thus, the primary investigator decided to drop them from the study’s analyses due to their effect of undue influence on measures of central tendency and variance in this small sample-sized pilot study (24).
This consequentially resulted in a more unequal number of participants between the two groups [OWAS, n = 10 (~50% female); NWNL, n = 15].

Comparison between groups on two variables (e.g., height, weight, etc.) were analyzed per independent samples’ t-tests. Comparisons on two variables that were matched (e.g., observed spirometry values vs. both “predicted” (49) values, and post-exercise observed values) were analyzed per paired samples’ t-tests. Data that were independent of body weight (e.g., HR max, RER max, etc.) or already corrected for body weight (e.g., VO₂ max in mL·kg⁻¹·min⁻¹, etc.) were analyzed by 2-way ANOVA (group by trial with repeated measures on trials), while data directly weight-dependent (e.g., O₂ pulse, VCO₂, Vₑ, etc.) were analyzed by 2-way ANCOVA (group by trial, with repeated measures on trials) with BMI or % fat as covariates.

*Post-hoc* testing was performed using Tukey HSD methodology. Observed power values from analyses were noted. The effect of group (OWAS vs. NWNL), using Cohen’s *d*, was calculated from the TM (gold standard) trials. For all statistical analyses the level of significance was P<0.05.

The steady-state oxygen kinetics’ data (e.g., VO₂ mL·kg⁻¹·min⁻¹ vs. time, at 40%, 60%, 80% of TM-VO₂ max) (50) were analyzed by independent samples’ t-tests for each minute to determine if VO₂ was similar in magnitude (and pattern) between groups. The slopes (unstandardized beta coefficients) (41) of different phases of the oxygen kinetics (Phase I, II, and III) (23) data were calculated using linear regression, and then using the student’s *t*-test (difference between the slopes/standard error difference between slopes) determined if differences could be detected between groups. All graphing of figures was completed with Sigma Plot 8.0 (IBM-SPSS, Chicago IL).

**RESULTS**

**Subject Characteristics, Spirometry, and Resting Cardiorespiratory Variables by Group**
Subject characteristics, spirometry, and resting cardiorespiratory variables by group are reported in Table 1. The groups were similar on height, age, FVC-percent-predicted, and FEV1.0-percent-predicted. Groups differed (P<0.05) on BMI, % fat, FVC (absolute), and FEV1.0 (absolute). Mathematically, but not statistically, the groups also differed on the FEV1.0/FVC with the OWAS having a higher ratio than the NWNL group. Regardless of the spirometry differences, both groups’ values (pre-exercise resting) were within normal limits (27,49).

The correlations between % fat and FVC and FEV1.0 were r = -0.195 and r = -0.068, respectively, of which neither correlation was statistically significant. Lastly, the only other difference between groups was on resting HR, where OWAS demonstrated statistically greater values. However, resting HR for both groups were within normal limits (60 to 100 beats·min⁻¹) (2,50). None of the OWAS subjects used their rescue metered dose inhaler before resting HR was obtained.
Table 1. Subject Characteristics, Spirometry Values, and Cardiorespiratory Variables.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Asthma Group (N = 10)</th>
<th>Normal Group (N = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
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<tr>
<td>Height (cm)</td>
<td>169.2 ± 9.3</td>
<td>172.7 ± 9.3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.8 ± 16.8</td>
<td>74.8 ± 16.9</td>
</tr>
<tr>
<td>BMI (kg⋅m⁻²)</td>
<td>27.8 ± 4.5</td>
<td>24.8 ± 3.5</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>22 ± 4</td>
<td>22 ± 2</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>18.9 ± 3.2</td>
<td>14.5 ± 1.5</td>
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<tr>
<td>Pre FVC (L) †</td>
<td>4.0 ± 0.7</td>
<td>4.6 ± 0.9</td>
</tr>
<tr>
<td>Pre FVC (%) predicted ‡</td>
<td>89 ± 8</td>
<td>94 ± 13</td>
</tr>
<tr>
<td>Pre FEV1 (L) §</td>
<td>3.3 ± 0.4</td>
<td>3.7 ± 0.7</td>
</tr>
<tr>
<td>Pre FEV1 (%) predicted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre FEV1/FVC (%) ¶</td>
<td>83.2 ± 5.4</td>
<td>79.8 ± 3.8</td>
</tr>
<tr>
<td>Pre FEF 25-75 (L·sec⁻¹) **</td>
<td>3.4 ± 0.7</td>
<td>3.5 ± 0.6</td>
</tr>
<tr>
<td>Vₑ (L·min⁻¹) ††</td>
<td>9.7 ± 2.6</td>
<td>11.1 ± 5.2</td>
</tr>
<tr>
<td>VCO₂ (mL·min⁻¹) ‡‡</td>
<td>296 ± 88</td>
<td>323 ± 175</td>
</tr>
<tr>
<td>VO₂ (mL·min⁻¹) §§</td>
<td>335 ± 99</td>
<td>328 ± 145</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
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</tbody>
</table>

Values represent means and standard deviations (M ± SD). Asterisk “*” indicates statistical difference (P<0.05) by independent samples t-tests † - before exercise (baseline), forced vital capacity (FVC) (BTPS); ‡ - before exercise (baseline), FVC percent predicted; § - before exercise (baseline), forced expiratory volume in one second (FEV1) (BTPS); || - before exercise (baseline), FEV1 percent predicted; ¶ - before exercise (baseline), FEV1/FVC percent; ** - before exercise (baseline), forced expiratory flow 25-75 (BTPS); †† - Vₑ before exercise (i.e., resting), minute ventilation (BTPS); ‡‡ - VCO₂ before exercise (i.e., resting), carbon dioxide production (STPD); §§ - VO₂ before exercise (i.e., resting), oxygen consumption (STPD); and |||| - HR before exercise (i.e., resting), heart rate.

Submaximal Exercise Cardiorespiratory Variables by Group and Trial
The submaximal (measured at the AT) (7,50) GXT cardiorespiratory variables by group and trial are reported in Table 2. Oxygen consumption at the AT showed that OWAS values were lower than the NWNL group, except for the Stand CE trial. In contrast, HR showed that the OWAS values...
were generally higher than the NWNL group values, except for the TM trial. Tidal volume was different by group except for the TM trial where OWAS and NWNL values were equivalent. There were no differences by group on the other ventilation variables.

Table 2. Submaximal Cardiorespiratory Exercise-Related Variables by Group and Trial.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Asthma Group</th>
<th>Normal Group</th>
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<tbody>
<tr>
<td></td>
<td>(N = 10)</td>
<td>(N = 15)</td>
</tr>
<tr>
<td></td>
<td>TM†</td>
<td>Sit CE‡</td>
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<tr>
<td></td>
<td>Stand CE§</td>
<td>TM</td>
</tr>
<tr>
<td></td>
<td>Sit CE</td>
<td>Stand CE</td>
</tr>
<tr>
<td>VO₂@AT (mL·min⁻¹)</td>
<td>1273 ± 328 a</td>
<td>859 ± 233 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1761 ± 631 b</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1222 ± 326 a</td>
</tr>
<tr>
<td>HR@AT (beats·min⁻¹)</td>
<td>152 ± 18 a</td>
<td>143 ± 20 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>152 ± 13 a</td>
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<tr>
<td></td>
<td></td>
<td>135 ± 16 b</td>
</tr>
<tr>
<td>VT@AT (L)</td>
<td>1.4 ± 0.25 a</td>
<td>1.1 ± 0.6 b</td>
</tr>
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<td></td>
<td></td>
<td>1.4 ± 0.6 b</td>
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<td></td>
<td></td>
<td>1.2 ± 0.5 a</td>
</tr>
<tr>
<td>RR@AT (beats·min⁻¹)</td>
<td>20 ± 6 a</td>
<td>22 ± 7 a</td>
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<td></td>
<td></td>
<td>23 ± 6 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>21 ± 5 a</td>
</tr>
<tr>
<td>VE/VO₂ @AT ‡‡</td>
<td>20.9 ± 2.8 a</td>
<td>23.4 ± 3.4 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24 ± 5.3 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>22.7 ± 2.5 a</td>
</tr>
<tr>
<td>VE/VCO₂ @AT §§</td>
<td>24.4 ± 3.4 a</td>
<td>26.7 ± 4.3 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>26.8 ± 4.0 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>26.6 ± 2.2 a</td>
</tr>
<tr>
<td>PETO₂ (mmHg)</td>
<td>92 ± 5 a</td>
<td>97 ± 7 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>99 ± 8 a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>96 ± 6 a</td>
</tr>
<tr>
<td>PETCO₂ (mmHg)</td>
<td>47 ± 4 a</td>
<td>45 ± 5 a</td>
</tr>
<tr>
<td></td>
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<td>42 ± 6 a</td>
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<td></td>
<td></td>
<td>43 ± 2 a</td>
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</tbody>
</table>

Values represent means ± standard deviations (M ± SD). Values with different superscripts ("a" or "b") were statistically different (P<0.05). || - VO₂@AT (STPD), oxygen consumption at the anaerobic threshold; ¶ - HR@AT, heart rate at the anaerobic threshold; ** - VT@AT (BTPS), tidal volume at the anaerobic threshold; †† - RR@AT, respiratory rate at the anaerobic threshold; ‡‡ - VE/VO₂@AT (L BTPS · L STPD⁻¹), ventilation equivalent for oxygen at the anaerobic threshold; §§ - VE/VCO₂@AT (L BTPS · L STPD⁻¹), ventilation equivalent for carbon dioxide at the anaerobic threshold; |||| - PETO₂, peak end-tidal oxygen tension at the anaerobic threshold; and PETCO₂, peak end-tidal carbon dioxide tension at the anaerobic threshold.

Maximal Exercise Cardiorespiratory Variables by Group and Trial

Maximal exercise cardiorespiratory variables by group and trial are reported in Table 3. Maximal oxygen consumption, O₂ pulse, and VE max were greater in the NWNL group versus the OWAS group. Maximal HR was similar across trials except for the NWNL group demonstrated a significantly lower HR in the Stand CE trial. Similarly, VCO₂ was not different between trials except for the NWNL group demonstrating a significantly greater value on the TM trial. Tidal volume was similar by group and trial except for the OWAS group demonstrating a smaller value on the Stand CE trial. There were no differences by group or trial on other ventilation variables. The mean TM breathing reserve value (not reported in Table 3), which was calculated from the measured VE max...
during the TM-GXT (Table 3) divided by the estimated $V_E$ max (pre-exercise TM-FEV1.0 * 40, Table 1) (50) was 67% for the OWAS group and 71% for the NWNL group.

Table 3. Maximal Cardiorespiratory Exercise-Related Variables by Group and Trial.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Asthma Group (N = 10)</th>
<th>Normal Group (N = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TM†</td>
<td>Sit CE‡</td>
</tr>
<tr>
<td>$VO_2$ max (mL·kg⁻¹·min⁻¹)</td>
<td></td>
<td>31.9 ± 6.5 a</td>
</tr>
<tr>
<td>HR max (beats·min⁻¹)</td>
<td></td>
<td>188 ± 13 a</td>
</tr>
<tr>
<td>$O_2$ pulse (mL·bpm⁻¹)</td>
<td></td>
<td>13.1 ± 2.9 a</td>
</tr>
<tr>
<td>$VCO_2$ max (mL·min⁻¹)††</td>
<td></td>
<td>3160 ± 750 a</td>
</tr>
<tr>
<td>RER max §§§</td>
<td></td>
<td>1.27 ± 0.08 a</td>
</tr>
<tr>
<td>VT max</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RR max ¶¶ (breaths·min⁻¹)</td>
<td></td>
<td>45 ± 12 a</td>
</tr>
<tr>
<td>$V_E$ max*** (L·min⁻¹)</td>
<td></td>
<td>88.6 ± 20.7 a</td>
</tr>
<tr>
<td>$V_E$/VO$_2$ max †††</td>
<td></td>
<td>35.5 ± 5.0 a</td>
</tr>
<tr>
<td>$V_E$/VCO$_2$ max §§§</td>
<td></td>
<td>28.3 ± 3.7 a</td>
</tr>
<tr>
<td>PETO$_2$ max ¶¶¶ (mmHg)</td>
<td></td>
<td>112 ± 5 a</td>
</tr>
<tr>
<td>PETCO$_2$ max **** (mmHg)</td>
<td></td>
<td>42 ± 4 a</td>
</tr>
</tbody>
</table>

Values represent means ± standard deviation (M ± SD). Values with different superscripts ("a" or "b") were statistically different (P<0.05). || - $VO_2$ max (STPD), maximal oxygen consumption; ¶ - HR max, maximal heart rate; ** - $O_2$ pulse, oxygen pulse ($VO_2$ max/HR max); †† - $VCO_2$ max (STPD), maximal carbon dioxide production; §§ - RER max, maximal respiratory exchange ratio ($VCO_2$ max/$VO_2$ max); |||| - VT max (BTPS), maximal tidal volume; ¶¶ - RR max, maximal respiratory rate; *** - $V_E$ max (BTPS), maximal minute ventilation; ††† - $V_E$/VO$_2$ max (L BTPS · L STPD⁻¹), maximal ventilation equivalent for oxygen; §§§ - $V_E$/VCO$_2$ max (L BTPS · L STPD⁻¹), maximal ventilation equivalent for carbon dioxide; ¶¶¶ - PETO$_2$ max, maximal peak end-tidal tension for oxygen; and **** - PETCO$_2$ max, maximal peak end-tidal tension for carbon dioxide.
Composite Cardiorespiratory Variables by Group
Composite (collapsed across trials, i.e., effect of group) maximal exercise cardiorespiratory variables are illustrated in Figure 1. The NWNL participants demonstrated significantly greater values on all variables except for RER max (equivalent) and HR max (OWAS > NWNL). The correlation of % fat and VO₂ max (mL·kg⁻¹·min⁻¹) was -0.724, which was significant with a calculated R² = 52%.

Figure 1. Composite Maximal Exercise Cardiorespiratory Values by Group. Histograms illustrate the means and standard deviations of all exercise trials (TM, Sit CE, and Stand CE). Statistical differences (P<0.05) by group are indicated by the asterisks.

Oxygen Consumption at 40%, 60%, and 80% of VO₂ Max
Treadmill submaximal steady-state VO₂ per time (i.e., oxygen kinetics) at 40%, 60%, and 80% of TM VO₂ max (Figure 2) demonstrated that the NWNL had significantly greater values than the OWAS at almost all intensities and times (except at 40/1, 40/7, and 80/3). The slopes of the regression lines for the plateaus (Phase III oxygen kinetics) at the 40% and 60% intensity were not different between groups. The slopes for the transition (Phase I and II oxygen kinetics) between the intensities of 40% to 60% VO₂ max and 60% to 80% VO₂ max were not different. The NWNL exercised for a greater duration than the OWAS (e.g., 80/5). During the 80% portion of the trial, a few participants in each group reached their TM-VO₂ max value.
Figure 2. Treadmill Oxygen Consumption at 40%, 60%, and 80% of TM VO₂ Max by Group.
Line and scatter plots represent means and standard deviations for both groups. 40/1 = 40% of TM VO₂ max at min 1; 60/3 = 60% of TM VO₂ max at min 3, etc. Each group’s TM VO₂ max values are shown to the far right. Values were statistically different (P<0.05) at almost all intensities and times.

Spirometry Values, Pre- and Post-Maximal Exercise
Composite (collapsed across trials) pre-exercise and the post-exercise spirometry values are illustrated in Figure 3. The OWAS group had a mathematically, but not statistically higher FEV1.0/FVC than the NWNL group. The most likely reason for this finding was that the OWAS group likely had an incomplete exhalation during the forced maneuver that caused air-trapping (22,27,49).

Post-exercise FVC and FEV1.0 remained significantly greater in the NWNL group over the OWAS group. There were no differences in the pre-exercise versus post-exercise FEV1.0/FVC and forced expiratory flow between 25% and 75% (FEF25-75) of the FVC for either group. No subject demonstrated a decrease in spirometry values (i.e., EIB), exhibited excessive coughing or inappropriate dyspnea during or following exercise.
Figure 3. Composite Spirometry Values Pre- and Post-Exercise by Group. Histograms represent the means and standard deviations of all (TM, Sit CE, and Stand CE) pre- and post-exercise trials. Pre FVC, pre exercise forced vital capacity (FVC, L BTPS); Pre FEV1, pre exercise forced expiratory volume in 1 sec (FEV1, L BTPS); Pre FEF 25-75, pre-exercise forced expiratory flow between 25 and 75% of the FVC (L·sec⁻¹ BTPS); Post FVC (L·BTPS), post-exercise FVC (L BTPS); Post FEV1, post-exercise FEV1 (L BTPS); Post FEF 25-75, post-exercise forced expiratory flow between 25% and 75% of the FVC (L·sec⁻¹ BTPS). Note in the figure that FEV1.0/FVC is a ratio/percentage, and is not reported in L BTPS (ordinate axis). Statistical differences (P<0.05) by group are indicated by the asterisks.

The observed power values for the statistical analyses for the most crucial anthropometric and cardiorespiratory variables are subsequently presented by exercise intensity (rest, submaximal, and maximal). The resting BMI power value by group was 0.393 while for % fat it was 0.995. Pre-exercise spirometry power values by group were: a. Pre FEV1.0 = 0.984, b. Pre FVC = 1.0, Pre FEV1.0/FVC = 0.966 and Pre FEF25-75 = 0.050. The submaximal intensity power values presented by diagnosis, trial, and group and trial, respectively, were: VO2@AT = 0.859, 0.625, and 0.257; HR@AT = 0.456, 0.745, and 0.200; VT@AT = 0.147, 0.172, and 0.113; RR@AT = 0.135, 0.165, and 0.180; VE/VO2 = 0.240, 0.214, and 0.281; VE/VCO2@AT = 0.185, 0.256, and 0.139; PETO2@AT = 0.479, 0.298, 0.373; and PETCO2 = 0.669, 0.091, and 0.258. The maximal intensity power values presented by diagnosis, trial, and group and trial, respectively, were: VO2 max = 1.0, 0.543, and 0.060; HR max = 0.858, 0.508, and 0.063; O2 pulse = 0.992, 0.243, 0.097; RER max 0.655, 0.488, and 0.478; VCO2 max = 0.776, 0.504, and 0.060; VT max = 0.514, 0.058, and 0.104; RR max = 0.093, 0.070, and 0.085; VE max = 0.423, 0.581, and 0.055; VE/VO2 max = 0.077, 0.581, and 0.055; VE/VCO2 max = 0.060, 0.835, and 0.065; PETO2 max = 0.065, 0.627, and 0.053; and PETCO2 max = 0.070, 0.876, and 0.082.

Effect values by group for the most crucial anthropometric and cardiorespiratory variables are subsequently presented by exercise intensity (rest, submaximal, and maximal). The resting BMI
effect value was 0.750, % fat was 1.193, and resting HR was 0.733. Pre-exercise spirometry effect values were 0.750 for FVC, 0.800 for FEV1.0, 0.739 for FEV1.0/FVC, and 0.154 for FEF25-75. The most important submaximal intensity effect value was 2.503 for VO2@AT. The maximal intensity effect values were 1.540 for VO2 max, 0.571 for HR max, 1.282 for O2 pulse, 0.727 for VCO2 max, 0.053 for RER max, 0.333 for VT max, 0.222 for RR max, 0.717 for VE max, 0.152 for VE/VCO2 max, 0.125 for VE/VO2 max, 0.001 for PETO2 max, and 0.250 for PETCO2 max.

DISCUSSION

Improvement in the Stand CE Graded Exercise Testing Approach on Cardiorespiratory Variables in Overweight People with Asthma

The primary purpose of this pilot study was to determine if Stand CE could generate cardiorespiratory values, especially VO2 max, equivalent to TM-VO2 max values in overweight people with asthma. If Stand CE-VO2 max was equivalent to TM-VO2 max in OWAS, the Stand CE might be used to assess similar populations that are unlikely to be able to stand (e.g., diseased, deconditioned, balance/gait-impaired, etc.) throughout an entire CE-GXT or TM-GXT (50). Also, if Stand CE-VO2 max values were equivalent in magnitude to TM-VO2 max values, this offers the advantages of CE-GXT safety and ease of physiological monitoring compared with the more risky TM-GXT (2,26,50). Also, it would allow the use of well-established TM-VO2 max prediction equations to be used for CE-GXT, which have been minimally established (19).

The findings (Table 3) of the current study statistically support our hypothesis as Stand CE-VO2 max values were equal to TM-VO2 max values for each group. The OWAS group’s Stand CE-VO2 max value was 95% of their TM-VO2 max value. The difference between the TM and Stand CE-VO2max absolute values for the OWAS group was ~3 mL·kg⁻¹·min⁻¹, which is slightly less than one MET. In general, a less than 1 MET difference would be of little, if any, significance in clinical GXT (2,6,50). Likewise, the NWNL group’s Stand CE and TM-VO2 max values were statistically equivalent. Unfortunately, these statistically favorable Stand CE findings in the NWNL group were not as encouraging on a percentagewise or mathematical basis. The absolute difference between TM and Stand CE for the NWNL group was ~4 mL·kg⁻¹·min⁻¹ (which is slightly greater than 1 MET). Furthermore, the NWNL group’s Stand CE VO2 max value was 90% of their TM VO2 max value, which is in general a typical finding when comparing TM and CE-GXT in non-athletic populations (2,26,33).

The GXT modality findings of the present study were generally better than our previous work using the Stand CE in a more aerobically fit (recreationally fit, non-diseased) population of males and females (26). In that study, the males’ TM-VO2 max was ~55 mL·kg⁻¹·min⁻¹ while their Stand CE VO2 max value was ~51 mL·kg⁻¹·min⁻¹, thus, male Stand CE VO2 max was 93% of TM VO2 max. In that study, female TM VO2 max was ~40 mL·kg⁻¹·min⁻¹ and their Stand CE VO2 max value was ~35 mL·kg⁻¹·min⁻¹. That is, female Stand CE VO2 max was 88% of TM VO2 max. In that study, the males had slightly greater than 1 MET difference between modes of exercise while the females had a slightly larger (~1.5 MET) difference. In an earlier investigation of ours, using our Stand CE and an intentionally diverse population (10 females and 24 males, 18 to 54 yrs), we demonstrated a ~1.75 MET difference between the TM VO2 max (~50 mL·kg⁻¹·min⁻¹) and the Stand CE VO2 max (~39 mL·kg⁻¹·min⁻¹). Thus, the current study’s comparison of TM-VO2 max and Stand CE VO2 max is improved over the earlier studies due to our addition of the Wingate workload. The Stand CE was mathematically equivalent or greater than (Table 3) the Sit CE on VO2 max.

To reiterate, the difference in our Stand CE GXT approach of the present study versus our earlier studies was that, for the current study, we added a final increased workload based upon the
Wingate protocol (6) that was applied after the RER was unity. This change appeared to be most beneficial for the OWAS (Table 3) who were more deconditioned (discussed below) and, perhaps, less familiar with cycling than the NWNL subjects. Albeit, mathematically, the Stand CE VO_{2} max values remain of slightly lesser magnitude than the TM VO_{2} max values, in both the NWNL and OWAS GXT’s, the study’s findings are encouraging enough for us to continue our efforts of refining the Stand CE approach to GXT. Thus, the findings of the present study were equivalent to or better than many CE studies where VO_{2} max values were substantially smaller than TM-VO_{2} max values (2,5,13,25,33). Our Stand CE offers an objective way to determine when to have CE subjects stand up and pedal (RER = 1.0), and what workload (Wingate) to add to achieve VO_{2} max during GXT.

We hypothesize that further technical refinement to the Stand CE protocol, using strategies such as standing up on the CE at a slightly lower RER (e.g., RER = 0.90) or at the nadir of the ventilation-equivalent (50) for oxygen or substantially increasing the revolutions per minute at the time of standing (e.g., + 20 rev·min^{-1}) may increase VO_{2} max and other cardiorespiratory values. These adjustments would likely allow the Stand CE metabolic values to become equal to TM cardiorespiratory values in many non-athletic, deconditioned, and/or diseased populations. We believe additional modifications to optimize and refine our Stand CE technique is warranted. We encourage researchers to test the Stand CE in other diseased populations.

**Effect of Being Overweight and Asthma on Cardiorespiratory Variables with Exercise**

The second purpose of this pilot study was to determine if the combined effects of being overweight and having asthma would affect submaximal and maximal exercise cardiorespiratory variables differently compared with normal weight individuals with normal lungs. We hypothesized that since both asthma and being overweight (a mild degree of obesity) are diseases with marked inflammatory components (14,43,44,53), that these two diseases might interact in either an additive or synergistic manner to show that overweight subjects with asthma have cardiorespiratory impairments that would be detectable during exercise. These exercise cardiorespiratory impairments might be manifested in ventilation-related variables and/or in oxygen kinetics’ variables during a progressive (submaximal to maximal) GXT or under steady-state submaximal exercise intensities (23,50).

**Ventilation Variables with Exercise**

We hypothesized that the ventilation impairments, if any, would occur via the mechanisms of a subtle increase in airway inflammation, as airway edema is persistent in asthma even if symptoms are episodic or EIB (i.e., airway smooth muscle contraction) (11,43,45). The results of this pilot study, in general, do not support our hypothesis of ventilation impairments during exercise due to having the combination of mild stable asthma and being overweight (Tables 2 & 3). The ventilation variables at submaximal intensities (Table 2) showed no statistical differences on RR@AT, V_{E}/VO_{2}@AT, V_{E}/VCO_{2}@AT, PETO_{2}@AT, and PETCO_{2}@AT across trials and groups. Likewise, the maximal ventilation variables (Table 3) showed no statistical differences between groups on RER max, RR max, V_{E}/VO_{2} max, V_{E}/VCO_{2} max, PETO_{2} max, and PETCO_{2} max across trials and groups. It is noteworthy to point out that the composite (collapsed across trials) breathing reserve (50) for the maximal GXT for both groups was 66%. In general, having a large breathing reserve at the conclusion of a GXT is taken as evidence of normal lung (bellows) function.

Also, it is important to point out that it was difficult to find studies (on breathing reserve and other cardiorespiratory variables) that closely matched our study’s population on degree (mild) of asthma, body composition (i.e., being overweight vs. obese), and on age (young adults vs. children or adolescents) to compare (18,48). Regardless, our breathing reserve findings were similar to
studies comparing normal lung individuals with people with asthma (12,42). One mixed-gender study used adolescents (42) and the other mixed-gender study used older adults (12), but both studies demonstrated that the breathing reserve in people with asthma was similar to individuals with normal lungs.

Of interest in our study, at the AT (Table 2), the submaximal exercise PETCO₂ values in the OWAS group, judged from a clinical basis, were either elevated (TM trial) or borderline elevated (Sit CE trial) (50). It may be that our OWAS demonstrated a dulled response to carbon dioxide tension early in exercise (16,50). At the time that the OWAS’s PETCO₂ values were slightly abnormal, their Vₑ/VCO₂@AT values were within normal limits and did not differ from the NWNL subjects. This implies a normal ventilation to perfusion ratio existed so as to rule out a ventilation-perfusion mismatch explanation of the elevated PETCO₂ values (50). However, at VO₂ max, the OWAS’s PETCO₂ values (Table 3) were within normal limits and were not different from the NWNL subjects. Thus, we cannot be certain as to whether the OWAS exhibited a dulled response to carbon dioxide early in exercise or if this was an aberration due to the small sample size of OWAS group. Further investigation is warranted.

Other possible ways that having asthma and being overweight could have affected exercise were through an increased work of breathing or gas exchange (external respiration) impairment (8,31,40). However, in general, since we observed no marked evidence of altered ventilation variables with submaximal (Table 2) or maximal exercise (Table 3), inappropriate dyspnea, or observable cyanosis, we conclude that there was no evidence of an increased work of breathing or gas exchange impairment in the OWAS group (40,50). However, it should be noted that others have shown gas exchange impairments during exercise in people with mild-moderate asthma (16,17).

Consistent with the lack of exercise ventilation impairments in the OWAS group was that the post exercise spirometry values (Figure 3), notably the FEV₁.0 and FEF₂₅₋₇₅, for both groups, did not decrease as would occur with a marked increase in airway inflammation or EIB (49,50). The lack of change in the FVC was suggestive that lung volumes did not change due to air-trapping (hyperinflation), as any increase in the functional residual capacity (FRC = expiratory reserve volume + residual volume) on a breath by breath basis, would ultimately encroach upon the FVC (17). To be noted was that the Vₑ values (Table 3) for the OWAS was similar between trials. This is important in regards to EIB as the volume of Vₑ is considered to be the crucial determinant in causing EIB, and not the exercise modality (51). Since there was no difference in Vₑ between the CE trials and TM trial, one would not expect a difference in the frequency of EIB between modes. Lastly, again, since no OWAS subject was observed to have inappropriate dyspnea, excessive coughing, during or after exercise, this suggests that there was not a subtle increase in airway inflammation that was not detected by spirometry (14,28,40,50).

It has been reported that a 1% increase in BMI is associated with a 9% increase in EIB (53). Thus, a few members of our OWAS group should have demonstrated EIB. However, this did not occur, and might simply be evidence of our subjects having very stable mild asthma (35). It should also be noted that spirometry values are generally not affected by being overweight (except perhaps expiratory reserve volume), in contrast to the effect of pronounced obesity, thus our FVC findings would be as expected based upon the OWAS’s BMI values (30,40). Lastly, in regards to ventilation and exercise, it is to be noted that in the present study, there was no significant correlation between FEV₁.0 and VO₂ max, which is generally consistent with most asthma and exercise (and training) investigations (12,16,18,32).
Effect of Being Overweight and Having Asthma on Oxygen Kinetics with Exercise

The most important finding of clinical significance, demonstrated during the maximal GXT trials, was that VO\textsubscript{2} max and maximal O\textsubscript{2} pulse of the NWNL group were substantially and consistently greater than the OWAS group (Figure 1 & Table 3). The differences between groups in these two variables suggest that these differences were independent of mode of exercise and, therefore, are an effect of group. We believe that the higher VO\textsubscript{2} max and O\textsubscript{2} pulse values of the NWNL group were simply a reflection of their greater aerobic fitness over the OWAS group. Deconditioning in the OWAS group (Table 3) was confirmed by the OWAS group’s reduced VO\textsubscript{2} max values that concomitantly occurred with RER max and HR max values that demonstrated that they exercised to an adequate intensity and didn’t quit GXT due to poor effort (7,21,33). Also consistent with poor conditioning were that the OWAS group’s VO\textsubscript{2}@AT and HR@AT (Table 2) were lower and higher, respectively, at submaximal intensities than the NWNL group’s during exercise (50). Additionally, the OWAS duration on the steady-state trial (Figure 2) was shorter than the NWNL participants. The statistically increased resting HR of the OWAS (Table 1) would also be consistent with deconditioning. The differences in VO\textsubscript{2} max between groups was so marked, that using logistic regression, employing only the TM-VO\textsubscript{2} max value as the single predictor variable, one could retrospectively accurately classify 80% of the OWAS and 93% of the NWNL participants into the correct groupings (24). Lastly, the effect size on VO\textsubscript{2} max was 1.540 while for VO\textsubscript{2}@AT it was 2.503, both of which values were markedly increased.

Mathematically, in support of the OWAS having decreased aerobic fitness, their composite VO\textsubscript{2} max value (~30 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}, Figure 1) would be well-below the 50th percentile (estimated 50th percentile value based upon the mix of exercise modes, age, and gender) for this group (20). This contrasts with the NWNL composite VO\textsubscript{2} max value (~38 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}), which would be at approximately the (estimated as above) 50th percentile. Since the OWAS had no history of heart disease, electrocardiogram abnormalities, or a decrease in O\textsubscript{2} pulse at higher exercise intensities, poor aerobic fitness was the most probable explanation. The effect of deconditioning on exercise in our study was greater than a study using subjects with less stable asthma (31). However, McNichol and colleagues (31) concluded that the low prevalence of deconditioning in their study was related to the young age and lack of comorbidities that would contribute to not exercising regularly. We assert that the main difference in deconditioning prevalence between these two studies is simply due to our less stringent criteria of defining deconditioning. Regardless, what is of most concern in the OWAS group of the present study is that their VO\textsubscript{2} max values are well below sedentary NWNL group at such a young age (Tables 1 & 3, and Figure 1), and the decrease in OWAS VO\textsubscript{2} max values occurred in the absence of obesity.

The VO\textsubscript{2} max findings of the present study are similar to mixed-gender study (42) where obese adolescents with asthma had lower VO\textsubscript{2} max values (VO\textsubscript{2} max ~21 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}) than healthy controls (~35 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}). However, our findings contrast with two mixed-gender studies where adolescents (34) with mild but persistent asthma had similar VO\textsubscript{2} max values compared to controls, or adults (mean age 31 yrs) (12) with mild-moderate stable asthma, which demonstrated VO\textsubscript{2} max values that were not different from predicted (normal) values. It is uncertain if the differences in VO\textsubscript{2} max findings between these last two cited studies and our study are due to our OWAS subjects being more deconditioned. In fact, a recent meta-analysis, the very first one on the effects of training on adults with asthma, is consistent with our findings of a reduced VO\textsubscript{2} max in young adults with asthma (18). Thus, our study and the meta-analysis, considered together, suggest that aerobic training interventions might be needed in adolescence or early adulthood in people with asthma to avoid a reduced VO\textsubscript{2} max later in life.
The steady-state submaximal trial (Figure 2) illustrates that the oxygen kinetics (23) of the OWAS group was parallel to, but simply lower in magnitude, than the NWNL group throughout the trial. The transition from the 40% intensity to the 60% intensity and the 60% to 80% intensity demonstrated similar slopes implying that Phase I oxygen kinetics (cardiac dynamic phase) and Phase II oxygen kinetics (venous blood returning to the lungs from exercising muscles) were preserved. This suggests the OWAS subjects showed no evidence of a reduced cardiovascular response, ventilation impairment, or ventilation to perfusion mismatching with exercise (23). The slopes of the plateaus (Phase III steady state oxygen kinetics) during the 40% and 60% VO2 max workloads were not different between groups. The steeper Phase III slope during the 80% of VO2 max workload of both groups, compared with the slopes at the 40% and 60% VO2 max workloads, was probably due to the subjects exercising above AT (23). The appearance (empirical) of the increased slope of the OWAS group during the 80% workload, compared to the NWNL group, was consistent with OWAS being deconditioned.

The third purpose of this study was to establish effect size, for a subsequent larger study, on the effects of asthma and being overweight on cardiorespiratory variables with exercise. The effect sizes on body composition, spirometry, and oxygen kinetics-related variables were, in general, substantial. In contrast, the effect size for several ventilation-related variables was inadequate. Regardless, we now have the apt information (effect sizes and standard deviations) to be able to estimate an adequate sample size for this subsequent study. In our sample size estimates, for this future study, we will use an idealized power value of 0.80 and a level of significance of 0.05 (24).

Limitations of Study
This pilot study has several limitations. The foremost issue was that we were unable to measure airway inflammation before, during, and immediately after exercise using an inflammation method/marker such as exhaled nitric oxide levels (8,32). It would have been of great value to know if the OWAS airway inflammation level was similar to the NWNL values and also to determine if GXT increased airway inflammation in the OWAS over the NWNL (8). A second limitation was our inability to recruit more OWAS subjects for an “exercise” study to provide for equal-sized groups. A third limitation of this study was that subjects may or may not have been at a “true” VO2 max using classic VO2 max criteria (plateau in VO2, RER >1.15, >85% HR max, etc.) (7,50). However, we believe our subjects were at, or close to, their true VO2 max values, and we have discussed this limitation in detail previously (26,33), as have others (13). Lastly, power values for some of our analyses, presented above, were inadequate. However, with the data obtained from this pilot study, we are now able to be able to estimate adequate sample size for a future larger-sized study investigating the effects of asthma and being overweight on cardiorespiratory variables with exercise (24).

CONCLUSIONS
The findings of this study cannot be generalized to overweight people with asthma due to the small sample size utilized in this pilot study. Yet, the results of this study do suggest that being both overweight and having mild stable asthma had little, if any, effect on cardiorespiratory variables during exercise due to the proposed effects of airway inflammation or EIB. The findings also suggest that the VO2 max of young adults with asthma should be measured to determine if an aerobic training program (intervention) is needed even before a person with asthma becomes overweight or obese. This study also suggests that young stable people with asthma can be safely exercised to high exercise intensities with little probability of an untoward event occurring, although adequate safety precautions should always be employed. Lastly, this study suggests that the
addition of final increased workload (Wingate) slightly improved the Stand CE approach to GXT over our previous work. We believe future refinement of the Stand CE may lead to improvements in the Stand CE GXT. More research of the Stand CE GXT is warranted because being able to obtain CE cardiorespiratory values that are statistically and mathematically equivalent to TM metabolic values, with CE’s concomitant safety and physiological monitoring advantages, may lead to the Stand CE GXT technique becoming either the preferred or alternate method of GXT.

LIST OF ABBREVIATIONS (and some brief definitions): AT, anaerobic threshold (alleged point where anaerobic metabolism begins); BTPS, body temperature and pressure saturated with water; EIB, exercise-induced bronchospasm; FEV1.0, forced expiratory volume in one second; FEV1.0 percent predicted, measured FEV1.0 divided by predicted FEV1.0 * 100; FVC, forced vital capacity; FVC percent predicted, measured FVC divided by predicted FVC * 100; GXT, graded exercise test (i.e. VO2 max test); HR@AT, heart rate at the anaerobic threshold; MET, metabolic equivalent (multiples of resting oxygen consumption); NWNL, normal weight group/participants; O2 pulse, oxygen pulse (VO2 max/HR max); OWAS, overweight and asthma group/participants; PETO2, peak end-tidal oxygen tension at the anaerobic threshold; PETCO2, peak end-tidal carbon dioxide tension at the anaerobic threshold; PETO2 max, maximal peak end-tidal tension for oxygen; PETCO2 max, maximal peak end-tidal tension for carbon dioxide; Phase I, II, & III, phases of oxygen kinetics (I = cardio-dynamic; II = venous blood arrival at lungs; III = steady state); RER, respiratory exchange ratio (VCO2 max/VO2 max); RER max, maximal respiratory exchange ratio; RR@AT, respiratory rate at the anaerobic threshold; SaO2, oxygen saturation; Sit CE, sit throughout cycle ergometry trial; Stand CE, stand up and cycle ergometry trial; STPD, standard temperature and pressure dry; TM, treadmill; TM-GXT, graded exercise testing via treadmill; VCO2, carbon dioxide production; VCO2 max, maximal carbon dioxide production; VE, minute ventilation; VE max, maximal minute ventilation; VE/VO2@AT, ventilation equivalent for oxygen at the anaerobic threshold; VE/VCO2@AT, ventilation equivalent for carbon dioxide at the anaerobic threshold; VE/VO2 max, maximal ventilation equivalent for oxygen; VE/VCO2 max, maximal ventilation equivalent for carbon dioxide; VT, tidal volume; VT max, maximal tidal volume; VT@AT, tidal volume at the anaerobic threshold; VO2 oxygen consumption; VO2 max, maximal oxygen consumption; and VO2@AT, oxygen consumption at the anaerobic threshold.

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Address for correspondence: William B. Kist, PhD, Basic and Pharmaceutical Sciences, St. Louis College of Pharmacy, 4588 Parkview Place, St. Louis MO 63110, Phone: 314-446-8484, FAX: 314-446-8460, and Email: william.kist@stlcop.edu

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