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Combined effects of mild-to-moderate obesity and asthma on physiological and sensory responses to exercise

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ABSTRACT

Despite the close link between asthma and obesity, there are no studies that have evaluated the sensory and physiological responses to exercise in obese asthmatics. We recently demonstrated that normal weight asthmatics with well controlled disease have preserved cardiorespiratory and sensory responses to exercise relative to non-asthmatic controls. However, these similarities may not hold true in patients with combined obesity and asthma. Accordingly, we sought to determine if combined asthma and obesity was associated with deleterious effects on cardiorespiratory fitness, exercise performance, dyspnoea, and physiological responses to exercise. Fourteen well-controlled obese asthmatics and fourteen age-matched normal weight asthmatics performed routine spirometry and underwent an incremental cardiopulmonary cycle test to assess the ventilatory, pulmonary gas exchange, cardiovascular, and sensory responses to exercise. Groups were well matched for age, height, spirometry, and asthma control. Obese asthmatics had a significantly greater body mass index (33 ± 3 vs. 23 ± 1 kg/m^2, $p < 0.001$) and lower self-reported activity levels by 47 % relative to normal weight asthmatics ($p < 0.05$). Obese asthmatics had a significantly lower maximal oxygen uptake (VO_2) (82 ± 14 vs. 92 ± 10 %predicted) and work rate (75 ± 8 vs. 89 ± 13 %predicted) relative to normal weight asthmatics ($p < 0.05$). The anaerobic threshold occurred at a lower VO_2 in obese asthmatics vs. normal weight asthmatics (54 ± 15 vs. 66 ± 16 %predicted, $p < 0.05$). Ventilatory responses were superimposed throughout exercise with no evidence of a ventilatory limitation in either group. Cardiovascular responses were normal in both groups. Dyspnoea responses were similar but the obese asthmatics experienced greater leg fatigue ratings at submaximal work rates. In conclusion, obese individuals with well controlled asthma have reduced cardiorespiratory fitness and greater leg fatigue ratings relative to normal weight asthmatics. The relatively reduced cardiorespiratory fitness and exercise performance in obese compared to normal weight asthmatics is most likely driven by their more sedentary lifestyle and resultant deconditioning rather than due to respiratory factors.

1. Introduction

The prevalence of obesity is rising at an alarming rate throughout the world, particularly in developing countries. Obesity is an established risk factor for cardiovascular disease, diabetes mellitus, certain cancers, and osteoarthritis among other conditions [1,2]. There is also a growing body of epidemiological data linking obesity and asthma [3–5]. Indeed, the vast majority of studies demonstrate an increased prevalence of asthma in overweight and obese individuals across multiple age groups and ethnicities [6–10]. Longitudinal data provides compelling evidence that obesity precedes asthma and that the relative risk of incident asthma increases with increasing body mass index (BMI) [11–13]. The link between asthma and obesity is reinforced by studies showing improvements in airway function, dyspnoea, use of rescue

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medications, and hospitalizations in asthmatics following surgical or diet-induced weight loss [14–17].

Obesity is associated with a number of physiological changes that may influence the cardiopulmonary and sensory responses to exercise. For example, obese individuals tend to adopt a more rapid and shallow breathing pattern relative to normal weight individuals [18,19]. Ofir et al. [19] found that obese women had higher metabolic and ventilatory requirements and greater dyspnoea and expiratory flow limitation during exercise compared to non-obese women. It stands to reason that the aforementioned ventilatory and sensory changes associated with obesity would be amplified in those with co-existing pulmonary conditions. Despite the close link between asthma and obesity, there are no studies that have evaluated the sensory and physiological responses to exercise in individuals with mild-to-moderate obesity and well controlled asthma. We recently demonstrated that normal weight asthmatics with well controlled disease have preserved cardiorespiratory and sensory responses to exercise relative to non-asthmatic controls [20]. However, these similarities may not hold true in patients with combined obesity and asthma. Therefore, we sought to determine if obese asthmatics have diminished aerobic fitness and altered cardiorespiratory and sensory responses to exercise relative to normal weight asthmatics. We hypothesized that obese asthmatics would have a reduced aerobic capacity, increased ventilatory limitations, and increases in dyspnoea and leg fatigue ratings relative to normal weight asthmatics.

2. Methods

2.1. Participants

Fourteen obese asthmatics (BMI $\geq$ 30 kg/m^2) were recruited from the Asthma Clinic at the National Institute of Respiratory Diseases in Mexico City. For comparison purposes, we also included data from fourteen normal weight asthmatics that participated in a previously published study [20]. The diagnosis of Asthma in both groups was determined using established criteria [21]. All asthmatics had to meet one of the following criteria: history of positive reversibility of 200 ml and 12% in forced vital capacity (FVC) or forced expiratory volume in 1 s (FEV_1) [22] and/or history of exercise induced bronchoconstriction [23]. Subjects in this study were also required to meet the criteria for well controlled asthma according to the Global Initiative for Asthma (GINA) Guidelines and the Asthma Control Test [24]. Participants were excluded if they had an acute exacerbation or were on oral corticosteroids, statins or beta blockers within 4 weeks of enrollment; had any disease that could limit or interfere with exercise testing; and if they were regularly participating in moderate or vigorous physical activity.

2.2. Experimental overview

The experimental protocol was based upon a similarly designed study in normal weight asthmatics [20]. The present study was approved by the National Institute of Respiratory Diseases Research Ethics Board (C15-11) and written informed consent was obtained from all participants. Participants were asked to refrain from their respiratory medications for 24 h; and caffeine, heavy meals and alcohol for at least 12 h prior to testing. On day 1, participants were screened for eligibility prior to the completion of medical history and physical activity (International Physical Activity Questionnaire – Short Form (IPAQ-SF)) questionnaires, anthropometric measurements, and pre- and post-bronchodilator spirometry. On day 2, subjects were instrumented with an arterial catheter followed by a symptom limited incremental cardiopulmonary exercise test.

2.3. Pulmonary function

Pre- and post-bronchodilator (400 μg salbutamol) spirometry was performed (Sensormedics Vmax 229, Yorba Linda, CA) according to recommended guidelines [25] and values were expressed as %predicted [26]. Spirometry and maximum voluntary ventilation (MVV) were performed prior to the exercise test by all participants on day 2.

2.4. Cardiopulmonary exercise testing

Exercise testing was performed using an electronically-braked cycle ergometer (Ergoselect 100, Ergoline, Germany) and a breath-by-breath cardiopulmonary testing system (Jaeger Oxycon-Pro, VIASYS Healthcare, Germany) with simultaneous measurement of electrocardiography. Details of the incremental exercise protocol have been described elsewhere [20]. The modified 10-point Borg scale was used to measure “breathing discomfort” and “leg fatigue” during exercise. The anaerobic threshold was determined using the V-slope method [27]. Predicted work rate values came from Jones [28], predicted VO_2 from Wasserman [29], and predicted heart rate was calculated as 210 − (age × 0.65).

2.5. Pulmonary gas exchange

Arterial catheterization of the radial artery was performed under local anesthesia (2% lidocaine). Arterial blood-gases were measured at moderate altitude (~2,240 m, barometric pressure = 585 mmHg, inspired PO_2 = 113 mmHg). Arterial blood-gases were measured after ~15 min of quiet breathing, with the subjects sitting upright, and at maximal exercise. Prior to each blood-gas sample, 5 ml of blood was withdrawn and discarded to eliminate dead space. Samples of 3 ml were then collected in pre-heparinized syringes. Air bubbles were immediately evacuated and the samples were analyzed within 30 s using a calibrated gas analyzer (ABL800 FLEX, Radiometer, Copenhagen, Denmark). Arterial oxygen saturation (%SaO_2) was measured using co-oximetry (i.e., multi-wavelength spectrophotometry). Arterial blood gases were corrected for axillary temperature (Suretemp® Plus 692, Welch Allyn, NY, USA). The ideal alveolar gas equation was used to calculate alveolar oxygen tension and the alveolar to arterial PO_2 difference (A-aDO_2).

2.6. Statistical analysis

Between-group comparisons of subject characteristics including maximal exercise responses were performed using unpaired t-tests. Comparisons of physiological and sensory responses at standardized submaximal work rates were examined using repeated measures ANOVA. To determine if group differences were present at various work rates, the interaction between group and work rate was tested, followed by Bonferroni-adjusted post hoc comparisons when results were significant. Statistical significance was set at $p < 0.05$. Results are reported as means ± SD unless otherwise specified.

3. Results

3.1. Subjects

Subject characteristics are reported in Table 1. Obese asthmatics and normal weight asthmatics were well matched for age, height, and years with asthma. Obese asthmatics included nine obese class I (BMI 30.0–34.9 kg/m^2) and five obese class II (BMI 35.0–39.9 kg/m^2). All obese subjects had body mass values $\geq$ 120% of ideal body