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Weight Loss Reduces Dyspnea on Exertion in Obese Women

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Abstract

During submaximal exercise, some otherwise healthy obese women experience breathlessness, or dyspnea on exertion (+DOE), while others have mild or no DOE (−DOE). We investigated whether weight loss could reduce DOE. 29 obese women were grouped based on their Ratings of Perceived Breathlessness (RPB) during constant load 60W cycling: +DOE (n=14, RPB≥4, 34±8yr, 36±3kg/m^2) and −DOE (n=15, RPB≤2, 32±8yr, 36±4kg/m^2) and then completed a 12-week weight loss program. Both groups lost a moderate amount of weight (+DOE: 6.6±2.4kg, −DOE: 8.4±3.5kg, $p<0.001$). RPB decreased significantly in the +DOE group (from 4.7±1.1 to 3.1±1.6) and remained low in the −DOE (from 1.5±0.7 to 1.6±1.1) (interaction $p<0.002$). Most physiological variables measured (i.e. body composition, fat distribution, pulmonary function, oxygen cost of breathing, cardiorespiratory measures) improved with weight loss; however, the decrease in RPB was not correlated with any of these variables ($p>0.05$). In conclusion, moderate weight loss was effective in reducing breathlessness on exertion in obese women who experienced DOE at baseline.

Keywords

Shortness of Breath; Body Composition; Pulmonary Function; Work of Breathing; Exercise

1. INTRODUCTION

The prevalence of obesity has increased dramatically over the past several decades. Currently, 33% of adults are classified as overweight and another 35% as obese in the United States (Ogden et al., 2014). Obesity is a complex multifactorial condition and is associated with a myriad of medical problems, such as type 2 diabetes, hypertension, stroke and heart attacks, sleep disordered breathing, and respiratory disorders (Azagury and Lautz, 2011; Kenchaiah et al., 2002; Van Gaal et al., 2006).

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Dyspnea on exertion (DOE) is also a very common symptom in obesity (Gibson, 2000; O'Donnell et al., 2010; Sin et al., 2002) and a major barrier in the management of obesity. We have repeatedly found approximately one-third of otherwise healthy obese women and men to experience an elevated intensity of dyspnea during submaximal constant load cycling exercise at 60 W or 105 W, respectively (or < 4 METs) (Babb et al., 2008a; Bernhardt and Babb, 2014; Bernhardt et al., 2013b). DOE and breathing discomfort may discourage obese individuals from being physically active.

It is unknown if weight loss alone (i.e. without aerobic exercise training) could decrease DOE in otherwise healthy obese women. This is a clinically important question as the American College of Sports Medicine, the National Institutes of Health, and other agencies recommend a combination of diet and aerobic exercise training for weight loss (1998; Donnelly et al., 2009). Additionally, the mechanism by which weight loss may improve DOE is also unclear; several factors could be involved, such as a decreased oxygen cost of breathing (Babb et al., 2008a), more efficient breathing mechanics and/or breathing pattern (Babb et al., 2002; Babb et al., 2008b), changes in body composition and fat distribution (Babb et al., 2008b), and changes is cardiorespiratory measures and/or exercise capacity (Babb et al., 2008a; Bernhardt and Babb, 2014).

The objectives of this study were to investigate 1) whether weight loss via a 12-week diet and resistance exercise program could reduce DOE in obese women who experienced DOE at baseline and 2) whether changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, and/or cardiorespiratory measures were associated with the potential reduction in DOE. We hypothesized that weight loss would decrease RPB, i.e. improve exertional dyspnea, during submaximal exercise in those obese women who experienced DOE before entering the weight loss program compared with those obese women who had no or only mild DOE at baseline. Furthermore, we hypothesized that changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, and/or cardiorespiratory measures during exercise, which could improve with weight loss, would be significantly associated with the decrease in RPB following weight loss.

2. METHODS

2.1 Subjects

Twenty-nine obese women were initially identified based on BMI ($\geq 30 \leq 50$ kg/m^2), which was confirmed by underwater weighing (body fat $\geq 30 \leq 55\%$). Exclusion criteria included history of smoking, asthma, cardiovascular disease, sleep disorders, or musculoskeletal abnormalities that would preclude maximal exercise. Subjects participating in regular vigorous exercise (exercise more than 2×/wk) during the last 6 months were also excluded. Written informed consent was obtained before participation in accordance with the University of Texas Southwestern IRB (STU122010-108).

The present study was designed as a pre-post intervention experiment. Some pre-intervention data have been previously published in manuscript (Bernhardt and Babb, 2014) and abstract form (Bassett et al., 2013; Bernhardt et al., 2013a; Bernhardt et al., 2014; Moran et al., 2013; Pineda et al., 2013).

Participants underwent the same testing procedures before and after a 12-week weight loss program. Testing was performed on four separate visits each before and after the intervention.

2.2. Body composition and pulmonary function (Visit 1)

Standard measures of height and weight were taken. Circumferences measurements were taken at the neck, chest, waist, and hips according to NHANES III guidelines (1994). Hydrostatic weighing was performed as previously described to determine percent body fat, lean body mass, and total body fat mass (Babb et al., 2008a; Babb et al., 2008b). Standard pulmonary function testing including spirometry, lung volume, and diffusing capacity was performed according to ATS/ERS guidelines (model V62W body plethysmograph, SensorMedics) (1995).

2.3 Cycling Exercise Testing (Visit 2)

2.3.1 Submaximal Constant Load Exercise—Before the exercise test, participants were given the following written instructions for Rating of Perceived Breathlessness (RPB): “The number 0 represents no breathlessness. The number 10 represents the strongest or greatest breathlessness that you have ever experienced. Each minute during the exercise test you will be asked to point to a number, which represents your perceived level of breathlessness at the time.” Exercise testing began with subjects seated on an electronically braked cycle ergometer (Lode Corival) with 3 minutes of resting baseline measurements after which a 6-minute constant load exercise cycling test at 60 W was initiated. This exercise work rate was chosen based on prior studies on obese women who obtained ventilatory threshold at approximately 60 W (DeLorey et al., 2005) and has been used previously to establish obese women with strong (i.e. +DOE) or no to mild DOE (i.e. −DOE) (Babb et al., 2008a; Bernhardt et al., 2013b). RPB was collected using the modified Borg scale (Borg, 1982) every two minutes of the test and the last value recorded was used for analysis. Cardiorespiratory responses, including heart rate (HR), blood pressure (SunTech Tango), ventilation ($\dot{V}_E$), and gas exchange ($\dot{V}O_2$ and $\dot{V}CO_2$), were measured at rest and throughout exercise (custom software, DUFIS).

2.3.2 Peak Cardiovascular Exercise Capacity—Peak exercise capacity was determined by graded cycle ergometry to volitional exhaustion or pedal rate ≤ 50 rpm. After resting baseline measurements, subjects started pedaling at 60–65 rpm with an initial work rate of 20 W. Work rate was increased by 20 W each minute until termination of the test; maximal effort was evidenced by achieving predicted peak heart rate > 90%, [lactate] > 7 mmol/L, and respiratory exchange rate > 1.1.

2.4 Oxygen Cost of Breathing (Visit 3)

The oxygen cost of breathing was determined from 6-min measurements of $\dot{V}O_2$ and $\dot{V}_E$ at rest and 4-min measurements of $\dot{V}O_2$ and $\dot{V}_E$ during eucapnic voluntary hyperpnea at 40L/min and 60L/min as previously described (Babb et al., 2008a). The oxygen cost of breathing was assessed by calculating the slope of the $\dot{V}O_2$ (ml/min) versus $\dot{V}_E$ (L/min) relationship at rest and during the two levels of hyperpnea. Linearity of the slope was checked for each subject (r^2 range 0.91–1.00).

2.5 Body fat distribution (Visit 4)

Multiple T2-weighted, water-suppressed, Magnetic Resonance Images (MRI) were taken from the sternal notch to the pubic symphysis to estimate fat distribution in the chest, abdominal, subcutaneous, visceral, and peripheral regions as previously described (Babb et al., 2008a; Babb et al., 2011; Bernhardt et al., 2013b). Images were analyzed using custom interactive software (Wafter 1.3).

2.6 Weight Loss Program

Each participant completed a 12-week weight loss program. They received a customized meal plan, weekly shopping lists and breakdown of individual meals and snacks. Additionally, they performed specific resistance exercises under supervision of a personal trainer three times per week. Subjects performed 8 resistive exercises (i.e. lifting weights) with 10 slow (i.e. concentric phase of ~10 s) repetitions each targeting all major muscle groups (i.e. upper body, lower body, core). The resistive exercises were utilized to increase - and/or minimize the loss of - lean body mass and thus increase basal metabolic rate. Aerobic exercise was not performed, so changes after the program could be attributed to weight loss only, not improvements in cardiorespiratory fitness. Participants were encouraged to lose 1–2 lb per week.

2.7 Data Analysis

The 29 obese women were assigned to one of two groups according to their RPB (0 – 10 Borg scale) during minute 6 of the constant load 60 W exercise test. Those with an RPB ≤ 2 were designated as obese women with no or mild dyspnea on exertion (−DOE, n = 15) and those with an RPB ≥ 4 were designated as obese women with strong dyspnea on exertion (+DOE, n = 14). Those women with an RPB = 3 were excluded from the study in order to better delineate differences between the +DOE and −DOE groups. The grouping was based on our previous finding that obese women have an average RPB of 2 ± 1 at ventilatory threshold during incremental exercise (DeLorey et al., 2005) and has been used in previous studies (Babb et al., 2008a; Bernhardt et al., 2013b).

Differences between +DOE and −DOE groups before and after the weight loss program were analyzed using a two-way ANOVA (i.e. weight loss and group) with a repeated measure on one factor (i.e. weight loss). Relationships among variables were determined with Spearman rank correlation. Data was analyzed using SAS 9.2.

3. RESULTS

Weight loss was achieved in all subjects (Figure 1). RPB during 60 W cycling decreased significantly in the +DOE group only (from 4.7±1.1 to 3.1±1.6), and expectedly remained low in the −DOE group (from 1.5±0.7 to 1.6±1.1) (group*WtLoss interaction p=0.002) (Figure 2).

3.1 Changes with weight loss

Table 1 shows the changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, and cardiorespiratory measures during constant load and peak exercise.

Body composition measures, including body mass index, lean body mass, fat mass, and body circumferences, decreased with weight loss in both groups ($p < 0.001$). Only waist:hip ratio did not change because both waist and hip circumferences decreased similarly.

Fat distribution did not change since fat mass was lost from all body regions, including chest, abdominal, visceral, subcutaneous, and peripheral areas.

Functional residual capacity (FRC) and expiratory reserve volume (ERV) improved by ~4% (as % of total lung capacity (TLC)) with weight loss ($p < 0.01$), with no significant changes in other lung subdivisions, spirometry, or diffusing capacity. Maximal voluntary ventilation (MVV), as a percent of predicted (%pred), was significantly different between the groups ($p < 0.001$), but it did not change with weight loss.

Oxygen cost of breathing decreased significantly with weight loss in both groups ($p < 0.01$) with no significant difference between groups before or after weight loss ($p > 0.05$).

Cardiorespiratory measures during submaximal constant load exercise at 60 W improved slightly after weight loss mainly in the +DOE group. Absolute $\dot{V}O_2$ (i.e. in L/min) was slightly, yet significantly decreased ($p < 0.001$) following weight loss with no difference between groups. Relative $\dot{V}O_2$ (i.e. as % of peak) was also decreased following weight loss with a significant difference between groups. Absolute (i.e. in L/min) and relative (i.e. as percent of MVV) $\dot{V}_E$ as well as RER and relative HR (as % of peak) showed an interaction effect (interaction weight loss * group, $p < 0.05$). Absolute HR (in bpm) and blood [lactate] decreased significantly with weight loss and were different between groups.

Cardiorespiratory measures at peak exercise were similar between groups. $\dot{V}O_2$, $\dot{V}_E$, and [lactate] were not different between groups and did not change with weight loss. There was a significant, yet small, increase in the achieved work rate in both groups (by ~8 W in the +DOE group and ~4 W in the −DOE group) and the time to exhaustion increased in the +DOE group by ~24 s and decreased in the −DOE group by ~6 s (interaction group * weight loss, $p = 0.019$). HR showed a significant, yet small, decrease following weight loss (by ~5 in the +DOE and by ~2 in the −DOE group).

3.2 Associations

Table 2 shows the Spearman rank correlations between the decreased RPB and changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, and cardiorespiratory measures during constant load and peak exercise in the +DOE group.

Even though all body composition and fat distribution measures decreased with weight loss, none of these changes were significantly correlated with the decrease in RPB ($p > 0.05$). Similarly, the changes in pulmonary function and oxygen cost of breathing following weight loss were not associated with the decreased RPB ($p > 0.05$).

Furthermore, even though there were some significant changes in cardiorespiratory measures during constant load and peak exercise (Table 1), none of these variables were significantly correlated with the decrease in RPB; except for RPE, which showed a weak correlation ($r = 0.16$, $p < 0.001$). Because of the changes in cardiorespiratory measures during exercise, we sought to estimate the effects of the most important measures on the change in RPB. We used a backward stepwise multiple regression analysis with ΔRPB as the dependent variable and $\Delta\dot{V}E$ (absolute and as a % of MVV), $\Delta\dot{V}O_2$ (absolute and as a % of peak $\dot{V}O_2$), ΔHR during 60 W cycling (absolute and as % of predicted), and Δpeak $\dot{V}O_2$ as the independent variables. Only the coefficients of ΔHR (%pred) [Partial $R^2 = 0.29$, $F = 6.95$, $p = 0.015$] and absolute $\Delta\dot{V}_E$ [Partial $R^2 = 0.14$, $F = 5.87$, $p = 0.023$] were significantly related to ΔRPB. The coefficient of multiple determination with these two variables was Model $R^2 = 0.43$, indicating that approximately 43% of the variance of the change in ΔRPB could be accounted for by ΔHR (%pred) and absolute $\Delta\dot{V}_E$. The regression equation generated yielded: $\Delta RPB = 0.28 + 0.12\ \Delta HR\ (\%pred) + 0.12\ \Delta\dot{V}_E$.

4. DISCUSSION

We found that 1) moderate weight loss via a 12-week diet and resistance exercise program could reduce ratings of breathlessness during submaximal exercise in obese women who experienced DOE at baseline (the +DOE group), which confirmed our hypothesis. Contrary to our hypothesis we found that 2) changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, or cardiorespiratory measures during exercise following weight loss were not associated with the reduction in RPB. Thus, although breathlessness during exercise is a common complaint in many obese individuals (Sin et al., 2002), the physiological mechanism(s) driving this symptom remain unclear. However, the significant decrease in dyspnea with only moderate weight loss of ~7 kg is very encouraging for obese individuals who struggle with breathlessness when exercising. Thus, moderate weight loss alone (i.e. without aerobic exercise training) appears to be an effective treatment to reduce exertional dyspnea in otherwise healthy, obese women.

To our knowledge, this is the first study to show the effect of weight loss on dyspnea ratings during exercise in otherwise healthy obese adults. Dyspnea at rest or low physical activity has been studied in obese patients undergoing invasive weight loss surgery. For example, bariatric surgery has been shown to result in significant improvements in self-reported dyspnea during activities of daily living (i.e. climbing two flights of stairs or walking with people of own age) (Karason et al., 2000) and quality of life (El-Gamal et al., 2005). Bariatric surgery results in dramatic weight loss and is a procedure with many associated risks. The present study demonstrates that moderate weight loss of 1–2 lb per week via a reduced calorie diet and resistive exercises three days per week significantly decreased DOE in obese women who had initially rated high breathlessness (i.e. RPB ≥ 4) during moderate cycling exercise at baseline testing. BMI decreased on average by 2.8 ± 1.3 kg/m^2 and total body fat decreased by 3.4 ± 2.3%. Only 6 of the 29 subjects (3 +DOE and 3 −DOE) had a drop in BMI into the overweight range (i.e. lowest BMI was 26), while all other subjects were still considered obese following the 12-week weight loss program (i.e. BMI > 30). Additionally, the decrease in DOE was independent not only of the amount of weight lost, but also of the initial body weight (data not shown). We conclude that moderate weight loss

achieved via diet and resistive exercises is effective in reducing DOE, which is very encouraging information for obese adults who get very breathlessness while exercising at moderate intensity.

Body composition and fat distribution could affect breathlessness, especially when excess fat is located primarily in the chest and abdomen. Abdominal fat displaces the diaphragm upward and impedes its downward movement during inspiration (DeLorey et al., 2005; Poulain et al., 2006). Fat deposition on the chest wall may impede expansion and excursion of the rib cage during inspiration, either via a direct loading effect or by changing intercostals muscle function. Central obesity, as measured by waist circumference and waist-to-hip ratio, has been shown to be detrimental to lung function (Steele et al., 2009). In the present study, all measures of body composition and fat distribution decreased with weight loss, except for waist-to-hip ratio due to similar decreases in both waist and hip circumferences (Table 1). However, these decreases were not correlated with the decrease in RPB during exercise (Table 2), suggesting that body composition and fat distribution do not have significant effects on breathlessness during exercise.

Obesity alters lung function; most consistently reported are reductions in FRC and ERV (Jones and Nzekwu, 2006; Pelosi et al., 1998). There is overwhelming evidence that significant weight loss improves lung function in both mild-to-moderate and severe obesity (Aaron et al., 2004; De Lorenzo et al., 1999; Emirgil and Sobol, 1973; Hakala et al., 1995; Santesson and Nordenstrom, 1978; Thomas et al., 1989; Wadstrom et al., 1991; Wei et al., 2011). We have previously shown that moderate weight loss improves breathing mechanics during exercise in obese men (Babb et al., 2011). In the present study, we also found improvements in FRC and ERV (Table 1); however, these changes were also not associated with the decreased RPB during exercise (Table 2). Thus, we conclude that pulmonary function also does not contribute significantly to the breathlessness experienced by some obese women during exercise.

In a previous study, we reported that the oxygen cost of breathing was significantly greater in obese women with DOE compared with obese women with no or mild DOE and that it was correlated with the RPB during submaximal exercise (Babb et al., 2008a). However, in the present cohort we failed to find this difference (Bernhardt et al., 2013a). This discrepancy may be due to the smaller numbers of subjects in the previous study (Babb et al., 2008a) and individual variability may have contributed to skewed results. In the present study, there was no significant difference in the oxygen cost of breathing between the two groups (Bernhardt et al., 2013a). Weight loss significantly decreased the oxygen cost of breathing in all subjects (Table 1), but it was not correlated with the decreased in RPB (Table 2), indicating that the work of breathing also did not play a role in the perception of DOE.

Obese individuals have higher oxygen consumption and minute ventilation at all work rates compared with normal weight subjects (Babb et al., 2002; Babb et al., 1991; Ofir et al., 2007; Whipp and Davis, 1984); however, breathlessness ratings at any given $\dot{V}O_2$ or $\dot{V}_E$ are similar (Ofir et al., 2007), suggesting that respiratory mechanical factors may not contribute to the DOE. In agreement with our previous studies (Babb et al., 2002; Babb et al., 2008a;

Lorenzo and Babb, 2011; Wood et al., 2008) and work by Dempsey (Dempsey et al., 1966), the subjects in the present study had normal ventilatory responses to exercise. There were no differences in the exercise responses between the obese women +DOE and −DOE that would explain why some women experienced much stronger breathlessness, implying that submaximal or maximal exercise variables also do not contribute significantly to breathlessness during exercise.

The lack of associations between the decreased RPB after weight loss and any of the physiological changes measured suggests that psychophysiological mechanisms might contribute to the decreased perception of DOE. Figure 3 shows the potential psychophysiological mechanisms of increased perception of breathlessness in obese individuals with DOE. Respiratory sensations are believed to be consciously perceived via a threshold-gated mechanism (Davenport and Reep, 1995). The idea is that only if ventilation is sufficiently changed or attended to, then this respiratory sensation will be gated through to the higher brain centers and the person becomes aware of his or her breathing (Chan and Davenport, 2008); whereas eucapnic breathing is usually subconscious. It is possible that the gating mechanism is different in those subjects who experience DOE compared with those who do not. Also, weight loss affect the gating of respiratory stimuli, i.e. fewer stimuli could be gated through to the cortex, such that the perception of breathlessness would be reduced following weight loss. There are at least 4 possibilities of why/how the respiratory sensation might be perceived higher in the +DOE subjects and why it might change with weight loss. 1) There could be a difference in the mechanical and/or chemical afferent feedback from the respiratory muscles. 2) There could be an adjustment in corollary discharge from the respiratory control center (el-Manshawi et al., 1986). 3) The gating threshold for sensory transmission might be altered (Davenport et al., 2007). And 4) input from gating modifiers, such as attention, experience, or emotions, may be different (Chan et al., 2012; Tsai et al., 2013; von Leupoldt et al., 2011). The more respiratory sensations are gated through to the sensory cortex, the higher the perception of the intensity of breathlessness would be. Also, the affective dimension of breathlessness could be increased. It is not clear if and how these mechanisms change with weight loss. Future studies are needed to investigate if any of these potential mechanisms play a role in DOE and if and how weight loss can change the gating of respiratory sensation.

4.1 Limitations

Subject recruitment and retention for this study was challenging due to various factors, such as intensive time commitment for the multiple testing sessions and the resistive exercise, and the requirement to follow a reduced calorie meal plan for 12 weeks. The limited number of subjects may reduce the generalization of the results, especially to higher obesity levels, older individuals, or patient populations. Nevertheless, a striking decrease in DOE was observed with a moderate weight loss in otherwise healthy obese women.

Future studies are needed to examine the effects of weight loss on DOE in obese men, as well as the effects of aerobic exercise training only (i.e. without weight loss) and a combined weight loss plus aerobic exercise training program on DOE in obese women and men in

order to investigate potential mechanism(s) of reduced DOE (i.e. fat mass reduction vs cardiovascular conditioning).

4.2 Conclusions

In conclusion, we found that moderate weight loss was effective in reducing breathlessness on exertion in obese women who experienced DOE at baseline. Thus, health care providers may recommend moderate weight loss as a first step to those otherwise healthy obese patients with strong exertional dyspnea. The physiological mechanism(s) of this reduction in DOE remains unclear since the decrease in RPB was not significantly associated with changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, or cardiorespiratory measures.

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Highlights

- Moderate weight loss effectively reduced dyspnea on exertion (DOE) in obese women
- Oxygen cost of breathing decreased following weight loss
- Body composition and pulmonary function improved with weight loss
- Reduction in DOE was not significantly associated with physiological changes measured

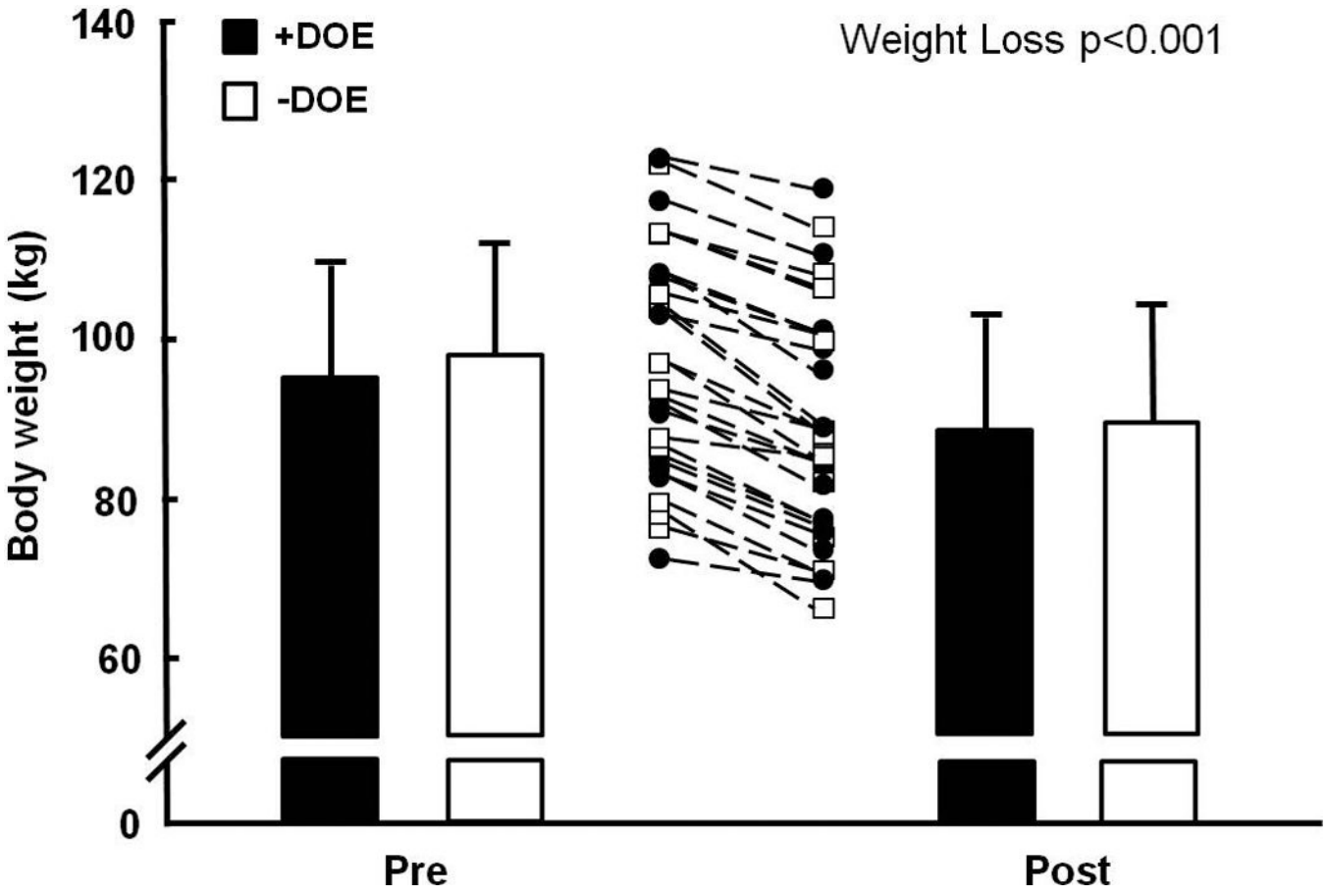

Figure 1.

Individual and mean decreases in body weight before and after a 12-week diet and resistance exercise program. +DOE, subjects with strong dyspnea on exertion; −DOE, subjects with no or mild dyspnea on exertion. Values are mean ± SD.

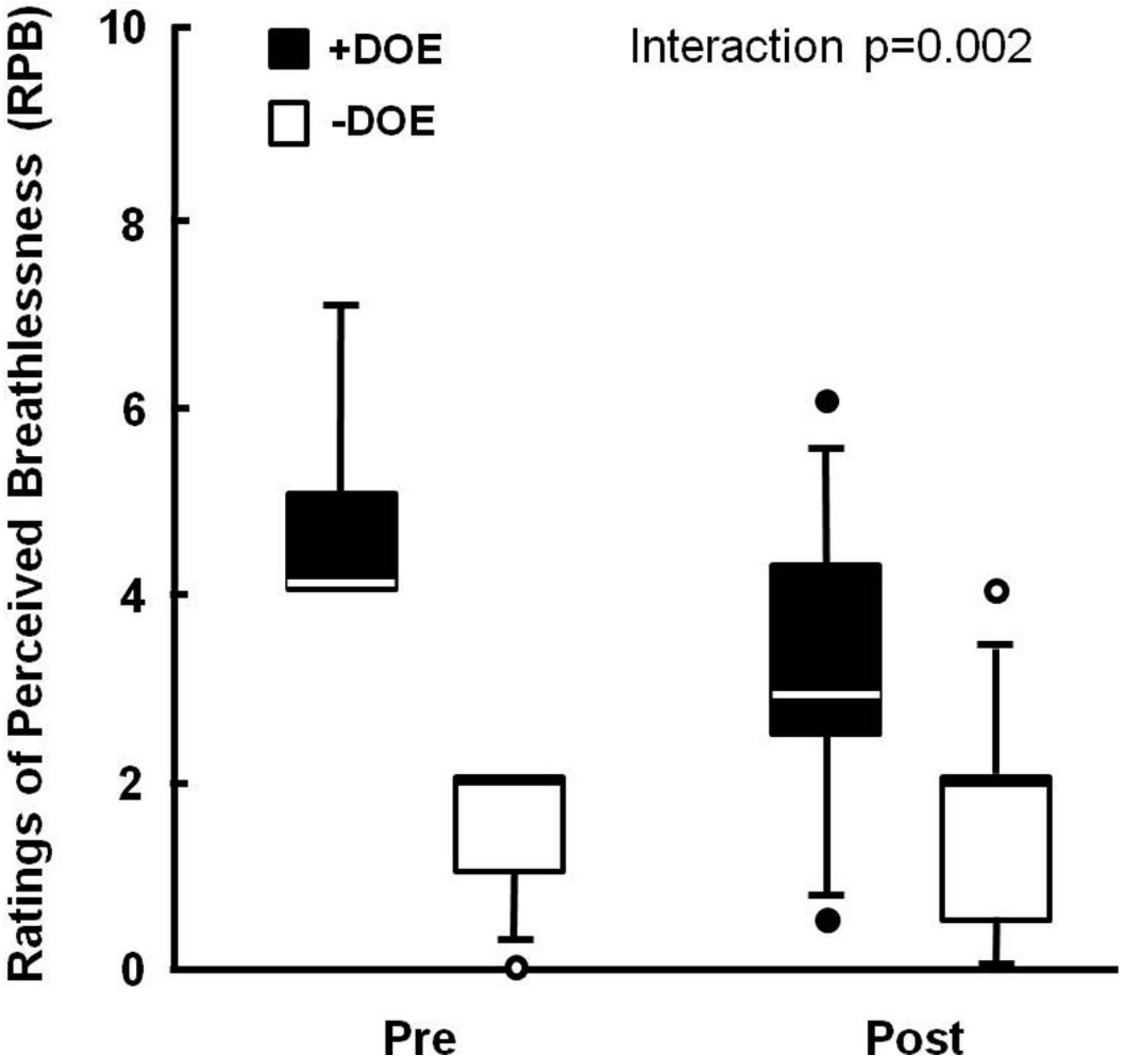

Figure 2.

Individual and group averages for Ratings of Perceived Breathlessness (RPB) before and after weight loss. RPB significantly decreased only in the +DOE group (Group × Weight Loss interaction p = 0.002). Box plots define the 25th and 75th percentiles by the ends of the boxes, with a line at the median and error bars defining the 10th and 90th percentiles.

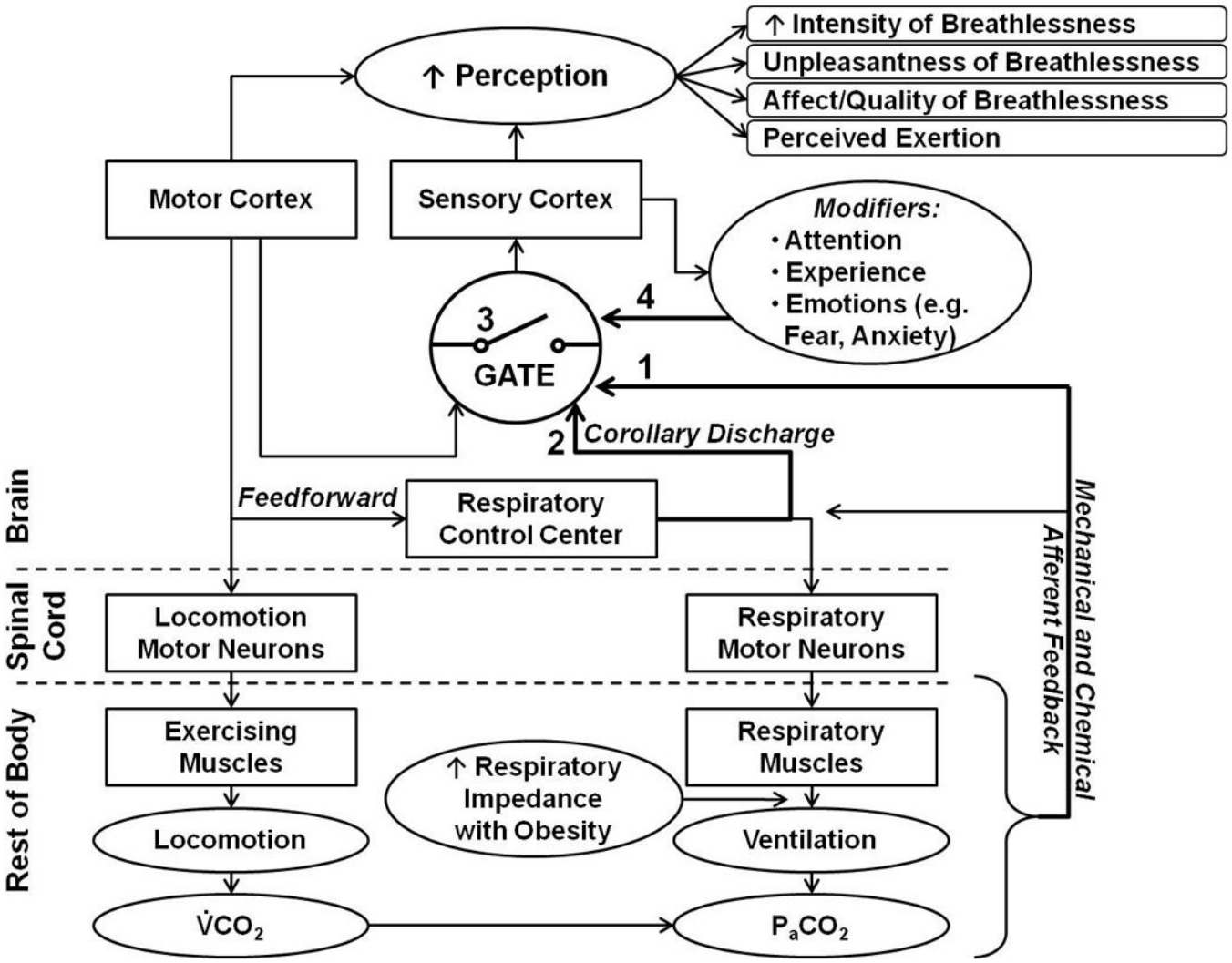

Figure 3.

Schematic of potential psychophysiological mechanisms of perception of breathlessness. Respiratory sensations are believed to be consciously perceived via a threshold-gated mechanism; only if ventilation is sufficiently changed or attended to, then this respiratory sensation will be gated through to the higher brain centers and the person becomes aware of his or her breathing. Potential mechanisms that might contribute to differential perception of DOE include: 1) altered afferent feedback, 2) modified corollary discharge from the respiratory control center, 3) changed gating threshold for sensory transmission, or 4) decreased or increased input from gating modifiers. The more respiratory sensations are gated through to the sensory cortex, the higher the perception of the intensity of breathlessness would be.

Table 1

Summary of mean changes in body composition, fat distribution, pulmonary function, oxygen cost of breathing, and cardiorespiratory exercise variables with weight loss (mean ± SD). P-value of interaction only shown when significant. NS, non-significant.

	+DOE	−DOE	P-value Weight Loss	P-value Group differences	P-value Interaction
Body composition					
ΔWeight (kg)	−6.6 ± 2.4	−8.4 ± 3.5	<0.001	NS	
ΔBody mass index (kg/m^2)	−2.4 ± 1.0	−3.1 ± 1.5	<0.001	NS	
ΔBody fat (%)	−2.8 ± 2.2	−3.9 ± 2.3	<0.001	NS	
ΔLean body mass (kg)	−1.3 ± 1.7	−1.3 ± 1.9	<0.001	NS	
ΔChest circumference (cm)	−3.2 ± 2.7	−5.0 ± 4.6	<0.001	NS	
ΔWaist circumference (cm)	−5.7 ± 4.6	−8.4 ± 8.3	<0.001	NS	
ΔHip circumference (cm)	−6.1 ± 3.9	−7.9 ± 4.5	<0.001	NS	
ΔNeck circumference (cm)	−1.0 ± 1.1	−1.2 ± 1.0	<0.001	NS	
ΔWaist:Hip ratio	−0.01 ± 0.04	−0.01 ± 0.07	NS	NS	
Fat distribution					
ΔChest Fat (kg)	−0.6 ± 0.4	−1.0 ± 0.5			0.036
ΔAbdominal Fat (kg)	−1.3 ± 0.7	−1.8 ± 1.1	<0.001	NS	
ΔVisceral Fat (kg)	−0.5 ± 0.3	−0.7 ± 0.6	<0.001	NS	
ΔSubcutaneous Fat (kg)	−1.8 ± 1.0	−2.5 ± 1.1	<0.001	NS	
ΔPeripheral Fat (kg)	−2.4 ± 2.2	−3.1 ± 1.4	<0.001	NS	
Pulmonary function					
ΔTLC (%pred)	0 ± 4	1 ± 3	NS	NS	
ΔFRC (L)	0.2 ± 0.2	0.2 ± 0.4	0.002	NS	
ΔFRC (%TLC)	4 ± 6	4 ± 8	0.005	NS	
ΔERV (%TLC)	4 ± 6	5 ± 11	0.008	NS	
ΔIC (%pred)	−4 ± 14	−1 ± 12	NS	NS	
ΔFVC (%pred)	1 ± 6	−1 ± 7	NS	NS	
ΔFEV$_1$ (%pred)	0 ± 5	−2 ± 6	NS	NS	

	+DOE	−DOE	P-value Weight Loss	P-value Group differences	P-value Interaction
ΔFEV$_1$/FVC ratio	−0.1 ± 2.9	−0.3 ± 2.1	NS	NS	
ΔMVV (%pred)	9 ± 13	5 ± 6	NS	0.001	
ΔDLCO (%pred)	1 ± 8	3 ± 8	NS	NS	
Oxygen cost of breathing					
ΔO$_2$cost Slope	−0.48 ± 0.70	−0.36 ± 0.67	0.003	NS	
Constant load exercise					
ΔRPB	−1.54 ± 1.45	0.10 ± 1.09			0.002
ΔRPE	−1.71 ± 2.49	0.27 ± 1.75			0.019
Δ$\dot{V}$O$_2$ (L/min)	−0.07 ± 0.05	−0.05 ± 0.05	<0.001	NS	
Δ$\dot{V}$O$_2$ (%peak)	−5 ± 5	−2 ± 6	0.034	0.002	
Δ$\dot{V}$E (L/min)	−7.20 ± 5.41	−2.57 ± 4.80			0.021
Δ$\dot{V}$E (%MVV)	−10 ± 5	−3 ± 3			0.001
ΔRER	−0.06 ± 0.07	−0.00 ± 0.08			0.044
ΔHR (bpm)	−12 ± 11	−2 ± 7	<0.001	0.013	
ΔHR (%peak)	−6 ± 6	−1 ± 4			0.029
Δ[Lactate] (mg/dL)	−1.0 ± 1.0	−0.4 ± 0.5	0.007	<0.001	
Peak exercise					
ΔWork rate (W)	8.6 ± 10.3	4.0 ± 13.5	0.009	0.014	
ΔTime (min)	0.40 ± 0.46	−0.10 ± 0.61			0.019
Δ$\dot{V}$O$_2$ (L/min)	0.04 ± 0.10	−0.03 ± 0.11	NS	NS	
Δ$\dot{V}$E (L/min)	1.56 ± 9.75	−1.10 ± 14.48	NS	NS	
ΔHR (bpm)	−5 ± 7	−2 ± 4	0.003	NS	
Δ[Lactate] (mg/dL)	−0.2 ± 1.1	−0.4 ± 1.0	NS	NS	

TLC, total lung capacity; FRC, functional residual capacity; ERV, expiratory reserve volume; IC, inspiratory capacity; FVC, forced vital capacity, FEV$_1$, forced expiratory volume in 1 s; MVV, maximal voluntary ventilation; DLCO, diffusing capacity of carbon monoxide; RPB, rating of perceived breathlessness; RPE, rating of perceived exertion; $\dot{V}O_2$, oxygen uptake; $\dot{V}E$, minute ventilation; RER, respiratory exchange rate; HR, heart rate.

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Table 2

Spearman correlations with decreased RPB in the +DOE group.

	Correlation *r* with ΔRPB	P-value
Body composition		
ΔWeight (kg)	0.15	0.61
ΔBody mass index (kg/m^2)	0.22	0.46
ΔBody fat (%)	0.07	0.82
ΔLean body mass (kg)	0.14	0.66
ΔChest circumference (cm)	0.40	0.15
ΔWaist circumference (cm)	−0.38	0.18
ΔHip circumference (cm)	0.24	0.41
ΔNeck circumference (cm)	0.38	0.18
ΔWaist:Hip ratio	−0.46	0.10
Fat distribution		
ΔChest Fat (kg)	−0.01	0.98
ΔAbdominal Fat (kg)	0.15	0.61
ΔVisceral Fat (kg)	0.17	0.57
ΔSubcutaneous Fat (kg)	0.03	0.93
ΔPeripheral Fat (kg)	−0.02	0.96
Pulmonary function variable		
ΔTLC (%pred)	0.17	0.56
ΔFRC (%TLC)	−0.35	0.23
ΔERV (%TLC)	−0.25	0.39
ΔFVC (%pred)	−0.05	0.87
ΔFEV_1 (%pred)	−0.11	0.72
ΔFEV_1/FVC ratio	−0.10	0.74
ΔMVV (%pred)	0.25	0.39
ΔDLCO (%pred)	−0.07	0.81
Oxygen cost of breathing		
ΔO_2cost Slope	0.12	0.68
Constant load exercise at 60 W		
ΔRPE	0.90	<0.0001
Δ$\dot{V}O_2$ (L/min)	0.14	0.64
Δ$\dot{V}O_2$ (%peak)	0.36	0.20
Δ$\dot{V}E$ (L/min)	0.40	0.16
Δ$\dot{V}E$ (%MVV)	0.39	0.17

	Correlation *r* with ΔRPB	P-value
ΔRER	−0.05	0.86
ΔHR (bpm)	0.29	0.32
Δ [Lactate] (mg/dL)	0.09	0.77
Peak exercise		
ΔWork rate (W)	0.06	0.85
ΔTime (min)	0.04	0.87
Δ$\dot{V}O_2$ (L/min)	−0.18	0.54
Δ$\dot{V}E$ (L/min)	−0.07	0.81
ΔHR (bpm)	0.32	0.27
Δ [Lactate] (mg/dL)	0.46	0.11

For abbreviations see Table 1.