



Ventilatory Responses at Peak Exercise in Endurance-Trained Obese Adults

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Background: Alterations in respiratory mechanics predispose healthy obese individuals to low lung volume breathing, which places them at risk of developing expiratory flow limitation (EFL). The high ventilatory demand in endurance-trained obese adults further increases their risk of developing EFL and increases their work of breathing. The objective of this study was to investigate the prevalence and magnitude of EFL in fit obese (FO) adults via measurements of breathing mechanics and ventilatory dynamics during exercise.

Methods: Ten (seven women and three men) FO (mean \pm SD, 38 ± 5 years, $38\% \pm 5\%$ body fat) and 10 (seven women and three men) control obese (CO) (38 ± 5 years, $39\% \pm 5\%$ body fat) subjects underwent hydrostatic weighing, pulmonary function testing, cycle exercise testing, and the determination of the oxygen cost of breathing during eucapnic voluntary hyperpnea.

Results: There were no differences in functional residual capacity ($43\% \pm 6\%$ vs $40\% \pm 9\%$ total lung capacity [TLC]), residual volume ($21\% \pm 4\%$ vs $21\% \pm 4\%$ TLC), or FVC ($111\% \pm 13\%$ vs $104\% \pm 15\%$ predicted) between FO and CO subjects, respectively. FO subjects had higher FEV₁ ($111\% \pm 13\%$ vs $99\% \pm 11\%$ predicted), TLC ($106\% \pm 14\%$ vs $94\% \pm 7\%$ predicted), peak expiratory flow ($123\% \pm 14\%$ vs $106\% \pm 13\%$ predicted), and maximal voluntary ventilation ($128\% \pm 15\%$ vs $106\% \pm 13\%$ predicted) than did CO subjects. Peak oxygen uptake ($129\% \pm 16\%$ vs $86\% \pm 15\%$ predicted), minute ventilation (128 ± 35 L/min vs 92 ± 25 L/min), and work rate (229 ± 54 W vs 166 ± 55 W) were higher in FO subjects. Mean inspiratory (4.65 ± 1.09 L/s vs 3.06 ± 1.21 L/s) and expiratory (4.15 ± 0.95 L/s vs 2.98 ± 0.76 L/s) flows were greater in FO subjects, which yielded a greater breathing frequency (51 ± 8 breaths/min vs 41 ± 10 breaths/min) at peak exercise in FO subjects. Mechanical ventilatory constraints in FO subjects were similar to those in CO subjects despite the greater ventilatory demand in FO subjects.

Conclusion: FO individuals achieve high ventilations by increasing breathing frequency, matching the elevated metabolic demand associated with high fitness. They do this without developing meaningful ventilatory constraints. Therefore, endurance-trained obese individuals with higher lung function are not limited by breathing mechanics during peak exercise, which may allow healthy obese adults to participate in vigorous exercise training.

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Abbreviations: CO = control obese; CRF = cardiorespiratory fitness; EELV = end-expiratory lung volume; EFL = expiratory flow limitation; FO = fit obese; IC = inspiratory capacity; MVV = maximal voluntary ventilation; PEF = peak expiratory flow; TLC = total lung capacity; \dot{V}_E = minute ventilation; \dot{V}_{O_2} = oxygen uptake; $\dot{V}_{O_{2peak}}$ = peak oxygen uptake; V_T = tidal volume

In contrast to the widespread belief that all obese adults are inactive, there are many obese individuals who exercise rigorously, compete in endurance races, and potentially have high levels of cardiorespiratory fitness (CRF). However, it is unclear whether this group of endurance-trained obese individuals encounters obesity-related respiratory limitations.¹⁻⁴

End-expiratory lung volume (EELV) increases in obese adults at or near maximal exercise,⁵⁻⁷ which may be in response to the presence of expiratory flow limitation (EFL).⁸ The high ventilatory demand in trained

athletes may exacerbate their work of breathing, as well as their risk of developing EFL.⁹⁻¹¹ However, little is known about CRF in “fit and obese” individuals; in addition, the mechanical mechanism by which they are able to generate the high ventilatory demand associated with increased physical fitness is unclear. Understanding these physiologic changes in endurance-trained obese individuals may provide valuable new insights for prescribing exercise training in obese adults.

We sought to investigate CRF, lung function, respiratory, and ventilatory dynamics during submaximal

and maximal exercise, and the oxygen cost of breathing in endurance-trained obese individuals. We hypothesized that endurance-trained obese subjects would experience considerable mechanical ventilatory constraints, which may cause them to hyperinflate especially during intense exercise. Although their oxygen cost of breathing would be similar to healthy sedentary obese adults, their work of breathing during peak exercise would be increased in proportion to their increase in peak ventilation.

MATERIALS AND METHODS

Subjects

In accordance with the institutional review board (University of Texas Southwestern Medical Center, approval number 122010-108), all details of the experiments were discussed with the volunteers, and informed consent was obtained before participation. All subjects were obese based on their percentage of body fat (body fat $\geq 30\%$) and had the same exclusion criteria: history of asthma, cardiovascular disease, or musculoskeletal abnormalities. We used percent body fat to determine obesity rather than BMI because BMI is a general measure of the relationship of weight to height and can underestimate the true level of obesity. For example, some of the subjects had a BMI that was $< 30 \text{ kg/m}^2$, but their percent body fat was $> 30\%$, which qualified them for inclusion in the study.

Fit Obese: A total of 19 fit obese (FO) participants were recruited for the study. However, nine were disqualified for different reasons: asthma (two), high BP (one), and body fat $< 30\%$ (six). Therefore, seven women and three men completed the study. Candidates for this study were screened carefully based on their exercise training history within the preceding 12 months. The subjects exercised aerobically at least four times per week, and their training sessions lasted 1 to 4 h. Moreover, they had recently (ie, within the preceding 12 months) competed in endurance events such as marathons, Ironmans, and road races, or were training to compete in future races.

Control Obese: Seven women and three men were randomly selected from our large database of prior and ongoing studies to

serve as a control group (control obese [CO]) for comparison purposes. These subjects had the same exclusion criteria as the FO subjects. However, they had not engaged in any regular exercise activities for 6 months prior to enrollment in the study.

Body Composition and Pulmonary Function

Hydrostatic weighing, with the measurement of residual volume, was performed to determine percent body fat, lean body mass, and total body fat mass. Participants underwent standard spirometry, lung volume, airway resistance, maximal inspiratory pressure, and maximal expiratory pressure determinations (model V62W body plethysmograph, SensorMedics).¹² Predicted values were based on published norms.¹³⁻¹⁶

Cardiorespiratory Responses and Rating of Perceived Breathlessness During Submaximal Exercise

A submaximal exercise test was performed to further evaluate and compare fitness levels between the groups. Testing began with the subjects seated on the cycle ergometer for 3 min; then the subjects performed a 6-min constant-load exercise cycling test at 60 W (women), or 105 W (men).¹⁷ The three CO men exercised at 90 W, rather than 105 W, as dictated by the requirements of a prior study. Physiologic data averaged from the last 2 min of the exercise stage were used in the analyses. Rating of perceived breathlessness and rating of perceived exertion were measured every 2 min of the test, and the last value recorded was used for analyses.

Peak Cardiorespiratory Exercise Capacity and Breathing Mechanics

Peak aerobic power, peak oxygen uptake ($\dot{V}_{O_2, \text{peak}}$) (open circuit spirometry), was determined by graded cycle ergometer exercise (model CPE 2000, Medical Graphics Corporation) to exhaustion as described previously.¹⁷ Expiratory and inspiratory flows were measured continuously at rest and during exercise as described previously.¹⁸ EELV was estimated from measurement of inspiratory capacity (IC) during each of the protocol stages, and total lung capacity (TLC) during body plethysmography ($\text{EELV} = \text{TLC} - \text{IC}$).^{5,6} All subjects performed an FVC maneuver before and 2 min after exercise, with the largest loop accepted. EFL was computed as the percentage of the expiratory tidal flow-volume loop that met or exceeded the expiratory boundary of the maximal flow-volume loop.

The Oxygen Cost of Breathing

The oxygen cost of breathing was determined from 6-min measurements of oxygen uptake (\dot{V}_{O_2}) and minute ventilation (\dot{V}_E) at rest and 4-min measurements of \dot{V}_{O_2} and \dot{V}_E during eucapnic voluntary hyperpnea at 40 L/min and 60 L/min (women), or 60 L/min and 90 L/min (men), as described previously.¹⁷ To maintain eucapnia during the voluntary hyperpnea maneuver, the subjects breathed from a 1,000-L inspiratory reservoir bag containing 4% or 5% CO_2 (21% oxygen and balance nitrogen).¹⁹ The oxygen cost of breathing was assessed by calculating the slope of the \dot{V}_{O_2} (mL/min) vs \dot{V}_E (L/min) relationship at rest and during eucapnic voluntary hyperpnea. Physiologic data were averaged from the 6-min measurements at rest and the 4-min measurements during the hyperventilation maneuvers.

Data Analyses

Differences between groups were determined by an independent Student *t* test. Values are reported as mean \pm SD. A *P* value of $< .05$ was considered significant.

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RESULTS

Subjects and Body Composition

All subjects were obese (body fat $\geq 30\%$). There were no differences ($P > .05$) between groups in body composition, age, height, or body size parameters (Table 1).

Pulmonary Function

Pulmonary function values are shown in Table 1 and Figure 1. The FO group had larger TLC than did the CO group ($106\% \pm 14\%$ vs $94\% \pm 7\%$ predicted, $P < .05$) (Fig 1A). However, there were no differences between the FO and CO groups in functional residual capacity ($43\% \pm 6\%$ vs $40\% \pm 9\%$ TLC) or residual volume ($21\% \pm 4\%$ vs $21\% \pm 4\%$ TLC).

FVC ($111\% \pm 13\%$ vs $104\% \pm 15\%$ predicted) was not different between the FO and CO groups, respectively (Fig 1B). However, the FO group had higher FEV₁ ($111\% \pm 13\%$ vs $99\% \pm 11\%$ predicted, $P < .05$), peak expiratory flow (PEF) ($123\% \pm 14\%$ vs $106\% \pm 13\%$ predicted, $P < .01$), and maximal voluntary ventilation (MVV) ($128\% \pm 15\%$ vs $106\% \pm 13\%$ predicted, $P < .01$) than did the CO group. There were no dif-

ferences between the FO and CO groups in maximal inspiratory pressure ($134\% \pm 26\%$ vs $122\% \pm 20\%$ predicted), maximal expiratory pressure ($109\% \pm 21\%$ vs $104\% \pm 28\%$ predicted), or airway resistance ($129\% \pm 30\%$ vs $129\% \pm 45\%$ predicted).

Cardiorespiratory Responses and Rating of Perceived Breathlessness During Submaximal Exercise

The FO group exercised at lower relative exercise intensities than did the CO group (Table 2). The FO group had significantly ($P < .05$) lower relative $\dot{V}O_2$ ($50\% \pm 7\%$ vs $64\% \pm 8\%$ $\dot{V}O_{2peak}$), respiratory exchange ratio (0.91 ± 0.08 vs 1.02 ± 0.09), relative heart rate ($66\% \pm 6\%$ vs $77\% \pm 9\%$ maximal heart rate), blood lactate (2.0 ± 1.2 mmol/L vs 4.5 ± 2.2 mmol/L), rating of perceived breathlessness (2.0 ± 1.6 vs 4.0 ± 1.6), and rating of perceived exertion (9.8 ± 2.1 vs 12.7 ± 2.3), respectively. In addition, the ventilatory response to exercise ($\dot{V}E/\dot{V}O_2$ slope) was significantly lower in the FO group compared with the CO group (26 ± 3 vs 31 ± 6). These observations strongly suggest that the individuals in the FO group had higher fitness levels than those in the CO group.

Table 1—Subject Characteristics and Pulmonary Function

Characteristic	Control Obese (n = 10 [7 Female])	Fit Obese (n = 10 [7 Female])
Age, y	37.6 \pm 5.0 (28-45)	37.6 \pm 5.3 (30-47)
Height, cm	170 \pm 9 (156-188)	169 \pm 10 (152-183)
Weight, kg	94.9 \pm 11.6 (84.6-116.8.0)	93.9 \pm 16.2 (73.5-112.0)
BMI, kg/m ²	33.1 \pm 3.2 (28.4-36.8)	32.6 \pm 3.6 (26.9-38.0)
Body fat, %	38.9 \pm 4.7 (32.5-45.7)	37.7 \pm 4.9 (29.7-43.9)
Fat mass, kg	36.6 \pm 3.5 (30.4-41.5)	35.3 \pm 7.9 (24.4-48.8)
Lean body mass, kg	58.3 \pm 11.0 (49.3-78.6)	58.6 \pm 10.8 (43.4-74.7)
TLC		
L	5.48 \pm 1.08 (4.09-7.89)	6.24 \pm 1.72 (3.97-9.44)
% predicted	94 \pm 7 (84-101)	106 \pm 14 ^a (88-129)
FRC		
L	2.20 \pm 0.62 (1.25-3.03)	2.65 \pm 0.71 (1.89-3.91)
% TLC	40 \pm 9 (27-51)	43 \pm 6 (35-57)
RV		
L	1.19 \pm 0.37 (0.79-1.94)	1.32 \pm 0.38 (0.74-2.05)
% TLC	21 \pm 4 (15-25)	21 \pm 4 (16-27)
FVC		
L	4.15 \pm 0.75 (3.20-5.75)	4.80 \pm 1.39 (3.23-7.23)
% predicted	104 \pm 15 (82-127)	111 \pm 13 (94-131)
FEV ₁		
L	3.24 \pm 0.50 (2.56-4.16)	3.94 \pm 1.09 (2.84-5.94)
% predicted	99 \pm 11 (81-112)	111 \pm 13 ^a (96-138)
PEF		
L/s	8.39 \pm 1.36 (6.76-10.81)	9.84 \pm 1.80 (8.41-14.03)
% predicted	106 \pm 13 (87-123)	123 \pm 14 ^a (106-147)
MVV		
L/min	133 \pm 23 (100-173)	161 \pm 36 (123-230)
% predicted	106 \pm 13 (89-126)	128 \pm 15 ^a (104-151)

Data are presented as means \pm SD (range). Predicted values for spirometry and lung volumes were based on the norms of Knudson et al,^{13,14} and Goldman and Becklake,¹⁶ respectively. FRC = functional residual capacity; MVV = measured maximal voluntary ventilation; PEF = peak expiratory flow; RV = residual volume; TLC = total lung capacity.

^a $P < .05$.

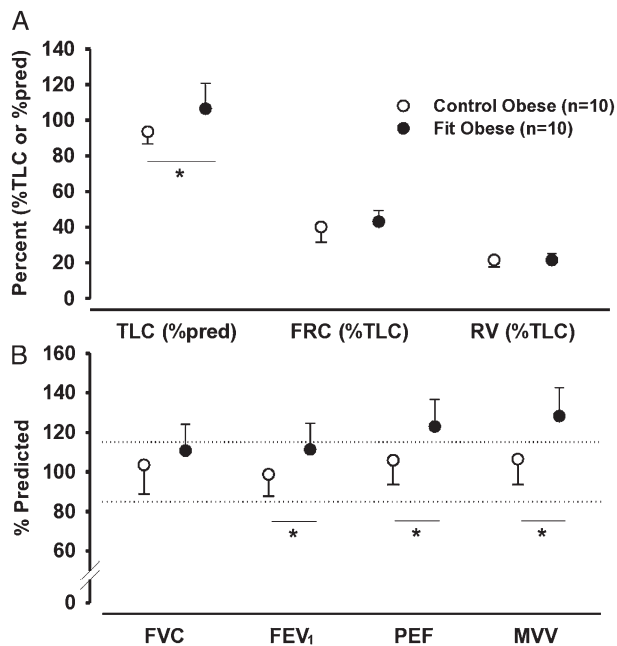


FIGURE 1. A, Lung volumes. B, Spirometry. Data are presented as mean and SD. Predicted values for spirometry and lung volumes were based on the norms of Knudson et al,^{13,14} and Goldman and Becklake,¹⁶ respectively. Dotted lines represent the normal range.²⁰ FRC = functional residual capacity (reported as %TLC); MVV = maximal voluntary ventilation; PEF = peak expiratory flow; %pred = percent predicted; RV = residual volume (reported as % total lung capacity); TLC = total lung capacity. **P* < .05.

Peak Cardiorespiratory Exercise Capacity

The FO group had increased CRF and exercise capacity (about 40%, as indicated by exercise time to exhaustion, peak work rate, and $\dot{V}O_{2peak}$) compared with the CO group (Table 3). The FO subjects further increased their $\dot{V}E$ by approximately 40% compared

with the CO subjects (128 ± 35 L/min vs 92 ± 25 L/min, *P* < .05). There were no differences in peak heart rate, oxygen saturation, end-tidal PCO_2 , respiratory exchange ratio, or peak lactate concentration between groups. FO individuals had shorter inspiratory time (0.562 ± 0.090 s vs 0.764 ± 0.185 s, *P* < .01) and expiratory time (0.631 ± 0.114 s vs 0.759 ± 0.172 s; *P* = .07) than did CO subjects. The FO group had significantly higher (*P* < .01) mean inspiratory (tidal volume [V_T]/inspiratory time) (4.65 ± 1.09 L/s vs 3.06 ± 1.21 L/s) and mean expiratory flows (V_T /expiratory time) (4.15 ± 0.95 L/s vs 2.98 ± 0.76 L/s) at peak exercise.

Figure 2 illustrates the ventilatory response ($\dot{V}E$ vs CO_2 output) (Fig 2A) and breathing pattern (breathing frequency [Fig 2B], V_T [Fig 2C]) during the peak exercise test. $\dot{V}E$ (128 ± 35 L/min vs 92 ± 25 L/min) and breathing frequency (51 ± 8 breaths/min vs 41 ± 10 breaths/min) at peak exercise were higher in the FO group (*P* < .05).

Breathing Mechanics

CO individuals with EFL and without EFL at peak exercise are shown in Figures 3A and 3B, respectively. FO subjects with EFL and without EFL at peak exercise are shown in Figures 3C and 3D. EFL was observed in four FO subjects (three women and one man) and in five CO subjects (3 women and two men) at peak exercise. However, the degree of flow limitation was mild (<20% V_T) and was not different between the FO and CO groups ($13\% \pm 6\%$ V_T vs $16\% \pm 7\%$ V_T , respectively). Figure 3E illustrates that there were no significant differences between the CO and FO groups regarding EELV and end-inspiratory lung volume at rest, submaximal, or peak exercise.

Table 2—Cardiorespiratory Responses to Submaximal (6-Min) Exercise

Parameters	Control Obese (n = 10 [7 Female])	Fit Obese (n = 10 [7 Female])
Work rate, ^a W	69 ± 14 (60-90)	74 ± 22 (60-105)
$\dot{V}O_2$, L/min	1.31 ± 0.24 (1.08-1.81)	1.54 ± 0.38 (1.14-2.17)
$\dot{V}O_2$, % peak	64 ± 8 (47-76)	50 ± 7 ^a (40-61)
$\dot{V}CO_2$, L/min	1.30 ± 0.21 (1.09-1.65)	1.41 ± 0.41 (1.05-2.07)
RER	1.02 ± 0.09 (0.91-1.19)	0.91 ± 0.08 ^a (0.77-1.04)
$\dot{V}E$, L/min	42 ± 7 (32-53)	39 ± 10 (28-54)
$\dot{V}E/\dot{V}CO_2$, slope	31 ± 6 (24-42)	26 ± 3 ^a (23-33)
PETCO ₂ , torr	41 ± 6 (32-51)	44 ± 3 (39-49)
SpO ₂ , %	99 ± 1 (98-100)	99 ± 1 (96-100)
Heart rate, bpm	139 ± 15 (108-155)	118 ± 13 ^a (99-143)
Heart rate, % max	77 ± 9 (61-91)	66 ± 6 ^a (58-75)
Lactate, mmol/L	4.5 ± 2.2 (2.3-8.8)	2.0 ± 1.2 ^a (0.9-4.9)
RPB, 0-10 scale	4.0 ± 1.6 (2-6)	2.0 ± 1.6 ^a (0-5)
RPE, 6-20 scale	12.7 ± 2.3 (9-15)	9.8 ± 2.1 ^a (7-13)

Data are presented as mean ± SD (range). The three fit obese men exercised at 105 W, whereas the three control obese men exercised at 90 W, rather than 105 W. All fit obese and control obese women exercised at 60 W. bpm = beats per min; PETCO₂ = end-tidal CO₂; RER = respiratory exchange ratio; RPB = rating of perceived breathlessness; RPE = rating of perceived exertion; SpO₂ = oxygen saturation; $\dot{V}CO_2$ = CO₂ uptake; $\dot{V}E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake.

^a*P* < .05.

Table 3—Peak Exercise Data

Parameters	Control Obese (n = 10 [7 Female])	Fit Obese (n = 10 [7 Female])
Exercise time, min	7.0 ± 1.1 (5.0-8.4)	10.0 ± 1.5 ^a (8.0-12.0)
Work rate, W	166 ± 55 (100-270)	229 ± 54 ^a (160-330)
Work rate, % pred	100 ± 17 (67-129)	140 ± 16 ^a (125-171)
$\dot{V}O_2$, L/min	2.10 ± 0.69 (1.39-3.29)	3.10 ± 0.87 ^a (2.13-4.80)
$\dot{V}O_2$, % predicted ^b	86 ± 15 (64-109)	129 ± 16 ^a (110-152)
$\dot{V}O_2$, mL/kg PWT/min	34 ± 8 (23-47)	50 ± 8 ^a (40-68)
$\dot{V}O_2$, mL/kg LBM/min	35 ± 6 (28-42)	52 ± 6 ^a (43-64)
$\dot{V}CO_2$, L/min	2.55 ± 0.85 (1.66-4.04)	3.73 ± 1.03 ^a (2.62-5.67)
RER	1.22 ± 0.07 (1.10-1.31)	1.21 ± 0.04 (1.13-1.26)
$\dot{V}E$, L/min	92 ± 25 (70-146)	128 ± 35 ^a (90-200)
$\dot{V}E$, % MVV	69 ± 12 (54-85)	79 ± 11 ^a (60-93)
$\dot{V}E/\dot{V}CO_2$	37 ± 5 (30-47)	35 ± 5 (30-43)
PETCO ₂ , torr	33 ± 5 (26-42)	33 ± 4 (25-38)
O ₂ saturation, %	99 ± 1 (97-100)	98 ± 1 (96-99)
Heart rate, bpm	181 ± 12 (164-200)	177 ± 7 (164-189)
Heart rate, % predicted	100 ± 6 (90-106)	96 ± 5 (87-105)
Lactate, mmol/L	7.9 ± 1.6 (6.1-11.0)	9.4 ± 2.0 (6.7-12.1)
RPB, 0-10 scale	7.5 ± 2.3 (4-10)	8.0 ± 1.6 (6-10)
RPE, 6-20 scale	17.6 ± 2.3 (13-20)	18.4 ± 1.3 (17-20)
Ti, s	0.764 ± 0.185 (0.472-1.113)	0.562 ± 0.090 ^a (0.453-0.744)
Te, s	0.759 ± 0.172 (0.502-1.053)	0.631 ± 0.114 ^a (0.502-0.862)
Vt/Ti, L/s	3.06 ± 1.21 (1.81-5.46)	4.65 ± 1.09 ^a (3.48-6.74)
Vt/Te, L/s	2.98 ± 0.76 (1.91-4.38)	4.15 ± 0.95 ^a (2.94-6.10)

Data are presented as mean ± SD (range). Exercise time = exercise time to peak; LBM = lean body mass; O₂ = oxygen; PWT = predicted weight²¹; TE = expiratory time; Ti = inspiratory time; Vt = tidal volume; Vt/Ti = mean inspiratory flow. See Table 1 and 2 legends for expansion of other abbreviations.

^aP < .05.

^bAlthough interpretation of peak $\dot{V}O_2$ in determining cardiovascular conditioning in obesity is a complex issue,²² the recommendation is to use a method whereby peak $\dot{V}O_2$ is compared with an age, sex, and weight-corrected predicted peak $\dot{V}O_2$.²¹ Thus, we used the following equation adapted from Wasserman et al,²³ Hansen et al,²⁴ and Wasserman and Whipp²⁵: predicted peak $\dot{V}O_2$ = (predicted peak $\dot{V}O_2$ in mL/min/kg × predicted weight) + [(actual weight – predicted weight) × 6 mL/kg]²³⁻²⁵ to predict peak $\dot{V}O_2$.

The Oxygen Cost of Breathing

The oxygen cost of breathing measured during eucapnic voluntary hyperpnea was not significantly different between the FO and CO groups (1.64 ± 0.63 mL O₂/L $\dot{V}E$ vs 2.14 ± 0.57 mL O₂/L $\dot{V}E$, respectively; P = .08). In addition, the $\dot{V}O_2$ of the respiratory muscles (estimated from the unit-corrected product of the oxygen cost slope and $\dot{V}E$) at peak exercise was not different between the FO and CO groups (206 ± 89 mL/min vs 201 ± 83 mL/min, P = .89; 6.9% ± 2.8% vs 9.6% ± 3.1% $\dot{V}O_{2peak}$, P = .05). This was despite the 40% increase in $\dot{V}E$ in the FO group.

DISCUSSION

The results of this study demonstrate several important findings. Obese individuals are capable of achieving high CRF levels. Only 40% to 50% of the FO or CO subjects experienced EFL (and then only minimal) at peak exercise, and operational lung volumes were not different between groups. Nevertheless, FO individuals were able to increase ventilation by about 40% at peak exercise without a considerable increase in mechanical ventilatory constraints com-

pared with CO subjects. This increase in $\dot{V}E$ resulted from the FO subjects having higher mean inspiratory and expiratory flows, which allowed them to shorten their breathing cycle time and increase $\dot{V}E$ by further increasing breathing frequency. In this sample of FO subjects, their pulmonary function was in the upper limits of normal. Finally, the oxygen cost of breathing in FO subjects was not higher, even at peak exercise, because of the increase in frequency strategy vs increasing VT.

Cardiorespiratory Fitness

This is the first study, to our knowledge, that compared CRF between sedentary and endurance-trained obese adults, and it showed that high CRF can be achieved in this population despite the presence of obesity. The FO group had a 43% increase in exercise time to exhaustion, a 38% increase in peak work rate, and a 48% increase in $\dot{V}O_{2peak}$ compared with their control counterparts. Although quantifying and interpreting CRF in obesity is a complex issue,²² we strongly believe that predicted $\dot{V}O_{2peak}$ values allow for a normalized evaluation of CRF in the obese population. $\dot{V}O_{2peak}$ and peak power output in the FO group

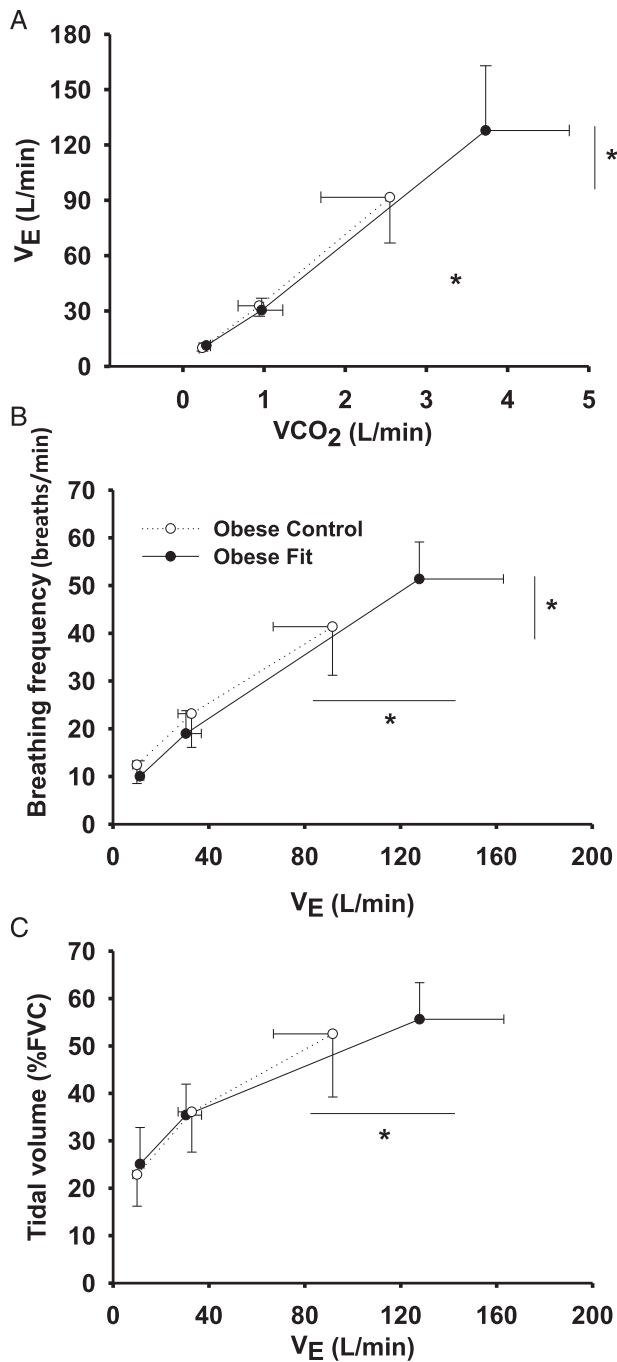


FIGURE 2. A-C, Ventilatory response and breathing pattern (B, breathing frequency; C, tidal volume) during the peak exercise test (rest, submaximal exercise, and peak exercise). $\dot{V}CO_2$ = CO_2 uptake; \dot{V}_E = minute ventilation.

were about 130% of their predicted values. Moreover, $\dot{V}O_{2peak}$ relative to their predicted body weight was about 50 mL/kg/min, which is above the 90th percentile according to the American College of Sports Medicine.²⁶ Moreover, the data from the submaximal exercise test further confirm that the FO group indeed had higher CRF than did the CO group. This was reflected by the 28% lower relative $\dot{V}O_2$, 12% lower

respiratory exchange ratio, 18% drop in heart rate, reduction in blood lactate levels by more than one-half, and 19% reduction in the ventilatory response to exercise, despite working at a higher absolute work rate. Collectively, these observations strongly suggest that the individuals in the FO group had higher fitness levels than those in the CO group. In fact, the increased CRF in this FO group was comparable to, if not higher than, values reported after exercise training studies in healthy obese individuals.²⁷⁻³¹ Finally, several studies on normal-weight and fit individuals have reported fitness levels comparable to this FO group.^{8,10,32,33}

Mechanical Ventilatory Constraints

We did not observe increased EFL in this FO group at peak exercise even though the FO group maximal \dot{V}_E was approximately 40% higher than in the CO group. This degree of flow limitation (<20% of \dot{V}_T) is considered to be a mild constraint according to Johnson et al.³⁴ The proportion of subjects who developed EFL in the current investigation is very similar to that in prior studies in sedentary obese individuals.^{5-7,35,36} For instance, Ofir et al⁷ reported that approximately 55% of sedentary obese subjects who underwent an incremental exercise test experienced a moderate degree of flow limitation near peak exercise (about 38% of \dot{V}_T). Babb et al⁵ reported that about 45% of sedentary obese women experienced mild EFL (about 12% of \dot{V}_T) at peak exercise. Likewise, DeLorey et al⁶ reported mild EFL at peak exercise in six of 10 obese sedentary adults (about 12% of \dot{V}_T). However, these comparisons should be done with care because of differences in the methodologies, the degrees of exertion, and the protocols used. The FO subjects had reduced EELV at rest. EELV also decreased during the early stages of exercise but returned to resting levels at peak exercise.⁵⁻⁷ This dynamic hyperinflation is also an index of ventilatory constraint³⁴ and allows subjects to further increase expiratory flow rates and minimize EFL.^{8,37} At peak exercise, end-inspiratory lung volume approached 90% of TLC and \dot{V}_E was approximately 79% of MVV, which may also indicate slight mechanical ventilatory constraint.³⁸ Despite some degree of mechanical ventilatory constraint observed, neither end-tidal CO_2 nor oxygen saturation revealed relative hypoventilation. Moreover, they do not appear to have exacerbated mechanical ventilatory constraints that could considerably alter breathing mechanics and ventilatory dynamics during heavy exercise. Part of this lack of increase in mechanical ventilatory constraints in the FO group despite the large increase in ventilatory demand at peak efforts could be due to the overall greater lung function in the fit group compared with the control group. The increase in TLC,

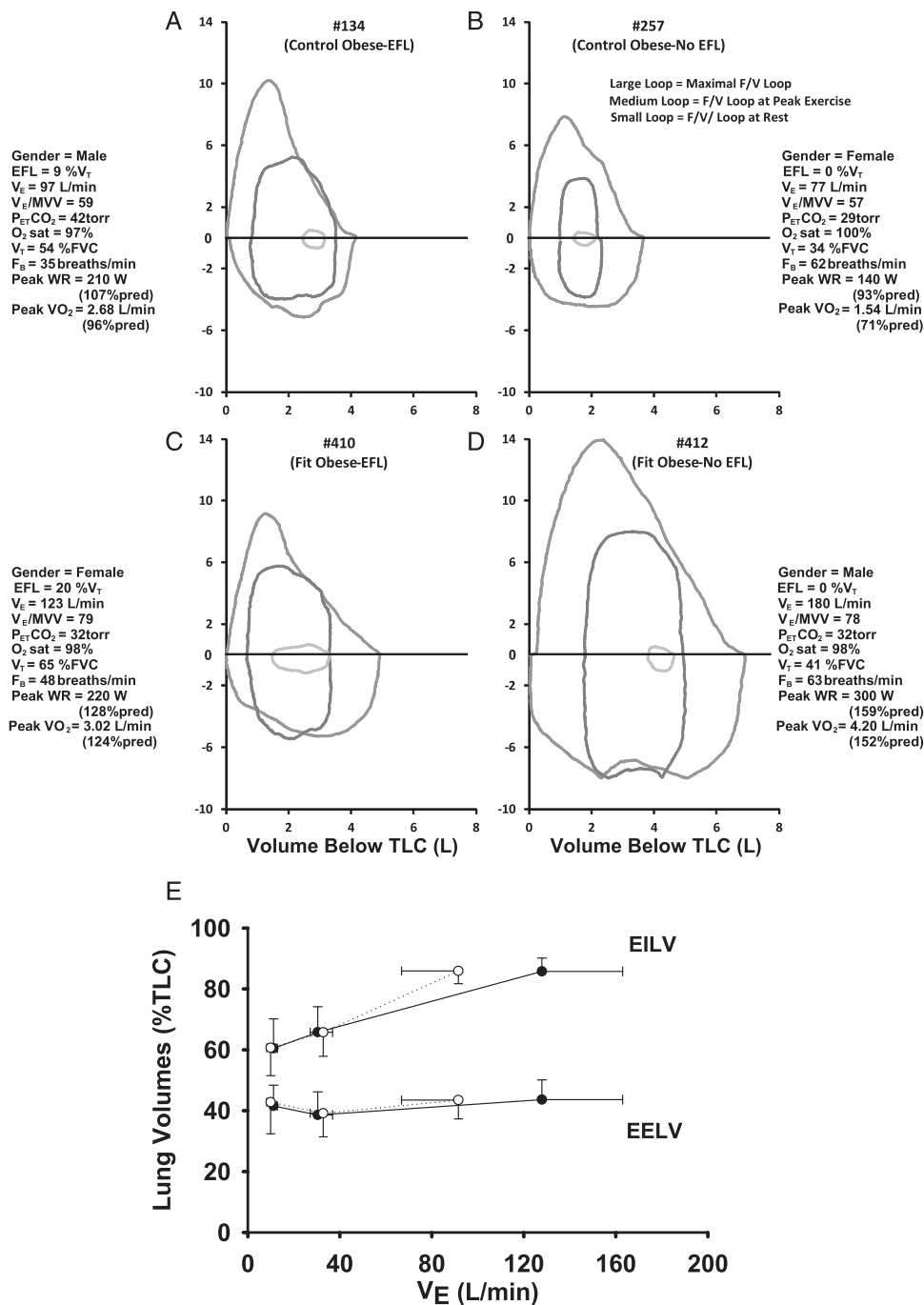


FIGURE 3. Sample flow volume loops from individuals at peak exercise. A, Control obese (CO) subjects with EFL. B, CO subjects without EFL. C, Fit obese (FO) subjects with EFL. D, FO subjects without EFL. EFL was observed in four (three women and one man) FO and five (three women and two men) CO subjects; however, the degree of flow limitation was mild (EFL < 25% V_T) and was not different between the FO and CO groups (13% ± 6% V_T vs 16% ± 7% V_T, respectively). E, Dynamic lung volumes (end-inspiratory lung volume and end-expiratory lung volume) during the peak exercise test (rest, sub-maximal exercise, and peak exercise). EELV = end-expiratory lung volume; EFL = expiratory flow limitation; EILV = end-inspiratory lung volume; F_B = breathing frequency; F/V = flow volume; O₂sat = oxygen saturation; P_{ET}CO₂ = end tidal CO₂; VO₂ = oxygen uptake; V_T = tidal volume; WR = work rate. See Figure 1 and 2 legends for expansion of other abbreviations.

FEV₁, PEF, and MVV may have masked some of the expected ventilatory constraints associated with a 40% increase in ventilatory demand. Furthermore, our

findings also show that our FO group did not have increased mechanical ventilatory constraints compared with normal-weight subjects of similar CRF.^{32,39}

Breathing Strategy

Increasing inspiratory and expiratory flows can shorten the breathing cycle for a given V_T and can, thus, further increase their \dot{V}_E by increasing breathing frequency. In fact, the FO group was able to achieve higher \dot{V}_E at maximal efforts by further increasing breathing frequency. Finally, others have also reported similar breathing frequency at peak exercise in non-obese adults with CRF similar to that of the FO group.^{10,40}

From a practical point of view of minimizing the work of breathing, increasing \dot{V}_E by increasing breathing frequency near peak exercise is a good strategy, despite the inevitable increase in the dead space ventilation. There are strong data to suggest that over the ventilatory range from 30 to 130 L/min, increasing ventilation by changing breathing frequency has little impact on the work of breathing.^{41,42} Subjects were able to maintain a normal V_T , limit EFL, and limit their encroachment on TLC. All these changes allowed the FO group to increase \dot{V}_E by increasing breathing frequency, which kept the increase in the work of breathing to a minimum.

Pulmonary Function

Data from the pulmonary function tests revealed that the “obesity effect” was present in the FO group, just as it was in the CO group. There are conflicting reports on the effects of endurance training on pulmonary function and lung volumes.⁴³⁻⁴⁵ Some reports suggest pulmonary volumes in endurance-trained adults are greater than in sedentary control subjects,^{46,47} whereas other investigators have found no consistent differences in lung volumes with endurance training.^{43,44} Our results suggest improved overall lung function with increased CRF and agree with some reports,^{44,46,47} but not with others.^{32,48} The overall consensus, however, is that increased CRF may have a stronger influence in “effort-dependent” lung function variables such as PEF, MVV, and FEV₁,^{21,49} while having less of an effect on “effort-independent” variables such as FVC and TLC.^{27,43,44,47} From our data, we cannot determine if the improved lung function in the FO group was a result of their training, or if they were able to achieve such a high CRF because of their already enhanced lung function. Further interventional research studies to answer this very important question are warranted.

Oxygen Cost of Breathing

Obesity reduces chest wall and total respiratory system compliance, which increases the work of breathing.^{1,4} Compared with our data on normal-weight subjects,⁴⁸ both the FO and CO subjects had an increase in the work of breathing. Our data from the FO group sug-

gest that their oxygen cost of breathing is about 40% higher than those reported in nonobese individuals.⁴⁸ The data from the FO group was somewhat lower compared with the CO group, but failed to reach significance ($P = .08$). Regardless, the FO subjects were able to increase \dot{V}_E at peak exercise by 40% without a significant tax on the work of breathing.

CONCLUSIONS

These novel data suggest that young, otherwise healthy, obese adults can participate in vigorous physical activity without being ventilatory compromised, even at peak efforts. Therefore, these findings are good news for those who carry extra weight but want to participate in physical activity, because many reports suggest that increased fitness is associated with lower risk of mortality regardless of the degree of obesity.⁴⁹⁻⁵¹ Although the results of the current investigation are encouraging for healthy individuals with mild-to-moderate obesity, more research is warranted to investigate these responses in more extreme stages of obesity.

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Dr Lorenzo: contributed to the data collection, data processing and analysis, critical input, and the writing of the manuscript.

Dr Babb: contributed to the planning of the project, supervising and assisting in data collection, directing data processing and analysis, and the writing of the manuscript.

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