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Does exercise test modality influence dyspnoea perception in obese patients with COPD?

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Study's Main Message

Reliable evaluations of exertional dyspnea can be obtained either by cycle or treadmill exercise tests in obese patients with COPD.

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ABSTRACT

The purpose of this study was to investigate whether differences in physiological responses to weight-bearing (walking) and weight-supported (cycle) exercise influence dyspnoea perception in obese COPD patients where such discrepancies are likely exaggerated.

We compared metabolic, ventilatory and perceptual responses during incremental treadmill and cycle exercise using a matched linearized rise in work rate in 18 (10 men, 8 women) obese (body mass index 36.4±5.0 kg/m^2; mean±SD) patients with COPD (FEV_1 60±11 % predicted).

Compared with cycle testing, treadmill testing was associated with a significantly higher oxygen uptake, lower ventilatory equivalent for oxygen, and greater oxyhemoglobin desaturation at a given work rate ($p<0.01$). Cycle testing was associated with a higher respiratory exchange ratio ($p<0.01$), earlier ventilatory threshold ($p<0.01$) and greater peak leg discomfort ratings ($p=0.01$). Ventilation, breathing pattern and operating lung volumes were similar between tests, as were dyspnoea/work rate and dyspnoea/ventilation relations.

Despite significant between-test differences in physiological responses, ventilation, operating lung volumes and dyspnoea intensity were similar at any given external power output during incremental walking and cycling exercise in obese COPD patients. These data provide evidence that either exercise modality can be selected for reliable evaluation of exertional dyspnea in this population in research and clinical settings.

INTRODUCTION

The prevalence of both obesity and COPD is increasing steadily throughout the world [1]. The combination of these common conditions is associated with increased activity restriction and health care burden [2]. The effective management of exercise intolerance in obese COPD patients remains a major challenge and awaits a better understanding of the underlying mechanisms.

It is widely believed that obese COPD patients experience greater dyspnoea than normal weight COPD during daily activities. However, while field tests indicate reduced walking distance in obese versus normal weight COPD patients [3,4], physiological studies using cycle ergometry have found that dyspnoea intensity, endurance time and peak oxygen uptake ($V'O_2$) are similar in the two groups when severity of airway obstruction is matched [5,6]. The question therefore arises: does measurement of perceived dyspnoea intensity and exercise performance in obese COPD during cycle ergometry (where leg muscles are selectively stressed [7,8]), accurately reflect the situation during daily activities such as walking? In this regard, no studies have compared the relationship between dyspnoea and physiological responses during carefully matched walking and cycling exercise protocols in obese patients with COPD.

It has previously been demonstrated that the dynamic respiratory mechanical derangements, and the associated dyspnoea, during exercise in COPD rise in direct proportion to the prevailing ventilatory requirement of the task [9]. It follows that differences in dyspnoea intensity between cycling and walking could occur at a given power output if the metabolic and ventilatory responses to these tasks are different. In the obese COPD patient, the relative importance of the higher metabolic cost of external work during treadmill exercise or the earlier metabolic acidosis of cycle exercise in driving ventilation (and consequent dyspnoea intensity) is

difficult to predict [10-12]. It is also conceivable that differences in dynamic respiratory mechanics or in the source of the ventilatory stimulation (i.e. metabolic loading, skeletal muscle recruitment, arterial oxygen saturation and hemodynamic responses) between the two exercise modalities could influence the dyspnoea/ventilation relation.

Accordingly, the main objective of this study was to better understand the influence of the physiological peculiarities of each exercise modality on dyspnoea perception in patients with combined obesity and COPD. We compared physiological responses, dyspnoea/work rate and dyspnoea/ventilation relations during symptom-limited incremental cycle and treadmill exercise tests, using matched linearized work rate protocols.

METHODS

Subjects

Eighteen clinically stable obese (body mass index (BMI) >30 kg/m^2) COPD (FEV_1/FVC <70%) patients between the ages of 40-80 with a post-bronchodilator FEV_1 <80% predicted were recruited. Patients were excluded if they had significant disease which affected breathlessness or exercise capacity (i.e. metabolic, cardiovascular, neuromuscular and/or musculoskeletal), received daytime oxygen therapy, had significant respiratory disease other than COPD, were too breathless to leave the house or too fit (Medical Research Council (MRC) dyspnoea scale 5 or 1, respectively) and had any contraindications to clinical exercise testing.

Study design

This cross-sectional study received University and Hospital Research Ethics Board approval (DMED-1187-09). After obtaining informed consent, subjects attended 3 visits each separated by at least 48 hours. Visit 1, all subjects underwent medical screening, detailed pulmonary function tests, and familiarization with all exercise testing procedures. Visits 2 and 3 included pulmonary function tests and either a cycle or treadmill test (randomized visit order). Before each visit, subjects withheld short-acting β_2-agonist and anticholinergic bronchodilators for at least 4 and 6 hours, respectively, and long-acting bronchodilators at least 12 hours. Subjects were instructed to avoid caffeine, heavy meals, alcohol, and major physical exertion prior to each visit. Subjects underwent a dual-energy X-ray absorptiometry (DEXA) scan within the study period to quantify body composition. When available, prior clinical chest computed tomography (CT) scan results were used to provide qualitative assessments of emphysema.

Procedures

Pulmonary function testing included routine spirometry, body plethysmography, single-breath diffusing capacity (D_LCO) and maximum voluntary ventilation (MVV) using automated testing equipment (Vs62j body plethysmograph with Vmax229d; SensorMedics, Yorba Linda, CA). Cardiopulmonary exercise tests were performed on an electronically braked cycle ergometer (Ergometrics 800S; SensorMedics, Yorba Linda, CA) and on a treadmill (Medtrack ST55; Quinton Instrument, Bothell, WA) using a Vmax229d Cardiopulmonary Exercise Testing System (SensorMedics). Cycle and treadmill exercise tests were both performed with 10-watt increments which increased every two minutes to a symptom-limited endpoint. The incremental treadmill protocol was individualized based on body weight: there was a linear increase in speed and a curvilinear rise in grade (***see online supplement for more detail***). Subjects rated "breathing discomfort" and "leg discomfort" on a modified 10-point Borg scale [13]. Inspiratory capacity (IC) maneuvers were performed during a steady-state resting period, during each stage of exercise and at peak exercise. Operating lung volumes were derived from IC measurements as previously described [14]. Immediately after exercise, subjects were asked why they stopped exercising. Breath-by-breath data from the last 30-s of loaded pedaling were averaged for each individual and analysed as "peak" exercise. Three independent observers, who were blinded to exercise modality, identified the ventilatory threshold (VTh) by the "V-slope" plot which was then double-checked with the inflection points suggested by the ventilatory equivalent and end-tidal pressure methods [15].

Statistical analysis

A sample size of 18 provided 80% power to detect a minimal clinically important difference in dyspnoea of ±1 Borg scale unit measured at a standardized work rate [16], assuming an α of 0.05

and a within-patient standard deviation of 1 unit. Comparisons of exercise modalities were made at rest, at standardized work rates (i.e., 10, 20, 30 and 40 watts) and at peak exercise using paired two-tailed t-tests. Results are reported as means ± SD.

RESULTS

Subjects

Subject characteristics and DEXA scan results are presented in Tables 1 and 2, respectively. Subjects had moderate airflow obstruction and lung volumes were within the predicted normal ranges with the exception of residual volume (RV) (124% predicted) and expiratory reserve volume (ERV) (66% predicted). Evidence of emphysema was shown on all available chest CT scans (13/18 subjects). DEXA scans from 15 subjects (equipment was unavailable for the last 3 subjects) revealed that total body mass was elevated by 15.5 kg (17.5%) in males and 16.1 kg (21.7%) in females compared to the population means for age-matched individuals [17]: this resulted from a greater total fat mass (males 11.0, females 7.1 kg) and lean mass (males 4.5, females 8.8 kg) compared to the population means.

Responses to cycle and treadmill exercise

Subjects reached a similar peak work rate during cycle and exercise tests (Table 3). Although exercise duration was longer by a mean difference of 52 seconds in the treadmill versus cycle test, this was not statistically significant ($p=0.11$). The distribution of main reasons for stopping exercise was also not different between tests ($p=0.56$): breathing discomfort (treadmill n=10; cycle 8), leg discomfort (treadmill 2; cycle 5) and a combination of breathing and leg discomfort (treadmill 6; cycle 5). Rest and peak exercise data are presented in Table 3.

Metabolic and gas exchange responses. Peak $V'O_2$ expressed as a percentage of predicted was higher during treadmill compared to cycle (91 ± 30 versus 78 ± 26 % predicted, respectively, $p<0.05$) using the recommended gender-specific predictive equations for obese men [18] and obese women [19] described by Lorenzo et al. [20]. There was a significant upward displacement of $V'O_2$ relative to work rate during treadmill compared with cycle exercise; the

$V'O_2$-work rate slope was 14.2±2.1 and 12.5±3.7 mL/min/watt during treadmill and cycle exercise, respectively (p=0.07) (Figure 1). Carbon dioxide production ($V'CO_2$) was higher at 30 and 40 watts and at the peak of treadmill exercise compared to cycling (Figure 1). The respiratory exchange ratio (RER) during cycle compared to treadmill tests showed an upward shift from the onset of exercise that was maintained through to peak exercise (Figure 1). Sixteen subjects had an identifiable VTh. Subjects reached an earlier VTh during cycle exercise at a $V'O_2$ of 1.01±0.23 L/min compared to 1.17±0.27 L/min during treadmill exercise ($p<0.05$). VTh relative to predicted peak $V'O_2$ was similar between modalities and above the lower limit of normal (cycle: 53%, treadmill: 58%) [18]. Subjects experienced greater arterial oxygen desaturation during treadmill compared to cycle exercise, with peak decreases of -5.2±3.2 % and -3.2±2.2 %, respectively (p=0.01) (Figure 1).

Cardiac responses. Heart rate (HR) rose in a similar fashion between modalities throughout submaximal exercise but reached approximately 9 beats/min higher ($p<0.05$) at peak treadmill compared to peak cycle exercise (Table 3).

Ventilatory responses. Ventilation (V'_E) was not different between test modalities at any given work rate (Figure 1). $V'_E/V'O_2$ was greater at all stages of cycle compared with treadmill exercise (Figure 1) but $V'_E/V'CO_2$ was only greater at 40 watts and at the peak of cycle exercise (Table 3) and $V'_E/V'CO_2$ slopes were similar (Figure 2). Breathing pattern and operating lung volumes were not statistically different at a given work rate or V'_E (Figure 2) between modalities throughout exercise. Subjects reached critical respiratory-mechanical reserves at an EILV >90 %TLC and an inspiratory reserve volume (IRV) of approximately 0.5L during both exercise modalities at peak exercise (Table 3).

Perceptual responses. Dyspnoea intensity ratings at standardized work rates and at peak exercise were similar between modalities (Figure 3). Leg discomfort was also similar during exercise up to 40 watts but became significantly ($p<0.05$) greater at the peak of cycle compared with treadmill exercise (Figure 3). Relationships between dyspnea intensity and both V'_E and IRV were not different during cycle and treadmill exercise (Figure 3).

DISCUSSION

The main findings of the study are as follows: 1) compared with cycle testing, treadmill testing was associated with a higher $V'O_2$, lower $V'_E/V'O_2$, and greater oxyhemoglobin desaturation for a given work rate; 2) cycle testing was associated with a higher RER, earlier VTh and greater peak leg discomfort ratings; 3) ventilation, breathing pattern, operating lung volumes and dyspnoea intensity were similar at comparative work rates; and 4) despite physiological differences, exercise modality had no effect on the dyspnoea/work rate or dyspnoea/V'_E relations in obese patients with COPD.

Participants had mild to severe obesity with body weights in excess of ideal body weight by an average of 32.8 kg. DEXA scans confirmed increased adipose mass deposition compared to the average for age-matched individuals. The largely preserved IC and FRC likely reflects the known effects of increasing BMI on lung volume components in COPD [3,21]. Participants had moderate airway obstruction but surprisingly, average D_LCO was diminished to 50% predicted (possibly reflecting underlying emphysema) in the absence of significant lung hyperinflation. Patients reported moderate chronic activity-related dyspnoea but had reasonably preserved cardiorespiratory fitness as assessed by peak $V'O_2$ relative to the predicted value based on ideal body weight.

The incremental exercise protocols were well matched for rate of increase in work rate and were of sufficient duration for accurate assessment of perceptual and physiological responses. Peak $V'O_2$ was significantly higher (by 17%) during treadmill compared with cycle exercise (Figure 1), likely reflecting the larger skeletal muscle mass recruitment previously described in health [22]. Of note, patients had significant mechanical constraints on ventilation as evidenced by a low breathing reserve (high V'_E/MVV) and a high EILV/TLC ratio at

relatively low peak work rates of 73 and 78 watts during cycle and treadmill, respectively. In contrast, participants had adequate cardiac reserve at peak exercise, reaching a mean heart rate of 72 and 77 % of the predicted maximum during cycle and treadmill tests, respectively.

The dyspnoea/work rate relation

A key finding of the present study was the similarity in dyspnoea/work rate relations during both exercise modalities. This indicates that differences in the metabolic cost of external work, $V'_E/V'O_2$, RER, VTh, oxyhemoglobin saturation, perceived leg discomfort and cardio-circulatory responses did not influence dyspnoea perception at a given power output during cycling and walking in obese COPD. The preserved dyspnoea/work rate relation ultimately reflected the between-test similarity in the V'_E, breathing pattern and operating lung volumes when the increase in work rate was carefully matched. It is remarkable that despite the differences in potential sources of ventilatory drive, V'_E remained tightly coupled with "pulmonary" $V'CO_2$ regardless of the exercise modality. These findings are in line with those from on- and off-exercise kinetics studies where the time course of V'_E has been found to closely follow the $V'CO_2$, and not VO_2 or arterial partial pressure of oxygen [23].

The dyspnoea/ventilation relation

Our results show that the effective coupling of ventilation to the metabolic demand of comparable external work – expressed as $V'CO_2$ - is achieved by different mechanisms in cycling and treadmill tests. A higher RER ($V'CO_2/V'O_2$) during cycling versus walking (or running) has been previously described in healthy individuals and may reflect preferential carbohydrate utilization with cycling [24]. We have previously shown that metabolic pathways in leg muscle are altered in COPD such that there is increased dependence on glycolysis and

blood glucose utilization (rather than free fatty acid) compared with healthy individuals [25]. To what extent the higher RER in our obese COPD patients during cycling can be explained by preferential utilization of carbohydrate by the contracting leg muscles could not be determined in the current study. Regardless the mechanism(s), these muscle metabolic differences between cycling and walking are likely to have contributed to higher $V'CO_2$ (and V'_E) for a given $V'O_2$ in the former modality.

The smaller locomotor muscle mass required to generate the same power output during cycling as walking means that the average metabolic rate per unit of contracting muscle mass is greater with cycling, which can force earlier metabolic acidosis [26]. Our findings of an earlier VTh, increased $V'_E/V'O_2$, preserved oxyhemoglobin saturation (at a given $V'O_2$) and reduced peak $P_{ET}CO_2$ during cycling are consistent with the results of recent studies which additionally show relatively increased alveolar ventilation during cycling [10]. It remains possible (but unproven) that differential ventilatory stimulation by mechanoreceptor/metaboreceptor activation (Type III and IV afferents) between active locomotor muscle groups (peculiar to the exercise test modality) may explain differences in $V'_E/V'O_2$ [27].

Despite the above-outlined differences in the sources of ventilatory stimulation during the two exercise modalities, perceived dyspnoea intensity was similar at comparable V'_E in obese COPD. Based on previous studies, the dyspnoea/V'_E relation during exercise in COPD is altered by change in dynamic respiratory mechanics but not by experimental manipulation of the central respiratory controller *per se* [28,29]. Thus, the preservation of the dyspnoea/V'_E relation in the current study mainly reflects the similarities in operating lung volumes at a given V'_E during cycling and walking, regardless of differences in metabolic loading and acid-base balance.

Limitations

Since dynamic respiratory mechanics and the extent of metabolic loading are different in normal weight COPD patients than in obese patients matched for airflow obstruction [3,30], the lack of effect of exercise modality on dyspnoea intensity seen in this study cannot be generalized to non-obese patients. The present study was performed in patients with moderate COPD and our results may not be generalized to more severe patients. However, we believe that given the close link of work to ventilation, dyspnea/work rate and dyspnea/ventilation relationships are likely to be similar across disease severity - if the work performed on different test modalities is closely matched, as in the current study. Measurements of blood lactate, arterial and mixed venous blood gases would be required to better elucidate the observed between-test differences in RER and oxyhemoglobin saturation but this was not the main focus of the current study.

Conclusions/implications

Despite consistent task-specific differences in physiological derangements that can influence the central respiratory controller, perception of dyspnoea intensity was most closely linked to CO_2 output and the attendant ventilatory and dynamic respiratory mechanical response. Dyspnoea/work rate and dyspnoea/V'_E relations were independent of exercise modality in obese COPD. Our results provide reassurance that either exercise modality can be selected for the reliable evaluation of dyspnoea in obese COPD patients, both in research and clinical settings. In obese COPD patients, the higher peak $V'O_2$ and lower oxyhemoglobin saturation during treadmill exercise (compared with cycle) have potentially important implications for the individualized evaluation of cardiorespiratory fitness and pulmonary gas exchange abnormalities, respectively. Another important clinical implication of the results is that interventions that reduce

CO_2 output (exercise training, energy substrate manipulation) during physical activity in obese COPD patients should effectively relieve exertional dyspnoea.

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TABLE 1. Subject characteristics

Age, years	66 ± 8
Male : Female, n	10 : 8
Height, cm	167.2 ± 2.0
Weight, kg	102.1 ± 20.1
Ideal body weight, kg	69.3 ± 2.0
Body mass index, kg/m^2	36.4 ± 5.0
Waist circumference, cm:	
Male	129 ± 10
Female	115 ± 7
Smoking history, pack-years	50 ± 32
COPD duration, years	8.8 ± 4.4
Baseline Dyspnoea Index, 0-12	6.6 ± 1.8
MRC dyspnoea scale, 0-5	2.4 ± 0.7
Pulmonary function:	
FEV_1, L (% predicted)	1.40 ± 0.40 (60 ± 11)
FVC, L (% predicted)	3.08 ± 0.82 (88 ± 14)
FEV_1/FVC, %	48 ± 9
IC, L (% predicted)	2.41 ± 0.76 (90 ± 23)
FRC, L	3.31 ± 0.71 (105 ± 19)
RV, L	2.67 ± 0.68 (124 ± 33)
TLC, L	5.73 ± 1.07 (98 ± 13)
RV/TLC, %	47 ± 8
sR_{AW}, % predicted	385 ± 151
D_LCO, mL/mmHg/min (% predicted)	12.2 ± 4.4 (50 ± 12)
D_LCO/V_A, % predicted	76 ± 16

Data are presented as means ± SD.
MRC: Medical Research Council; FEV_1: forced expired volume in 1 second; FVC: forced vital capacity; IC: inspiratory capacity; FRC: functional residual capacity; RV: residual volume; TLC: total lung capacity; sR_{AW}: specific airway resistance; D_LCO: diffusing capacity of the lung for carbon monoxide; V_A: alveolar volume.

TABLE 2. DEXA scan results

	Obese COPD	Population Averages *
Male and Female (n=15)		
Total body:		
Mass, kg	97.7 ± 12.2	
Lean mass, kg	56.0 ± 8.1	
Fat mass, % total body mass	40.2 ± 4.9	
Trunk:		
Mass, kg	51.3 ± 8.1	
Fat, % total trunk mass	42.4 ± 5.1	
Both legs:		
Mass, kg	29.9 ± 3.6	
Lean mass, kg	17.4 ± 2.5	
Fat mass, kg	11.5 ± 2.9	
Fat, % total leg mass	38.5 ± 6.9	
Male (n=8)		
Total body:		
Mass, kg	104.2 ± 11.0	88.7
Lean mass, kg	62.3 ± 5.4	57.8
Fat mass, % total body mass	37.5 ± 4.4	31.2
Trunk:		
Mass, kg	55.8 ± 7.6	45.8
Fat mass, % total trunk mass	41.1 ± 5.8	33.1
Both legs:		
Mass, kg	30.3 ± 3.3	27.0
Lean mass, kg	19.1 ± 2.9	17.7
Fat mass, kg	10.2 ± 1.9	8.1
Fat, % total leg mass	33.5 ± 4.1	29.7
Female (n=7)		
Total body:		
Mass, kg	90.2 ± 9.1	74.1
Lean mass, kg	48.9 ± 3.1	40.1
Fat mass, % total body mass	43.2 ± 4.0	42.5
Trunk:		
Mass, kg	46.2 ± 5.3	37.1
Fat mass, % total trunk mass	43.5 ± 4.6	41.2
Both legs:		
Mass, kg	29.3 ± 4.0	24.5
Lean mass, kg	15.4 ± 1.2	12.3
Fat mass, kg	13.1 ± 3.3	11.3
Fat, % total leg mass	44.3 ± 4.6	45.5

Values are means ± SD.

*Population averages are means for non-hispanic white people between the ages of 60 and 79 [17].

TABLE 3. Measurements during symptom-limited incremental cycle and treadmill exercise

	Rest		Peak	
	Cycle	**Treadmill**	**Cycle**	**Treadmill**
Work rate, watts	0 ± 0	0 ± 0	73 ± 20	78 ± 28
Exercise time, mm:ss	0 ± 0	0 ± 0	14:13 ± 01:15	15:05 ± 01:06
Dyspnoea, Borg scale	0.1 ± 0.2	0.1 ± 0.2	6.6 ± 2.8	6.6 ± 2.4
Leg discomfort, Borg scale	0.1 ± 0.3	0.2 ± 0.3	6.6 ± 2.9	5.0 ± 2.7 ¶
$V'O_2$, L/min	0.41 ± .14	0.42 ± 0.14	1.53 ± 0.40	1.79 ± 0.52
$V'O_2$, % predicted maximum	20.4 ± 6.7	20.5 ± 4.9	78.1 ± 25.7	90.7 ± 30.3 ¶
$V'CO_2$, L/min	0.35 ± .12	0.34 ± 0.12	1.49 ± 0.39	1.65 ± 0.51 ¶
RER	0.84 ± .06	0.82 ± .09	0.98 ± .07	0.92 ± .10 ¶
V'_E, L/min	14.8 ± 4.2	15.1 ± 4.2	47 ± 10.8	49 ± 13.7
V_T, L	0.78 ± 0.21	0.85 ± 0.35	1.30 ± 0.34	1.34 ± 0.41
F_b, breaths/min	20 ± 5.3	20 ± 6.0	36 ± 6.1	37 ± 7.1
T_I/T_{TOT}	0.38 ± 0.05	0.38 ± 0.04	0.40 ± 0.04	0.41 ± 0.04
IC, L	2.50 ± 0.75	2.44 ± 0.71	1.84 ± 0.37	1.79 ± 0.47
V_T/IC, %	32.5 ± 9.9	34.6 ± 8.5	72.3 ± 11.1	74.6 ± 9.2
IRV, L	1.73 ± .69	1.59 ± .48	0.51 ± .22	0.45 ± .18
EELV, L	3.31 ± 0.79	3.4 ± 0.79	3.97 ± 0.92	4.07 ± 0.94
EILV, L	4.09 ± 0.82	4.30 ± 0.89	5.31 ± 1.12	5.42 ± 1.13 *
EILV/TLC, %	70.7 ± 7.9	72.8 ± 6.1	91.1 ± 3.8	92.1 ± 3.5 *
V'_E/MVV, %	29 ± 1	29 ± 1	90 ± 2	92 ± 2
$V'_E/V'O_2$	36.8 ± 5.0	37.1 ± 7.7	31.7 ± 6.1	28.0 ± 5.9 ¶
$V'_E/V'CO_2$	43.9 ± 6.8	45.3 ± 7.7	32.4 ± 5.0	30.4 ± 4.8 ¶
$P_{ET}CO_2$, mmHg	34 ± 2.8	33 ± 3.8	37 ± 5.5	39 ± 6.0 ¶
HR, beats/min	77 ± 2.7	79 ± 10	119 ± 12.8	128 ± 14.6 ¶
SpO_2, %	95 ± 1.8	95 ± 2	92 ± 2.7	90 ± 3.3 ¶

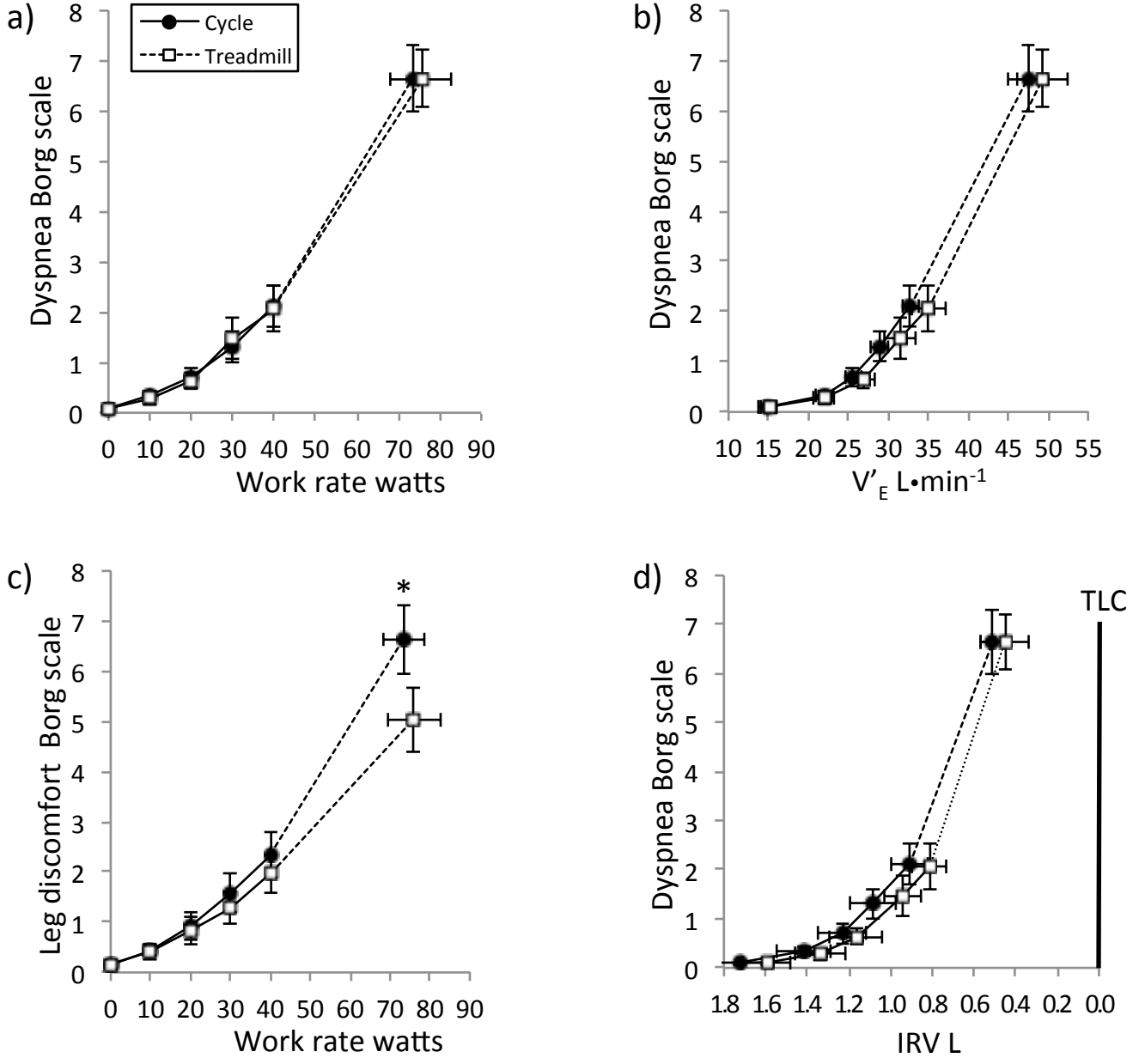
Values are means ± SD.
$V'O_2$: oxygen uptake; $V'CO_2$: carbon dioxide production; RER: respiratory exchange ratio; V'_E: ventilation; V_T: tidal volume; F_b: breathing frequency; T_I/T_{TOT}: inspiratory duty cycle; IC: inspiratory capacity; IRV: inspiratory reserve volume; EELV: end-expiratory lung volume; EILV: end-inspiratory lung volume; $V'_E/V'O_2$: ventilatory equivalent for oxygen uptake; $V'_E/V'CO_2$: ventilatory equivalent for carbon dioxide production; $P_{ET}CO_2$: end-tidal partial pressure of carbon dioxide; HR: heart rate; SpO_2: arterial pulse oxygen saturation.
* $p<0.05$; # $p=0.05$; ¶ $p< 0.01$: cycle versus treadmill.

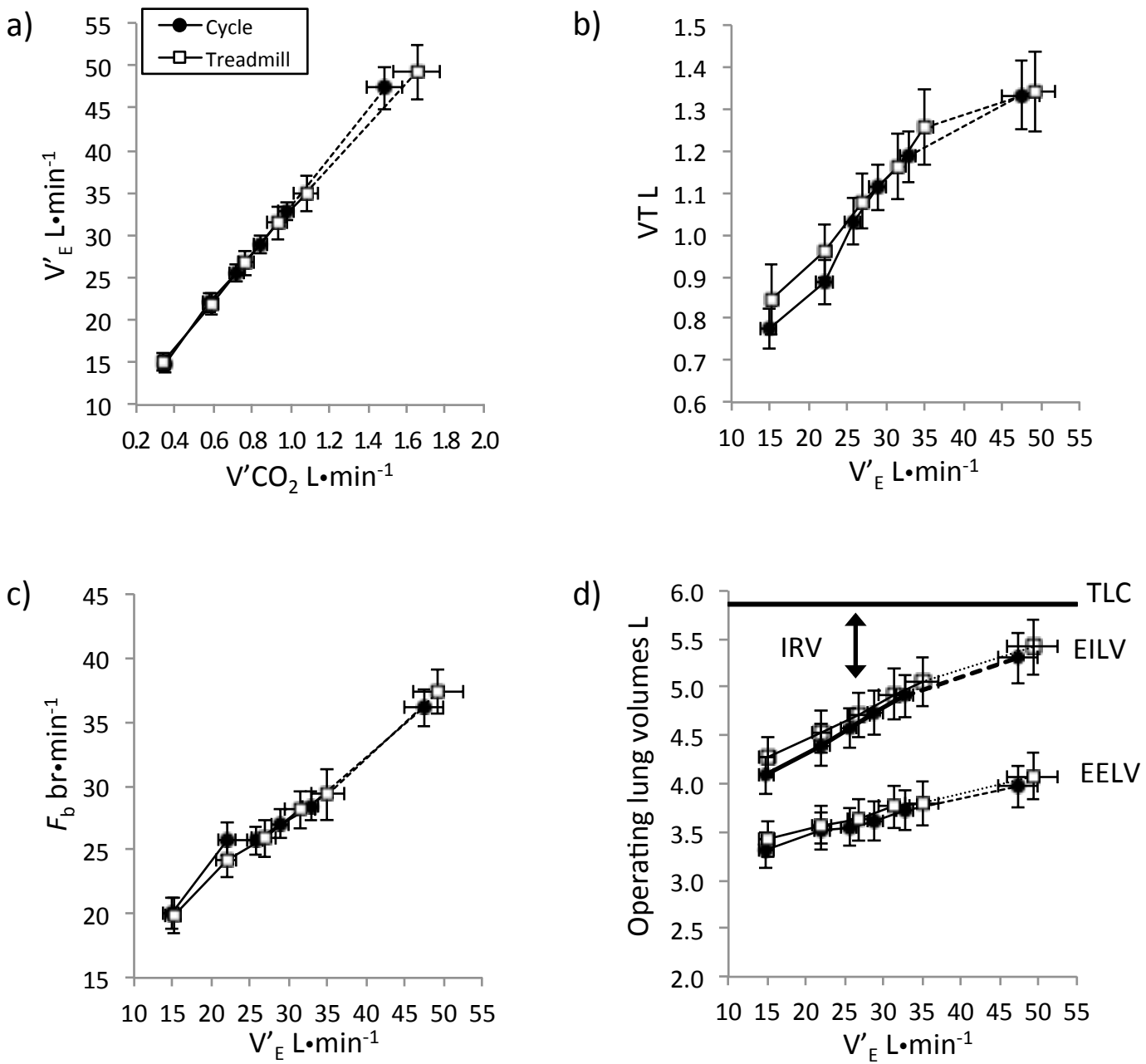
FIGURE LEGENDS

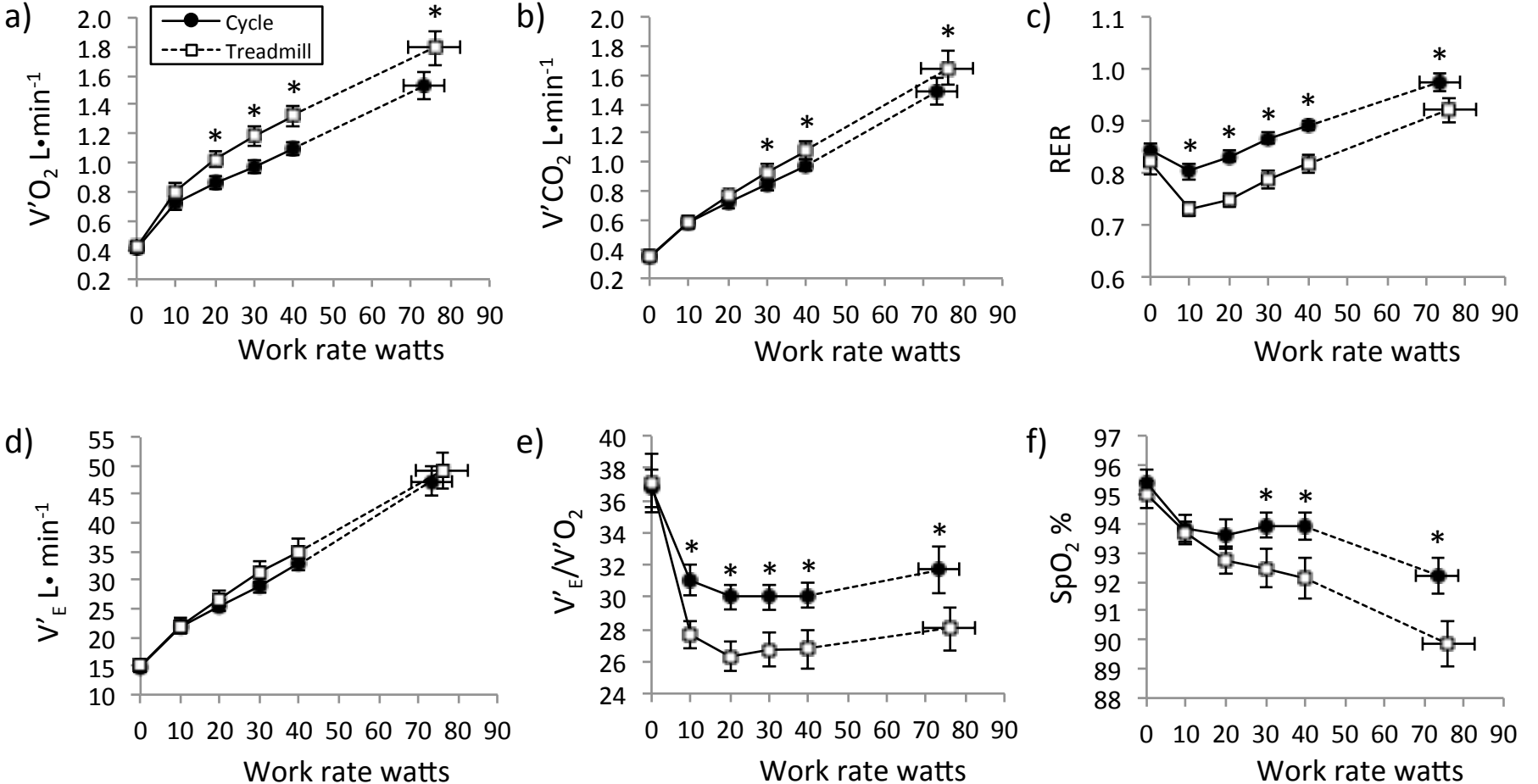
Figure 1. **(a)** Oxygen consumption ($V'O_2$), **(b)** carbon dioxide production ($V'CO_2$), **(c)** respiratory exchange ratio (RER), **(d)** minute ventilation (V'_E), **(e)** the ventilatory equivalent for oxygen consumption ($V'_E/V'O_2$), and **(f)** arterial oxygen saturation (SpO_2) are shown relative to work rate during cycle (closed circles) compared to treadmill (open squares). Values are means ± SEM. *$p<0.05$ cycle versus treadmill at a standardized work rate or at peak exercise.

Figure 2. **(a)** The relationship between minute ventilation (V'_E) and carbon dioxide production ($V'CO_2$) was similar during cycle (closed circles) compared to treadmill (open squares). **(b)** tidal volume (V_T), **(c)** breathing frequency (F_b) and **(d)** operating lung volumes expressed relative to V'_E were also similar during cycle and treadmill exercise. TLC: total lung capacity; EELV: end-expiratory lung volume; EILV: end-inspiratory lung volume; IRV: inspiratory reserve volume. Values are means ± SEM.

Figure 3. Relationships between dyspnea intensity and **(a)** work rate, **(b)** minute ventilation (V'_E) and **(d)** inspiratory reserve volume (IRV) were similar during cycle (closed circles) compared to treadmill (open squares) exercise. **(c)** Intensity of leg discomfort relative to work rate was similar up to 40W then became significantly greater by peak exercise during cycle compared with treadmill exercise (*$p<0.05$). Values are means ± SEM.


a)
Cycle
Treadmill
Dyspnea Borg scale
Work rate watts
b)
Dyspnea Borg scale
V'E L•min-1
c)
Leg discomfort Borg scale
Work rate watts
*
d)
Dyspnea Borg scale
TLC
IRV L


a)
Cycle
Treadmill
V'E L•min-1
V'CO2 L•min-1
b)
VT L
V'E L•min-1
c)
Fb br•min-1
V'E L•min-1
d)
Operating lung volumes L
TLC
IRV
EILV
EELV
V'E L•min-1


a)
Cycle
Treadmill
V'O2 L•min-1
Work rate watts
b)
V'CO2 L•min-1
Work rate watts
c)
RER
Work rate watts
d)
V'E L• min-1
Work rate watts
e)
V'E/V'O2
Work rate watts
f)
SpO2 %
Work rate watts