

Comparative Study of Respiratory Recovery Following Aerobic Versus Resistance Exercise in Healthy Adults

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Abstract

Background: Ventilation recovery following exercise is a composite of metabolic, cardiovascular, autonomic and respiratory recovery. Comparisons of early respiratory recovery patterns following aerobic and resistance exercise in healthy adults have been limited.

Method: This was a randomized crossover study in which 60 adults between 18 and 35 years of age performed 30 minutes of treadmill exercise at 70% heart-rate reserve and a resistance exercise that was matched in duration, comprising eight exercises for three sets at 70% of one-repetition maximum (1RM), separated by 72 hours. Respiratory rate, oxygen uptake, heart rate, peripheral oxygen saturation, and Borg dyspnea scores were obtained at rest, immediately post-exercise and after 15 minutes of seated recovery. The main outcome was the time to return to 10% of baseline respiratory rate.

Results: Aerobic exercise had a higher immediate respiratory rate than resistance exercise (31.8 ± 4.3 vs 28.6 ± 4.1 breaths/min; $p < 0.001$) but recovery was quicker. The mean difference in time to recovery for the respiratory rate after aerobic exercise versus resistance exercise was 2.8 minutes (95% CI 1.9–3.7; $p < 0.001$). After 15 minutes, oxygen uptake was still elevated following resistance exercise (6.3 ± 1.2 vs 5.4 ± 1.0 mL/kg/min; $p < 0.001$) but oxygen saturation was unchanged in both groups. One-minute heart-rate recovery was greater after aerobic exercise (29.4 ± 7.2 vs 22.1 ± 6.5 beats/min; $p < 0.001$).

Conclusions: Aerobic exercise resulted in a greater acute ventilatory response and quicker recovery of the respiratory and autonomic systems, while resistance exercise resulted in a higher post-exercise ventilatory response and faster recovery of oxygen consumption.

Keywords: Aerobic Exercise; Resistance Exercise; Respiratory Recovery; Oxygen Uptake; Heart-Rate Recovery; Healthy Adults.

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Introduction

Exercise causes an immediate rise in oxygen demand, carbon dioxide production, pulmonary ventilation and cardiovascular work. This response is dependent on the intensity, duration, active muscle mass and the proportion of oxidative and non-oxidative energy systems.

The maximal and submaximal oxygen transport are functions of the pulmonary, cardiovascular, hematological and skeletal-muscle systems, and the rate of oxygen-uptake adjustment offers information in addition to a single peak fitness value [1]. Transitions between rest, exercise, and recovery are thus often characterized by oxygen-

uptake kinetics [2]. Ventilation and oxygen consumption do not rapidly return to resting values after the end of exercise. The excess post-exercise oxygen consumption is a fast component that is linked to phosphocreatine replenishment, reoxygenation of blood and muscle, early autonomic adjustment, and a slower component linked to temperature, catecholamines, substrate cycling, lactate handling and tissue repair.

The intensity of the exercise is usually the most important factor determining this response, but the duration and mode of the exercise also affects the size and duration of this response [3]. As a result,

recovery data can include responses to physiological demands which are not reflected in peak exercise data. Cardiovascular and autonomic recovery are closely related to respiratory recovery. During the first minute after exercise, heart rate drops quickly, primarily due to the reactivation of the parasympathetic system and then the gradual withdrawal of the sympathetic system. A late HR decrease has proven to be clinically and prognostically relevant [4]. The first minutes of recovery are a sensitive period for assessing autonomic balance recovery, as shown by experimental studies of post-exercise parasympathetic reactivation [5]. During the same time interval, the respiratory rate, oxygen uptake, and perceived breathlessness give complementary information concerning metabolic and ventilatory normalization.

Aerobic exercise typically involves large muscle groups, a relatively constant oxygen demand and high minute ventilation during exercise. Resistance exercise is intermittent and causes repeated mechanical compression, Valsalva-like behavior, and increased use of anaerobic metabolism and localized accumulation of metabolites. Acute resistance training may have greater potential to reduce cardiac parasympathetic modulation than aerobic training, especially when training large muscle groups, low rest intervals and moderate to high loads are employed [6]. However, regular aerobic exercise can enhance autonomic modulation and recovery properties of healthy young people [7]. Direct comparisons are difficult due to the differences between aerobic and resistance protocols in duration, external work, muscle recruitment and energy expenditure. Some previous studies have compared the post-exercise oxygen consumption for continuous, interval, and resistance exercise, but most of these studies recruited small samples or selected subjects and focused on total energy expenditure rather than the clinically measurable recovery of respiratory function. Comparative exercise studies have demonstrated that the post-exercise oxygen-consumption profile is significantly different between aerobic and resistance exercise [8] but the duration needed for RR to return to baseline after a duration-matched session of aerobic and resistance exercise has not been well defined.

The aim of the present randomized crossover study was to compare early respiratory recovery following aerobic and resistance exercise in healthy adults. The main goal was to compare the time needed to reach 90% of the individual's resting value of RR. Secondary outcomes included comparison of serial respiratory rate, oxygen uptake, one-minute heart-rate recovery, peripheral oxygen saturation, excess oxygen consumption during recovery, and perceived dyspnea. It was

hypothesized that aerobic exercise would elicit a greater immediate increase in respiratory rate, while resistance exercise would elicit a slower return to baseline respiratory rate due to continued metabolic and autonomic demands.

Materials and Methods

Design and setting of the study: The study was a randomized, counterbalanced crossover design in a university exercise-physiology laboratory. Participants were asked to perform one aerobic and one resistance exercise session in a random order with a washout of 72 hours or more between each session. The laboratory temperature was kept at 22–24°C and relative humidity at 40–60%. Testing was done at the same time of day for each person to minimize circadian variation.

Participants: Subjects were healthy adult volunteers aged 18–35 years, who were recruited by campus notices. The inclusion criteria were: BMI of 18.5–29.9 kg/m², participation in 90–300 minutes of recreational exercise per week and safe ability to perform treadmill and resistance exercise. The exclusion criteria included any known cardiovascular, pulmonary, metabolic, neurological or musculoskeletal disease, current smoking, use of beta-blockers, bronchodilators, stimulants or other drugs affecting cardiorespiratory responses, respiratory infection within the past four weeks, pregnancy, competitive endurance or strength training, and resting blood pressure of 140/90 mmHg or greater. Sixty-four people were screened, 62 were enrolled, and 60 people finished both sessions.

Determine the sample size and randomize the sample: The sample size was calculated for a paired comparison of the time it takes to recover from a respiratory-rate change. With a standardized paired effect size of 0.45, two-sided alpha of 0.05, a power of 0.90, and an expected attrition rate of 15%, a minimum of 58 participants was needed. A computer-generated sequence determined who was assigned to aerobic-first or resistance-first testing, with four participants assigned to each group. Allocation was hidden in sequentially numbered opaque envelopes opened following baseline assessment.

Preliminary evaluation and calibration: Data collected included height, body mass, resting blood pressure, history of health and physical activity. The incremental treadmill test with breath-by-breath gas analysis was used to determine peak oxygen uptake. One-repetition maximum for eight resistance exercises was estimated using submaximal repetition tests. Equipment and the Borg 0–10 dyspnea scale were introduced to the participants. They refrained from strenuous exercise, alcohol and caffeine for 24 hours and

from food for two hours prior to each session. Following 10 minutes of seated rest, baseline respiratory rate, heart rate, oxygen saturation, blood pressure, oxygen uptake, and dyspnea were measured.

Aerobic exercise protocol: The aerobic session began with a five-minute warm-up at 40% HRR, followed by 30 minutes at 70% HRR and a two-minute slow walk to the end of the session. The speed and grade of the treadmill was modified for the first 5 minutes to keep the target zone. The participants were asked not to grip the handrails unless for a short period of time to maintain their balance. Continuous recording of gas exchange and heart rate.

Resistance exercise protocol: There was a 5-minute cycle warm-up at the start of the resistance session. Three sets of 10 repetitions of eight exercises (alternating upper and lower body) were performed at 70% of estimated 1RM with 60 seconds between sets and 90 seconds between exercises. The load was reduced by 5% when necessary to maintain technique and exhalation during concentric phase was emphasized. Aerobic condition was matched within two minutes of session duration.

Recovery measures and outcomes: Participants sat upright, with no cool down activity, immediately after each exercise session. Measurements were taken over a 15 minute period. Prespecified analysis points were baseline, immediate recovery, and 1, 3, 5, 10, and 15 minutes. The primary outcome was the time to reach respiratory-rate recovery, which was the first time that respiratory rate fell into and stayed within $\pm 10\%$ of baseline for 60 seconds. Secondary outcomes included: respiratory rate at each time point, oxygen-uptake recovery time constant from a monoexponential model, cumulative excess oxygen consumption over 15 minutes, one-minute heart-rate recovery, oxygen saturation, and Borg dyspnea scores. Documentation of adverse symptoms was carried out.

Statistical Analysis: The data were analyzed with SPSS version 29.0. The Shapiro–Wilk test was used to check the continuous variables, which are presented as mean $\pm$ standard deviation or mean difference with 95% confidence interval.

Categorical variables are presented as frequency and percentage. Summary recovery endpoints were compared using paired t-tests. A 2 condition $\times$ time repeated measures analysis of variance was used to evaluate serial respiratory, oxygen-uptake, heart-rate and dyspnea responses, with Greenhouse–Geisser correction and Bonferroni adjusted

pairwise comparisons used when appropriate. The effect size was measured using partial eta-squared. Possible period, sequence, and sex effects were tested using a linear mixed model. Two-sided $p < 0.05$ was considered statistically significant.

Results

Sixty participants completed both exercise conditions and were included in the analysis. The sample comprised 32 men (53.3%) and 28 women (46.7%), with a mean age of 25.8 ± 4.6 years. Baseline cardiorespiratory measurements were within expected healthy ranges (Table 1). No participant experienced chest pain, syncope, oxygen desaturation below 94%, or a musculoskeletal adverse event. Exercise order was balanced, and no significant period or sequence effect was detected for the primary outcome ($p = 0.61$ and $p = 0.74$, respectively).

Respiratory rate showed a significant condition-by-time interaction ($F = 31.6$, $p < 0.001$, partial $\eta^2 = 0.349$). Immediately after exercise, respiratory rate was higher following aerobic exercise than resistance exercise (31.8 ± 4.3 vs 28.6 ± 4.1 breaths/min; $p < 0.001$). The pattern reversed during later recovery: at 10 minutes, respiratory rate remained higher after resistance exercise (18.9 ± 2.7 vs 17.2 ± 2.4 breaths/min; $p < 0.001$), and at 15 minutes it remained modestly elevated relative to the aerobic condition (16.5 ± 2.1 vs 15.6 ± 1.9 breaths/min; $p = 0.002$) (Table 2).

The primary outcome favored aerobic exercise. Mean respiratory-rate recovery time was 10.8 ± 3.2 minutes after aerobic exercise and 13.6 ± 3.9 minutes after resistance exercise, corresponding to a paired difference of 2.8 minutes (95% CI 1.9–3.7; $p < 0.001$; Cohen $d = 0.68$). Forty-seven participants (78.3%) recovered within 15 minutes after aerobic exercise compared with 33 participants (55.0%) after resistance exercise ($p = 0.006$). Oxygen uptake declined in both conditions but remained higher after resistance exercise from minute 5 onward. The modeled oxygen-uptake recovery time constant was 44.2 ± 9.1 seconds after aerobic exercise and 51.6 ± 10.7 seconds after resistance exercise ($p < 0.001$). Cumulative excess oxygen consumption during the 15-minute recovery period was also greater after resistance exercise (1.42 ± 0.31 vs 1.19 ± 0.28 L; $p < 0.001$). One-minute heart-rate recovery was larger after aerobic exercise (29.4 ± 7.2 vs 22.1 ± 6.5 beats/min; $p < 0.001$). Oxygen saturation remained stable, with no clinically important between-condition difference. Immediate dyspnea was greater after aerobic exercise, but scores were higher after resistance exercise at 10 minutes (Table 3). Sex did not significantly modify respiratory-rate recovery time (interaction $p = 0.28$).

Table 1: Baseline characteristics of participants (n=60)

Characteristic	Value	Range or n (%)
Age (years)	25.8 ± 4.6	18–35
Sex	—	Men 32 (53.3); women 28 (46.7)
Body mass index (kg/m ²)	23.4 ± 2.5	18.7–29.2
Weekly recreational exercise (min)	168 ± 54	90–290
VO ₂ peak (mL/kg/min)	42.8 ± 6.7	31.6–57.4
Resting respiratory rate (breaths/min)	14.7 ± 1.8	11–19
Resting heart rate (beats/min)	72.6 ± 8.4	56–91
Resting SpO ₂ (%)	98.0 ± 0.8	96–100

Data are mean ± standard deviation unless otherwise indicated. VO₂ peak, peak oxygen uptake; SpO₂, peripheral oxygen saturation.

Table 2: Serial respiratory rate and oxygen uptake during recovery

Outcome and time	Aerobic exercise	Resistance exercise	Paired difference	p-value
Respiratory rate, immediate (breaths/min)	31.8 ± 4.3	28.6 ± 4.1	3.2 (2.3 to 4.1)	<0.001
Respiratory rate, 3 min	24.9 ± 3.5	24.1 ± 3.3	0.8 (0.2 to 1.4)	0.010
Respiratory rate, 5 min	20.9 ± 3.0	22.8 ± 3.1	−1.9 (−2.5 to −1.3)	<0.001
Respiratory rate, 10 min	17.2 ± 2.4	18.9 ± 2.7	−1.7 (−2.2 to −1.2)	<0.001
Respiratory rate, 15 min	15.6 ± 1.9	16.5 ± 2.1	−0.9 (−1.5 to −0.3)	0.002
VO ₂ , immediate (mL/kg/min)	18.6 ± 3.4	16.1 ± 3.0	2.5 (1.8 to 3.2)	<0.001
VO ₂ , 5 min	7.8 ± 1.4	8.6 ± 1.5	−0.8 (−1.1 to −0.5)	<0.001
VO ₂ , 10 min	6.1 ± 1.2	7.0 ± 1.3	−0.9 (−1.2 to −0.6)	<0.001
VO ₂ , 15 min	5.4 ± 1.0	6.3 ± 1.2	−0.9 (−1.2 to −0.6)	<0.001

Values are mean ± standard deviation. Paired differences are aerobic minus resistance exercise with 95% confidence intervals. VO₂, oxygen uptake.

Table 3: Summary recovery endpoints following aerobic and resistance exercise

Recovery endpoint	Aerobic exercise	Resistance exercise	Effect estimate	p-value
Respiratory-rate recovery time (min)	10.8 ± 3.2	13.6 ± 3.9	MD −2.8 min	<0.001
Recovered within 15 min, n (%)	47 (78.3)	33 (55.0)	OR 2.96	0.006
VO ₂ recovery time constant (s)	44.2 ± 9.1	51.6 ± 10.7	MD −7.4 s	<0.001
15-min excess oxygen consumption (L)	1.19 ± 0.28	1.42 ± 0.31	MD −0.23 L	<0.001
One-minute heart-rate recovery (beats/min)	29.4 ± 7.2	22.1 ± 6.5	MD 7.3 beats/min	<0.001
SpO ₂ at 15 min (%)	98.1 ± 0.7	98.0 ± 0.8	MD 0.1%	0.31
Borg dyspnea, immediate	5.7 ± 1.3	4.8 ± 1.2	MD 0.9	<0.001
Borg dyspnea, 10 min	1.3 ± 0.8	1.7 ± 0.9	MD −0.4	0.001

MD, mean paired difference; OR, odds ratio; SpO₂, peripheral oxygen saturation; VO₂, oxygen uptake.

Discussion

This randomized crossover study revealed that there are different early recovery patterns following aerobic and resistance exercise in healthy adults. Aerobic exercise resulted in greater acute increases in RR, VO₂ and dyspnea, but these were more rapid to return to baseline.

Resistance exercise resulted in a lower immediate ventilatory peak, but a longer time to respiratory-rate normalization, slower oxygen-uptake recovery time constant, more cumulative excess oxygen consumption, and a smaller one-minute heart-rate recovery. The findings confirm the idea that the measure of peak physiological disturbance and recovery duration are two not mutually interchangeable measures of exercise stress. This increase in immediate respiratory rate following

treadmill exercise was predicted due to the increased requirement for continuous large muscle activity at 70% of the heart-rate reserve for oxidative metabolism and carbon dioxide elimination. The transition kinetics of oxygen uptake are sensitive to the workload and exercise domain, and the oxygen uptake increases dynamically until the cardiopulmonary system reaches the metabolic requirement [2]. When work is stopped, the oxygen demand of the muscles decreases rapidly, allowing a relatively large decrease in ventilation. In the current study, aerobic exercise thus created a higher starting point after exercise and a shorter trajectory towards baseline. Physiologically, it makes sense that the slower recovery after resistance exercise occurs. Repeated sets at 70% of 1RM cause intermittent ischemia, hydrogen ion accumulation, inorganic phosphate

accumulation, phosphocreatine depletion, elevated levels of catecholamines, and ongoing requirement for substrate replenishment. The literature on excess post-exercise oxygen consumption suggests that the nature of the exercise and its intensity affect the quick and slow recovery phases [3]. The increased 15 min oxygen uptake and cumulative excess oxygen consumption following resistance training are indicative of continued metabolic recovery despite the lower initial respiratory rate.

The autonomic findings confirm this interpretation. After aerobic exercise, one-minute HRR was 7.3 beats/min higher, indicating quicker early parasympathetic reactivation. Vagal reactivation during the first minute is the primary determinant of HRR [4] with sympathetic withdrawal and circulating catecholamines over the following minutes. Goldberger and colleagues showed that post-exercise measures can reflect parasympathetic reactivation, but that no single measure is a complete measure of autonomic recovery [5]. There was a similar delay in HR and RR recovery following resistance exercise, indicating a more general persistence of post-exercise physiological activation.

Our results also confirm that acute resistance training can result in a greater short-term decrease in parasympathetic modulation compared to aerobic training [6]. Aerobic exercise, however, has been linked to better cardiac autonomic regulation among healthy young people [7]. These chronic training observations do not provide direct evidence of the acute mechanisms observed in the current study, but do suggest that exercise mode affects the balance of sympathetic activation, parasympathetic restoration and cardiorespiratory recovery.

There have been conflicting reports on post-exercise metabolism in comparative studies, as the structure of the protocols is important. Jung et al. noted various excess oxygen-consumption patterns in continuous, interval and resistance training [8].

In aerobically fit women, Greer et al. showed that both circuit resistance training and HIIT can induce significant post-exercise oxygen consumption, depending on the exercise dose and time window used [9]. Pilon et al. showed that in older adults, recovery demand after isocaloric resistance was higher than after aerobic exercise, favoring a mode-specific persistence of recovery demand [10]. The present study builds on this theme and focuses on normalizing respiratory rate, not just energy expenditure, in younger healthy adults. There is evidence of elevated metabolism for many hours following intense resistance training [11]. The present protocol was not as severe, and recovery was noted for 15 minutes, but the direction of effect was similar. It has also been suggested that the

sequence of exercise may not be as important as the dose and intensity of the individual sessions in studies of concurrent exercise [12].

In general, high-intensity interval exercise resulted in an increase in excess oxygen consumption compared to moderate continuous exercise, but the magnitude of the increase is relatively small and depends on the protocol used [13] (Panissa et al.). Recovery is also influenced by intensity distribution, in addition to the aerobic or resistance designation. The extended recovery period following resistance training may have been due to repeated local efforts and brief recovery periods.

Recovery can be altered by training status: resistance training has enhanced heart-rate recovery and complexity in young men, and this can be reversed following detraining [14]. Other reviews indicate that factors such as intensity, fitness, posture, environment, and the duration of observation affect autonomic recovery estimates [15]. These sources of variation were minimized by restricting the sample to recreationally active adults and by standardizing the testing conditions.

A high respiratory rate immediately following aerobic exercise does not necessarily mean that recovery is poor, it could just be a big, but quick, response. Resistance exercise might appear less demanding at termination and longer respiratory and autonomic normalization. There should therefore be a focus on both peak response and recovery slope when scheduling repeated bouts and interpreting clinical exercise tests; 3-5 minutes of observation may be insufficient to assess residual load following resistance exercise.

There are some restrictions to be taken into account. The duration of the sessions was matched but not exactly the same for energy expenditure and muscle work. Only 15 minutes of recovery were observed and blood lactate, catecholamines, tidal volume and minute ventilation were not primary measures. Estimated one-repetition maximum was used and the young healthy sample limits generalizability. Future studies should employ isocaloric protocols, longer monitoring duration, and mechanistic measurements in athletic and clinical populations.

Conclusion

Aerobic exercise resulted in higher immediate increase in RR and dyspnea and faster recovery of RR, O₂ uptake and HR. Resistance exercise caused a longer increase in ventilation and oxygen consumption, suggesting that there was continued metabolic and autonomic demand following the end of the visible exercise bout. The magnitude and duration of recovery can be monitored to optimize exercise prescription, recovery scheduling and

interpretation of post-exercise cardiorespiratory responses in healthy adults.

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