Acute Normobaric Hypoxia Exposure and Excess Post-Exercise Oxygen Consumption

James W. Navalta; Elizabeth A. Tanner; Nathaniel G. Bodell

BACKGROUND: Excess postexercise oxygen consumption (EPOC) is an elevation in oxygen consumption ($\dot{V}O_2$) following exercise. Altitude decreases maximal oxygen uptake; however, studies are equivocal concerning the effect on resting metabolic rate. The EPOC response has not been studied with normobaric hypoxia. The purpose was to observe EPOC following constant-load cycling in normobaric hypoxia.

METHODS: Subjects ($N = 7$ women, 7 men) completed resting metabolic rate testing between 06:00 and 08:30. Constant workload cycle exercise was performed (10 min at 100 W) while breathing air from an altitude simulator under the following conditions: normoxic control (CON), 3353 m (11,001 ft; HI), and 6401 m (21,001 ft; EXT). Subjects completed remaining conditions in a counterbalanced order. Upon completion, participants were reconnected to the metabolic system until a running 5-min average of $\dot{V}O_2$ values returned to baseline (EPOC duration). Magnitude was determined by summing the net oxygen consumption each minute during the EPOC period. Data were analyzed using 2×3 repeated measures ANOVA.

RESULTS: No sex differences were detected for any variable. EPOC duration increased significantly at each simulated altitude increase (CON = 15.2 ± 1.9 vs. HI = 20.7 ± 1.7 min) (HI vs. EXT = 28.1 ± 2.6 min). Likewise, EPOC magnitude increased significantly at each simulated altitude (CON = 73.5 ± 9.9 vs. HI = 99.1 ± 9.3 ml O_2) (HI vs. EXT = 139.7 ± 14.3 ml O_2).

DISCUSSION: The EPOC response to simulated altitude represents elevated caloric expenditure that must be accounted for. Individuals who are active at altitude must consider the increased caloric deficit despite a loss of appetite that is common with altitude exposure.

KEYWORDS: simulated altitude, aerobic exercise, magnitude, metabolic response.

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The interaction between physiological exercise responses and altitude increases has received recent renewed attention and has specific applications. Some findings suggest benefits in the exercise and altitude interaction, whereas other findings point to caution. For example, Helfer et al. found that 4 wk of voluntary isocapnic hyperpnea respiratory muscle training (3 d/wk, 30 min) improved exercise time at normobaric simulated altitude and proposed it to be beneficial for individuals who complete physical activity tasks at altitude.[16] Furthermore, there is evidence that mathematical processing and running memory are unaffected by time spent in normobaric hypoxia (30 min) and that improvements to these tests of executive function occur with exercise.[34] On the other hand, DiPasquale et al. reported that exercise of longer duration exacerbated the severity of acute mountain sickness induced by simulated hypoxia,[10] and Webb et al. proposed that increased physical activity increases the risk of decompression sickness in a flight simulator with ascent to 6858 m (22,500 ft).[36] While the previously mentioned investigations focused on the interplay between exercise and altitude, it is unknown what to expect in the postexercise recovery period with respect to oxygen uptake.

Oxygen consumption ($\dot{V}O_2$) that remains elevated above the baseline values after exercise has terminated is known as excess

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postexercise oxygen consumption (EPOC).[13] As oxygen remains elevated, the increased metabolism cost continues to be a factor in the thermic effect of activity.[21] EPOC can be affected by a number of factors, including the intensity of activity,[19,20,29] exercise duration,[20] and modality.[21,23] In general, as exercise intensity increases both the magnitude and duration of EPOC increases.[29,31] If exercise intensity is held constant, the EPOC response increases linearly as exercise duration is lengthened.[20,21] Excess postexercise oxygen consumption has been reported following various exercise modalities, including treadmill running,[9] cycling,[29,31] resistance training,[11,35] and upper body arm cranking.[23,30] Across modalities it appears that the engagement of a greater amount of muscle mass results in a larger EPOC response.[9,11,23]

Arrival at altitude has the effect of decreasing maximal oxygen uptake and this corresponds with the decrease of the partial pressure of oxygen and lower blood oxygenation levels.[5,26] With the introduction of exercise at higher altitude, any given absolute workload represents a greater intensity when compared to sea level due to the associated decrease in aerobic capacity (i.e., the workload is carried out closer to the $\dot{V}O_{2peak}$).[26] Early studies have reported basal metabolic rate to increase upon initial exposure to altitude and then to decrease with acclimation.[4,18] However, other investigations have reported no change in resting metabolic rate with high altitude exposure.[1,27] Taken together, it is unclear what effect acute altitude exposure will have on the EPOC response.

To our knowledge, the excess postexercise oxygen consumption response has not been studied with normobaric hypoxia as an independent variable. Determining the EPOC response to altitude is valuable, as it represents a source of potentially elevated caloric expenditure that must be accounted for given that carbohydrates are preferentially consumed with altitude exposure.[3] This would have an influence on recovery from exercise as well as future bouts of work. Thus, individuals who are active at altitude may need to account for this increased caloric deficit despite a loss of appetite that is common with altitude exposure.[33] We hypothesized that both the EPOC magnitude and duration would increase linearly in response to exercise carried out at the same absolute workload, but at increasing simulated altitudes. Therefore, the purpose of this investigation was to observe the EPOC response to constant-load cycle exercise performed under the simulated normobaric altitudes of 3353 m (11,001 ft; HI) and 6401 m (21,001 ft; EXT).

METHODS

Subjects

A repeated measures research design was used, with all subjects completing baseline oxygen consumption measures, a 10-min cycle ergometry protocol, and a postexercise EPOC session each day. Testing sessions were completed in a counterbalanced order, applied in a normalized Latin square design. The independent variable was the fraction of oxygenated air consumed during the cycle period (19.4% O_2 or the equivalent of a normobaric control, 12.7% O_2 corresponding to high altitude, and 8.7% O_2 corresponding to extreme altitude). Dependent variables were obtained during the course of the excess postexercise oxygen consumption period and include EPOC duration and EPOC magnitude.

Potential participants were screened using the ACSM Health Risk Questionnaire and those who were stratified as low risk according to the American College of Sports Medicine algorithm were provided an informed consent document that was approved by the institutional review board (protocol #813,184-3). All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study. There were 14 individuals who participated (women $N = 7$, men $N = 7$) with similarities in age between sexes (women = 27 ± 8 yr, men = 29 ± 7 yr, $P = 0.59$). Men were significantly taller than the women (women = 165 ± 7 cm, men = 179 ± 5, $P = 0.001$) and had more body mass (women = 60 ± 9 kg, men = 86 ± 18, $P = 0.005$).

Equipment

Cycle exercise was performed on a Wattbike Pro (Nottingham, UK), which is a stationary cycle ergometer that features a dual air and magnetic braking system. This ergometer provides output on a real-time LED display and is capable of providing medium to high resistance up to approximately 3760 W. Increases in altitude were simulated with the Everest Summit II generator (Hypoxico Inc., New York, NY) fitted with a high altitude adapter capable of simulating altitudes up to 6401 m (21,001 ft). This hypoxicator reduces the fraction of oxygen in the inspired air through digital controls; this air was delivered to subjects through a HEPA filter, a series of plastic hosing and rebreathing bags, and was fitted to the face with a neoprene mask. Oxygen consumption following the cycle bout was obtained using the Moxus modular metabolic system (AEI Technologies, Pittsburgh, PA). This metabolic analysis system features independent oxygen and carbon dioxide analyzers, a pneumotach breath volume measurement system, and sample pump and flow controller. Subjects were connected to the metabolic system through plastic tubing and a two-way breathing valve with mouthpiece and headgear.

Procedures

Participants reported to the Exercise Physiology Laboratory for testing on three separate occasions separated by at least 48 h: 1) cycle exercise performed at acclimated altitude (the altitude of the laboratory and surrounding community is 619 m/2031 ft; CON); 2) cycle exercise performed at a simulated normobaric altitude of 3353 m (11,001 ft) elevation (effective O_2 percentage = 12.7%; HI); and 3) cycle exercise performed at a simulated normobaric altitude of 6401 m (21,001 ft) elevation (effective O_2 percentage = 8.7%; EXT). Testing sessions were completed by participants in a counterbalanced order according to a standard form Latin square design.

All testing was carried out in the morning starting at between 06:00 and 08:30 following a usual night of sleep. There is no standard period for obtaining resting oxygen measurements to be used in EPOC, as previous research has used no period,[25] 20 min,[22] 30 min,[23] 40 min,[30] and 1 h.[29] Therefore, prior to each exercise test, a resting oxygen measure was obtained by having participants sit upright quietly in a comfortable chair for 15 min while breathing room air into a metabolic analysis system. A 5-min average of the final rest period was taken as the baseline value, as has been used in other investigations.[22] Participants then performed 10 min of cycle exercise at a constant workload of 100 W while breathing air from an altitude simulator. Upon completion of the exercise bout, participants were reconnected to the metabolic analysis system and sat quietly breathing room air until a running 5-min average of oxygen values returned to, or below, baseline values (taken as the EPOC duration). Magnitude was determined by summing the net oxygen consumption for each minute during the EPOC period.

Statistical Analysis

EPOC and performance data were analyzed using a 2 (sex) $\times$ 3 (condition) ANOVA with repeated measures on the second variable (IBM SPSS Statistics, version 21, Chicago, IL). Significance was accepted at the $P \leq 0.05$ level.

RESULTS

No sex by condition interactions were detected for any variable ($P > 0.05$). Additionally, there was no difference between condition for resting metabolic rate, or any cycle performance variable (see **Table I**).

EPOC duration increased significantly with each step up in simulated altitude (CON = 15.2 $\pm$ 1.9 vs. HI = 20.7 $\pm$ 1.7 min, $P = 0.002$; HI vs. EXT = 28.1 $\pm$ 2.6 min, $P = 0.006$) (**Fig. 1**). Likewise, EPOC magnitude increased significantly as simulated altitude was raised (CON = 73.5 $\pm$ 9.9 vs. HI = 99.1 $\pm$ 9.3 ml O_2, $P = 0.002$; HI vs. EXT = 139.7 $\pm$ 14.3 ml O_2, $P = 0.001$) (see **Fig. 2**).

DISCUSSION

The aim of this investigation was to determine the excess postexercise oxygen consumption response to increasing levels of simulated altitude following a bout of cycle ergometry. We hypothesized that EPOC duration and magnitude would increase with simulated altitude increases and exercise carried out at the same absolute workload. Our hypotheses were confirmed, as both EPOC duration as well as magnitude increased between 619 and 6401 m (2031 and 21,001 ft) of normobaric hypoxia. Ergometry at each new simulated altitude level produced EPOC responses that were significantly greater.

To our knowledge, this is the first investigation to determine the excess postexercise oxygen consumption response to normobaric hypoxia. EPOC has been reported following different exercise modalities such as treadmill running,[9] cycling,[29,31] resistance training,[11,35] and upper body arm cranking.[23,30] It appears that across modalities, using a greater amount of muscle mass or increasing the exercise intensity results in an increased EPOC response.[9,11,23] In the current investigation, the workload and muscular contribution were held constant (i.e., no significant differences in cycle performance variables between conditions), but increasing simulated altitude was applied. Using this constant, it can be inferred that increases in simulated altitude represent increases in relative intensity level.

The EPOC duration in the current investigation increased significantly as normobaric hypoxia reflecting simulated altitude was raised despite exercise being performed at the same absolute workload. The closest investigation that we have to compare the current results with was carried out by Sedlock et al.[29] in which high intensity exercise (cycle ergometry at 75% $\dot{V}O_{2max}$ to expend 300 kcal) produced a significantly longer EPOC duration when compared to low intensity exercise (50% $\dot{V}O_{2max}$ to expend 300 kcal). Similarly, Short and Sedlock[31] observed a longer EPOC duration in untrained compared to trained participants who completed cycle exercise at the same relative or absolute workloads. As we noted previously, exercising at increasing simulated altitudes represents a relative increase in intensity level, and as such it should not be surprising that each successive step in normobaric hypoxia resulted in a significantly greater EPOC duration.

EPOC magnitude in the current study increased significantly as simulated altitude was applied in a stepwise manner. We are unaware of similar reports in the literature and relay this phenomenon for the first time. Direct comparisons to the literature are difficult, as there is no established criterion for the EPOC magnitude measurement. Nevertheless, magnitude significantly increases with exercise intensity,[29] incorporating a greater amount of muscular engagement,[23] and with intermittent bouts of exercise when compared to one continuous session where the caloric expenditure is controlled.[9] We can add that

Table I. Metabolic and Cycle Performance Variables at Differing Levels of Simulated Altitude.

	NORMOXIC CONTROL	HIGH SIMULATED ALTITUDE	EXTREME SIMULATED ALTITUDE	*P*-VALUE
RMR ($ml \cdot kg^{-1} \cdot min^{-1}$)	3.9 $\pm$ 0.5	3.9 $\pm$ 0.3	3.9 $\pm$ 0.4	0.937
Cycle duration (min)	10 $\pm$ 0	10 $\pm$ 0	9.2 $\pm$ 2	0.16
Power (W)	98 $\pm$ 4	100 $\pm$ 4	95 $\pm$ 9	0.08
Cadence (rpm)	64.3 $\pm$ 8.9	64.2 $\pm$ 7.8	64.4 $\pm$ 9.2	0.998
Revolutions (total)	650.7 $\pm$ 89.9	652.8 $\pm$ 86.0	597.1 $\pm$ 172.8	0.416
Distance (km)	4.8 $\pm$ 0.1	4.8 $\pm$ 0.1	4.3 $\pm$ 1.1	0.103

RMR: resting metabolic rate.

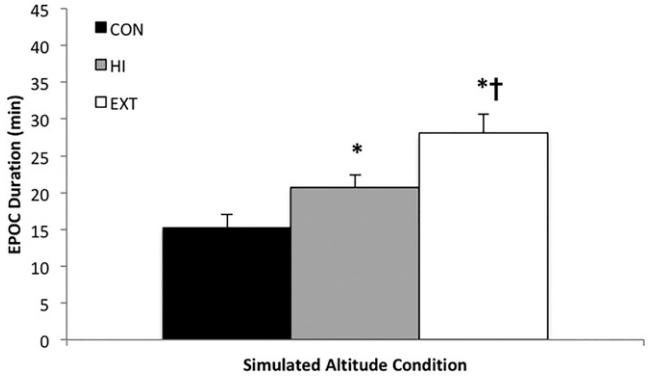

Fig. 1. Excess postexercise oxygen consumption duration in participants (N = 14) who performed 10 min of cycling at 100 W under various simulated altitude conditions: CON = 619 m (2031 ft); HI = 3353 m (11,001 ft); EXT = 6401 m (21,001 ft). Data indicate means and standard deviations (bars). *Indicates significant difference from CON; †indicates significant difference from HI.

magnitude significantly increases as altitude is applied in a normobaric simulated environment.

Many investigations into the effect of exercise on the EPOC response have controlled the subject pool to only men[2,14,29] or women.[6,28,35] A limited number of investigations have used both men and women, and the results are equivocal. For example, Smith and McNaughton[32] reported a sex difference in participants who completed cycle ergometry at intensities between 40–70% $\dot{V}O_{2max}$ (men having a higher EPOC response compared to women), whereas Short and Sedlock[31] reported no sex difference in both trained and untrained male and female participants who performed cycle ergometry at the same relative and absolute workloads. While our results are specific to a simulated altitude condition, we also observed no sex difference between the healthy young men and women that participated in the current investigation.

There are some limitations to the current investigation, the primary being the use of simulated altitude (normobaric hypoxia) to mimic changes in elevation.[8] Thus, the application of these findings should be used with caution for individuals traveling to various altitudes, as the elevation changes accompanying hypoxia will occur in a hypobaric environment. Additionally, caution should be used when generalizing these results to the population at large, as increasing evidence suggests that there are responders and nonresponders to altitude training.[7,12,15] In the current investigation, two individuals were unable to complete the 10-min exercise bout at the highest simulated altitude condition (while anecdotal, each noted ventilatory insufficiencies), suggesting that they may not have an appropriate response to altitude exposure. Future studies should attempt to identify these potentially susceptible individuals and determine whether a modulated EPOC response occurs. A final limitation is that maximal aerobic capacity of participants in the current investigation was not determined. Such a measurement would have allowed for the determination of EPOC values relative to aerobic capacity and for potential stratification based on fitness level. Future investigators are encouraged to include $\dot{V}O_{2max}$ as a measurement in order to more fully explore potential postexercise oxygen recovery differences that may be present at increasing simulated altitudes between individuals of different cardiorespiratory capacities.

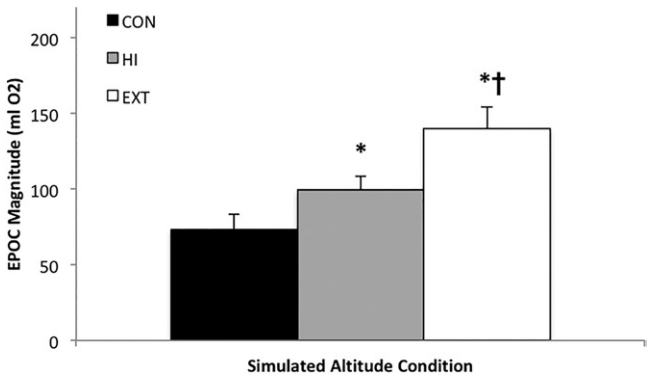

Fig. 2. Excess postexercise oxygen consumption magnitude in participants (N = 14) who completed 10-min cycle trials at 100 W under three simulated altitude conditions: CON = 619 m (2031 ft); HI = 3353 m (11,001 ft); EXT = 6401 m (21,001 ft). Data indicate means and standard deviations (bars). *Indicates significant difference from CON; †indicates significant difference from HI.

Determining the EPOC response to altitude is important because it represents a source of elevated caloric expenditure that must be accounted for given that carbohydrates are preferentially used with altitude exposure.[3] This has an influence on recovery from exercise as well as future bouts of work. Thus, individuals who are active at altitude must account for this increased caloric deficit despite a loss of appetite that is common with altitude exposure in order to further minimize the loss of cross-sectional muscle area that occurs.[17,24]

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