Exercise Improves Mood State in Normobaric Hypoxia

Yongsuk Seo; Curtis Fennell; Keith Burns; Brandon S. Pollock; John Gunstad; John McDaniel; Ellen Glickman

BACKGROUND: The purpose of this study was to quantify the efficacy of using exercise to alleviate the impairments in mood state associated with hypoxic exposure.

METHODS: Nineteen young, healthy men completed Automated Neuropsychological Assessment Metrics-4th Edition (ANAM4) versions of the mood state test before hypoxia exposure, after 60 min of hypoxia exposure (12.5% O_2), and during and after two intensities of cycling exercise (40% and 60% adjusted $\dot{V}O_{2max}$) under the same hypoxic conditions. Peripheral oxygen saturation (SpO_2) and regional cerebral oxygen saturation (rSO_2) were continuously monitored.

RESULTS: At rest in hypoxia, Total Mood Disturbance (TMD) was significantly increased compared to baseline in both the 40% and 60% groups. TMD was significantly decreased during exercise compared to rest in hypoxia. TMD was also significantly decreased during recovery compared to rest in hypoxia. SpO_2 significantly decreased at 60 min rest in hypoxia, during exercise, and recovery compared to baseline. Regional cerebral oxygen saturation was also reduced at 60 min rest in hypoxia, during exercise, and recovery compared to baseline.

DISCUSSION: The current study demonstrated that exercise at 40% and 60% of adjusted $\dot{V}O_{2max}$ attenuated the adverse effects of hypoxia on mood. These findings may have significant applied value, as negative mood states are known to impair performance in hypoxia. Further studies are needed to replicate the current finding and to clarify the possible mechanisms associated with the potential benefits of exercise on mood state in normobaric hypoxia.

KEYWORDS: Mood state, Hypoxia, Exercise.

Seo Y, Fennell C, Burns K, Pollock BS, Gunstad J, McDaniel J, Glickman E. *Exercise improves mood state in normobaric hypoxia.* Aerosp Med Hum Perform. 2015; 86(11):976–981.

Monitoring mood state is important for everyday activities and sports performance, as well as occupational activities. Transient mood state is related to many cognitive functions and mental processes such as planning, attention, decision making, working memory, problem solving, and behavior control.[36] At high altitudes, especially above 4,300 m, there is a marked reduction in positive mood state and concurrent increases in negative mood state.[29] Specifically, vigor decreases with an increase in fatigue and seven physical symptoms (i.e., cerebral acute mountain sickness (AMS), respiratory AMS, cold, distress, exertion, muscular discomfort, and fatigue).[29] Another study by de Aquino Lemos et al. reported levels of depression, anger, and fatigue were significantly higher and vigor was significantly lower at altitude.[10]

There have been numerous studies that have observed the beneficial effects of acute bouts of exercise performed in normoxic conditions on mood state.[11,32,33] An acute bout of exercise performed at 60% $\dot{V}O_{2max}$ for 10 min promoted vigor and decreased fatigue and negative mood state. In addition Hansen and colleagues[14] reported that confusion was attenuated during 20 min of exercise. A review paper by Yeung[38] reported that moderate intensity (50–70% $\dot{V}O_{2max}$) exercise improved mood states such as anxiety, vigor, and exhilaration. Also, Ekkekakis et al.[11] reported that exercise duration in excess of 10-15 min may have a positive impact on mood state. However, very little is known about the mechanisms in which exercise enhances mood state and whether it can counteract or abolish the mood depressing effects observed in a hypoxic environment. Previous reports indicate acute or chronic exercise improves an individual's mood state by enhanced brain neural circuitry.[9] It has been reported that aerobic exercise changes blood flow and hormone release which enhances activity in neural circuits within the prefrontal cortex and other neural structures.[35] The neural

From the Department of Exercise Physiology, Kent State University, Kent, OH.

This manuscript was received for publication in January 2015. It was accepted for publication in August 2015.

Address correspondence to: Yongsuk Seo, Kent State University, Department of Exercise Physiology, 162 MACC Annex, Kent, OH 44242; yseo@kent.edu.

DOI: 10.3357/AMHP.4263.2015

circuits enhanced by exercise improve mood state, cognition, and motor control.[35] In detail, acute exercise reduces negative mood state (e.g., anxiety, tension, depression)[8,10,24] and enhances positive mood state.[18] The possible mechanism of improvement in mood state and cognition by exercise might be changed in neurotransmitter and central neural activity.[7,13] The change in electro cortical activity regulate emotion and cognitive function. Specifically, low frequency activity (α-activity) ranging from 7.5 to 12.5 Hz of electroencephalography (EEG) demonstrated an enhancement of arousal and mood state.[4,28] As aforementioned, hypoxia also induced negative mood effect[10] and, although mood disturbances may be trivial in those who live at altitude or are at least acclimatized to altitude, many people work or enjoy outdoor activities at altitude who are not acclimatized and, based on previous reports,[11,30] these individuals may experience negative mood disturbances.

As such, the purpose of this study was to quantify the effects of exercise on mood state at rest and during exercise and recovery in hypoxic conditions. It is hypothesized that mood state in hypoxia would be impaired following 60 min of hypoxic exposure, but would be improved during two cycle ergometer exercise intensities (40% and 60% $\dot{V}O_{2max}$) and recovery.

METHODS

Subjects

There were 19 young, healthy Caucasian men who volunteered for the current investigation and reported to the laboratory on two separate occasions (familiarization and hypoxia trial) separated by at least 3 d. Through completion of a medical history screening, participants were excluded if they reported history of medical, neurological, developmental, or psychiatric disorders. Also, subjects were excluded if they were a smoker or were exposed to normobaric hypoxia or an altitude above 2500 m within 2 mo prior to participation in the study. The participants were assigned as either Low (40% $\dot{V}O_{2max}$) or Moderate (60% $\dot{V}O_{2max}$) exercise intensities. The separation of the two groups was selected to minimize exposure to hypoxia, boredom, and possible learning effects. The study protocol was approved by the Institutional Review Board at Kent State University. All participants were given a written informed consent form before participating. The characteristics of the participants are displayed in **Table I**.

Equipment

Mood state (MS) was assessed with Automated Neuropsychological Assessment Metrics-4th Edition (ANAM4). The ANAM4 mood state is designed to assess seven specific categories of mood: anger, anxiety, depression, fatigue, happiness, restlessness, and vigor. Specifically, through the use of a laptop, 42 words expressing various emotions were presented to the subject and they were instructed to choose a number between 0 and 6 with 0 being "Not at all" and 6 being "Very Much" for each emotion presented. These emotions are associated with the seven categories of mood state. Total Mood Disturbance (TMD) was calculated as: TMD = (negative mood – positive mood) where negative mood is the sum of the anger, anxiety, depression, fatigue and restlessness scores, and positive mood is the sum of happiness and vigor scores. The higher TMD score indicates greater negative mood state.

Table I. Physiological Characteristics Between Groups.

	LOW ($N = 10$)	MODERATE ($N = 9$)	*P*-VALUE
$\dot{V}O_{2max}$ (ml · kg^{-1} · min^{-1})	48.3 ± 8.6	45.2 ± 6.6	0.441
Adj $\dot{V}O_{2max}$ (ml · kg^{-1} · min^{-1})	35.3 ± 6.3	33.0 ± 4.8	0.443
HRmax (bpm)	183 ± 13	185 ± 13	0.781
40%W	58 ± 16	43 ± 13	0.234
60%W	107 ± 23	93 ± 22	0.064
Age (yr)	24 ± 3	25 ± 4	0.905
Height (cm)	177 ± 4.8	175.6 ± 8.6	0.634
Weight (kg)	78.4 ± 11.4	78.1 ± 6.9	0.949

Data are presented as mean ± SD.

Procedures

Each group underwent a prescreening followed by two experimental sessions in a thermoneutral environment (22–24°C). On the first day, participants were introduced to the simulated altitude chamber and familiarized with the protocol and instrumentation including performing the mood state tests a minimum of three times. Participants then conducted two cycling protocols on a Lode Excalibur 1300W ergometer (Lode Excalibur Sport, Lode, Groningen, Netherlands) to determine the submaximal exercise intensities that would be used during the subsequent experimental trial. The first protocol required participants to pedal through three 4-min stages at 50, 100, and 150 W to determine their $\dot{V}O_2$ -workrate relationship. Upon completion of the first protocol, participants rested for at least 20 min or until their HR returned to baseline. The second protocol was a $\dot{V}O_{2max}$ test which required participants to pedal on the cycle ergometer through increasing stages of intensity until volitional fatigue to estimate ventilatory threshold (VT) and maximal oxygen consumption ($\dot{V}O_{2max}$). The max test began at 20 W and increased by 25 W every minute until volitional fatigue of the participant.[2] During both protocols, $\dot{V}O_2$ and HR were measured with indirect open circuit spirometry (Parvo, Metabolic Cart, Sandy, UT) and a Polar heart rate monitor (Polar RS800 CX, Polar Electro Oy, Kempele, Finland). The combination of these two protocols allowed the determination of the power output required to elicit 40% and 60% of altitude specific adjusted $\dot{V}O_2$ work rates during the subsequent visit.[12,39]

On the day of the experimental trial (**Fig. 1**), participants reported to the Exercise Physiology Laboratory at Kent State University following a 3 h fast to reduce swings in blood sugar (i.e., obtain a more stable substrate utilization),[26] reduce the risk of subjects becoming nauseous, and reduce the thermic effect of feeding. Participants were initially equipped with Near-Infrared Spectroscopy (NIRS) sensors over the frontal lobe (Somanetics, Troy, MI) for regional cerebral oxygenation (rSO_2) monitoring as well as a digit pulse-oximeter (Oxi-Go, Roslyn, NY) to

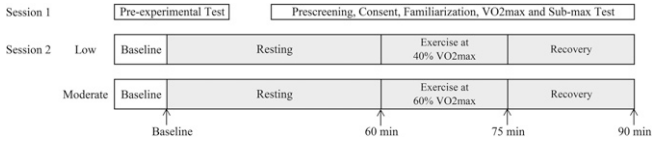

Fig. 1. Experimental design and procedure. Shaded gray area occurred in hypoxic conditions.

monitor peripheral oxygen saturation (Spo_2). Subjects sat on a chair quietly during the 5 min baseline measurement of Mood State (MS), baseline Spo_2, rSo_2, and rating of perceived exertion (RPE) were also obtained. The RPE[5] was assessed simultaneously with Spo_2 and rSo_2. Specifically an RPE scale was held up directly in front of the subject and they were asked to point to their perceived effort just prior to mood state assessment. Thus, the individual's subjective perception of muscle fatigue and physical stress was assessed with measurement of Spo_2 and rSo_2. Although the near-infrared spectroscopy (NIRS) technique for rSo_2 appears to underestimate the cerebral oxygenation compared to transcranial Doppler or positron emission tomography,[22] it has become an acceptable, noninvasive method for the measurement of cerebral oxygenation.[37]

Following baseline measurements, participants entered the hypoxia chamber where the oxygen concentration was "set" at 12.5% and had resulting similar increases in %N2 but no changes in the $\%CO_2$. The 12.5% O_2 is equivalent to the partial pressure of oxygen present at an altitude of 4300 m (14,110 ft). We applied normobaric hypoxia to minimize the risk of decompression sickness associated with low barometric pressure, and focused on the effect of oxygen concentration in ambient air. Participants then rested in a chair for 60 min. Mood state, Spo_2, rSo_2, and RPE were measured from min 57–60. Upon completion, the individuals were instructed to sit on the cycle ergometer.

As depicted in Fig. 1, participants performed a 15-min bout of exercise on the cycle ergometer at either 40% or 60% of adjusted $\dot{V}o_{2max}$ followed by a 15 min recovery period. Exercise intensity was assigned in a counterbalanced fashion. Mood state, Spo_2, rSo_2 and RPE were again measured from 7–10 min during exercise and following the 15-min recovery period. Upon completion of the measurements, participants were removed from the hypoxia chamber.

Statistical Analysis

Using SPSS 17.0, a two group (40%, 60%) by four time points (Baseline, Rest, Exercise, Recovery) repeated measures ANOVA was conducted for total mood disturbance as well as the seven categories of mood state: anger, anxiety, depression, fatigue, happiness, restlessness, vigor, Spo_2, RPE and rSo_2. If a significant interaction was found, post hoc paired-sample and unpaired tests were used to determine the differences between group(s) at each time points. For TMD, happiness, vigor, rSo_2, and Spo_2, paired sample *t*-test was used to determine the main effect for time within the 40% and 60% exercise intensities. Independent sample *t*-test was used for RPE between the 40% and 60% exercise intensities. For fatigue, independent sample *t*-test and paired sample *t*-test were used to determine the main effect for group (independent) and time (paired). Data are presented as mean ± SD and the level of significance was set a priori at $P \leq 0.05$.

RESULTS

Total Mood Disturbance demonstrated a main effect for time [$F(3,51) = 5.604$, $P = 0.002$, power = 0.927], but no main effect for group [$F(1,17) = 1.480$, $P = 0.240$, power = 0.210] and no main effect for time by group interaction [$F(3,51) = 2.167$, $P = 0.103$, power = 0.520]. TMD did not differ between the 40% and 60% groups. Therefore, for all subsequent analysis the 40% and 60% groups were combined into one group. Baseline TMD values were similar between groups (Low: -73.8 ± 71.1 vs. Moderate: -52.9 ± 33.1, $P = 0.432$). TMD increased at 60 min rest in hypoxia compared to baseline ($P = 0.002$) and decreased during exercise compared to 60 min rest in hypoxia ($P = 0.009$). Further, TMD was significantly decreased during recovery compared to 60 min rest in hypoxia ($P = 0.015$) (**Fig. 2**).

Individual two-way repeated measures ANOVA indicated that anger, anxiety, depression, and restlessness did not show a main effect for group, time, and group × time interaction ($P \geq 0.05$). However, fatigue demonstrated a main effect for time [$F(3,51) = 5.996$, $P = 0.001$, power = 0.943] and group × time interaction [$F(3,51) = 3.388$, $P = 0.025$, power = 0.733] but no main effect for group [$F(1,17) = 0.298$, $P = 0.592$, power = 0.081]. Independent sample *t*-test revealed that fatigue was significantly higher in the Moderate group during exercise compared to the Low group (Low: 7.6 ± 9.5 vs. Moderate: 17.4 ± 8.6, $P = 0.031$). Within the Low group, fatigue was significantly increased at 60 min rest in hypoxia compared to baseline ($P = 0.05$). Fatigue subsequently decreased during exercise ($P = 0.019$) and recovery ($P = 0.029$) compared to 60 min rest in hypoxia. Fatigue within the Moderate group did not differ

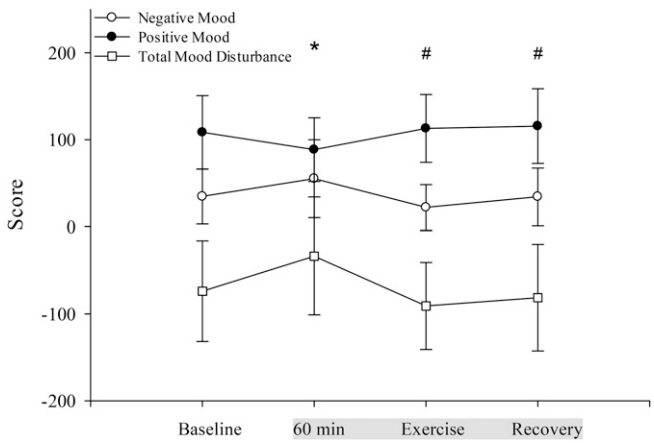

Fig. 2. Change in Total Mood Disturbance, Negative Mood, and Positive Mood at baseline, 60 min rest, exercise and recovery in hypoxia. Shaded gray area occurred in hypoxic conditions. $^{*}P < 0.05$, vs. Baseline; $^{\#}P < 0.05$, vs. Rest in hypoxia; mean ± SD.

across time points ($P > 0.05$). Happiness demonstrated a main effect for time [F(3,51) = 3.161, $P = 0.032$, power = 0.699] but no main effect for group [F(1,17) = 1.069, $P = 0.321$, power = 0.164), and no group × time interaction [F(1,17) = 0.926, $P = 0.397$, power = 0.149]. Vigor demonstrated a main effect for time [F(3,51) = 3.161, $P \leq 0.001$, power = 0.987] but no main effect for group [F(1,17) = 1.043, $P = 0.321$, power = 0.161], and no group × time interaction [F(3,51) = 0.634, $P = 0.397$, power = 0.174]. Thus for these two categories of mood state (Happiness and Vigor), the 40% and 60% groups were combined into one group for all subsequent analysis. Happiness decreased at 60 min rest in hypoxia compared to baseline ($P = 0.006$), also happiness did not significantly improve during exercise compared to 60 min rest in hypoxia ($P = 0.602$). Vigor decreased at 60 min rest in hypoxia compared to baseline ($P = 0.002$) and vigor then significantly increased during exercise and recovery compared to 60 min rest in hypoxia ($P \leq 0.001$ and $P = 0.006$, respectively) (**Table II**).

Peripheral oxygen saturation demonstrated a main effect for time [F(3,51) = 320.659, $P \leq 0.001$, power = 1.000], but no main effect for group [F(1,17) = 0.002, $P = 0.969$, power = 0.050], and no group by time interaction [F(3,51) = 0.738, $P = 0.534$, power = 0.197].

Again, for all subsequent analysis the 40% and 60% groups were pooled into one group. Spo_2 significantly decreased after 60 min of hypoxia, compared to baseline ($P \leq 0.001$). During exercise Spo_2 further decreased compared to 60 min rest in hypoxia ($P \leq 0.001$). After cessation of exercise, Spo_2 was significantly increased compared to during the exercise bout ($P \leq 0.001$) (**Fig. 3**).

Regional cerebral oxygen saturation demonstrated a main effect for time [F(3,51) = 275.693, $P \leq 0.001$, power = 1.000], but no main effect for group [F(1,17) = 0.020, $P = 0.888$, power = 0.052], and no group × time interaction [F(3,51) = 1.388, $P = 0.257$, power = 0.347]. Regional cerebral oxygen saturation decreased after 60 min of hypoxia exposure, during exercise, and after recovery compared to baseline ($P \leq 0.001$ for all comparison). During exercise, rSo_2 decreased compared to 60 min rest ($P \leq 0.001$). During recovery, rSo_2 significantly increased compared to exercise ($P \leq 0.001$) (**Fig. 4**).

Table II. Mood State of Participants at Baseline, 60 min of Rest, Exercise and After Exercise in Hypoxia.

SUBSCALE	BASELINE	REST	EXERCISE	RECOVERY
Negative Mood				
Anger	2.1 ± 5.0	3.7 ± 6.3	3.0 ± 4.8	3.7 ± 6.1
Anxiety	4.3 ± 6.8	6.3 ± 8.1	3.5 ± 4.6	4.6 ± 5.6
Depression	2.6 ± 5.6	4.3 ± 7	3.1 ± 5	2.9 ± 4.5
Fatigue	16 ± 14.3	25.3 ± 23.3*	12.3 ± 10.2†	11.6 ± 13.5†
Restlessness	6.4 ± 7.9	8.8 ± 10.8	6.7 ± 8.2	7.4 ± 9.5
Positive Mood				
Happiness	60.4 ± 23.5	51.1 ± 21.1*	52.8 ± 19.6	56.3 ± 22.1
Vigor	43.4 ± 22.9	31.5 ± 18.3*	48.3 ± 20.7†	47.6 ± 23.3†

Participants responded to the question at each time point, "How much do the following words describe how you feel right now? *P<0.0.5, vs. at Baseline, †P<0.05, vs. at Rest in hypoxia, mean ± SD.

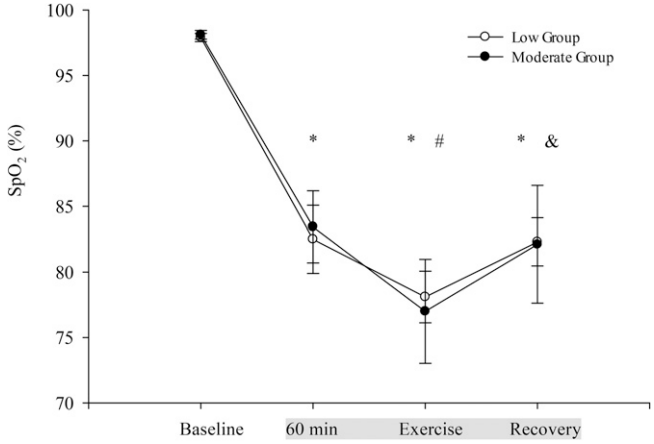

Fig. 3. Change in Spo_2 at baseline, 60 min, exercise and recovery in hypoxia. Shaded gray area occurred in hypoxic conditions. *$P < 0.05$ vs. Baseline; #$P < 0.05$ vs. Rest in hypoxia; &$P < 0.05$ vs. Exercise; mean ± SD.

Rate Perceived Exertion demonstrated a main effect for time [F(3,51) = 38.872, $P \leq 0.001$, power = 1.000] and main effect for group [F(1,17) = 5.769, $P = 0.028$, power = 0.620], and no group × time interaction [F(3,51) = 1.647, $P = 0.190$, power = 0.490]. RPE was significantly higher during exercise in the Moderate group compared to the Low group ($P = 0.05$). RPE was significantly increased during exercise in both the Low and Moderate group ($P \leq 0.001$ and $P = 0.009$, respectively) compared to 60 min rest in hypoxia. Several physiological parameters may have caused impairment and improvement in mood state at rest, exercise, and recovery. Exploratory correlation did not significantly relate rSo_2 and TMD across time points. Spo_2 and TMD also did not show a significant correlation ($P > 0.05$).

DISCUSSION

The purpose of this study was to quantify changes in mood state during rest, exercise, and recovery in hypoxia. We hypothesized that mood state would be negatively impacted during rest in

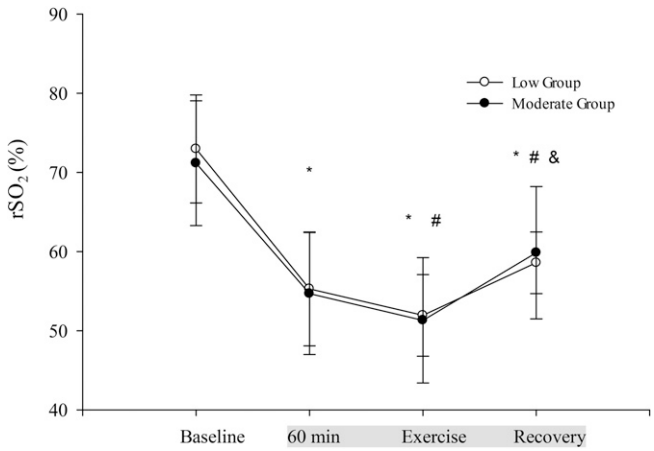

Fig. 4. Change in rSo_2 at baseline, 60 min, exercise and recovery in hypoxia. Shaded gray area occurred in hypoxic conditions. *$P < 0.05$ vs. Baseline; #$P < 0.05$ vs. Rest in hypoxia; &$P < 0.05$ vs. Exercise; mean ± SD.

hypoxia and that exercise would work to restore mood state to baseline levels. The data from this investigation support the hypothesis in that TMD increased in hypoxia at rest, but was reversed both during the exercises and after recovery from exercise. Cerebral oxygenation, however, cannot explain the changes in total mood state as rSo_2 decreased with hypoxia and further decreased during exercise.

Along with TMD, the individual categories of mood were also compared across time points. Across the four time points, we observed no change in anger, anxiety, depression, and restlessness. However, we observed an increase in fatigue and decrease in happiness and vigor at 60 min of rest in hypoxia compared to baseline. This is in agreement with a previous study in which impaired fatigue and vigor were experienced during acute exposure to hypoxia.[29] Furthermore, Shukitt and Banderet[30] reported that friendliness, clear thinking, dizziness, sleepiness, and unhappiness were affected at 1 and 4 h after ascent to 4300 m. A study by Li et al.[17] suggested that negative mood (tension, fatigue, and confusion) increased and the positive mood (vigor) decreased during exposure to hypoxia for 1 h.

Exercise in hypoxia significantly improved vigor and fatigue compared to the resting condition. Fatigue was significantly higher in the Moderate (intensity) group compared to the Low (intensity) group during exercise. Furthermore, RPE was higher in the 60% group during exercise compared to the 40% group. This result is to be expected during the higher exercise intensity as a greater stress induces more perceptual responses of muscle fatigue and physical stress.[27]

The interaction between mood state and cognition such as working memory and decision making is not a simple process. As mentioned earlier, a number of cognitive functions are affected by mood state.[20] The working memory is required in various cognitive functions in both psychology and neuroscience.[3] Neuroimaging investigations revealed that the prefrontal cortex in the brain plays an important role in both working memory and modulating the working memory by mood state.[31]

The average of both left and right rSo_2 as well as Spo_2 were significantly decreased at 60 min of rest in hypoxia compared to baseline and further decreased during exercise. This is in agreement with previous studies that rSo_2 decreased during low-intensity exercise in hypoxia.[16,34] This decrease in rSo_2 and Spo_2 with exercise and improved TMD indicates that although these two variables may be related in resting conditions across various levels of hypoxia, exercise dissociates these two variables. Above the ventilation threshold during exercise, reduction of cerebral oxygenation has been observed. The reduction of cerebral oxygenation could be explained by a decrease in partial pressure of end-tidal carbon dioxide ($P_{ET}CO_2$) which leads to cerebral vasoconstriction and decreased cerebral oxyhemoglobin (O_2Hb). This could induce the differences between Spo_2 and rSo_2.[15] There are likely some other stimulating factors involved with exercise that offset the decrease in cerebral oxygenation. Subsequent research in this area may consider additional physiological measurements (i.e., cerebrovascular function, sympathetic nerve activity) to better predict mood state for future experiments. Furthermore, it is possible that administration of high concentration oxygen (above 20.9%) improves mood state, since hyperoxia improves physical and cognitive performance at sea level.[21,23] Application and generalization of the present study need to be interpreted with caution since only healthy young men were recruited in this investigation. Generally, reported mood state in women seemed to easily transition to negative mood state when compared to men, and women generally reported significantly higher scores on tension, depression, fatigue, and confusion than men.[1,6] Lastly, nomobaric hypoxia was utilized instead of hypobaric hypoxia. Effects of hypobaric hypoxia and normobaric hypoxia on mood state have not been compared, though acute mountain sickness symptoms scores were higher in hypobaric hypoxia than normobaric hypoxia.[25]

In conclusion, caution should be taken when one attempts to predict health related symptoms and physical or psychological performance during hypoxia. The monitoring of mood state is important in optimizing performance and safety during work and recreational activities during normobaric hypoxia. Based on the data presented from the present investigation, single bouts of exercise between 40–60% $\dot{V}o_{2max}$ appear to be beneficial to partially improve impaired mood state (vigor and fatigue) in hypoxia. Further research is needed in this area to elucidate the mechanism underlying the improvement in mood state.

ACKNOWLEDGMENTS

Authors and affiliations: Yongsuk Seo, Ph.D., Curtis Fennell, M.S., Keith Burns, Ph.D., Brandon S. Pollock, Ph.D., John McDaniel, Ph.D., and Ellen L. Glickman, Ph.D., M.S.Ed., Exercise and Environmental Physiology Laboratory, and John Gunstad, Ph.D., M.A., Department of Psychology, Kent State University, Kent, OH.

REFERENCES

1. Abramson LY, Andrews DE. Cognitive models of depression: implications for sex differences in vulnerability to depression. Int J Ment Health. 1982; 11:77–94.
2. Amann M, Subudhi A, Foster C. Influence of testing protocol on ventilatory thresholds and cycling performance. Med Sci Sports Exerc. 2004; 36(4):613–622.
3. Baddeley A. Working memory: looking back and looking forward. Nat Rev Neurosci. 2003; 4(10):829–839.
4. Bonnet MH, Arand DL. Impact of activity and arousal upon spectral EEG parameters. Physiol Behav. 2001; 74(3):291–298.
5. Borg G. Perceived exertion as an indicator of somatic stress. Scand J Rehabil Med. 1970; 2:92–98.
6. Boyle GJ. Self-report measures of depression: some psychometric considerations. Br J Clin Psychol. 1985; 24(Pt. 1):45–59.
7. Buckworth J, Dishman RK, O'Connor PJ, Tomporowski PD, editors. Exercise Psychology, 2nd ed. Champaign (IL): Human Kinetics, Inc.; 2013.
8. Byrne A, Byrne DG. The effect of exercise on depression, anxiety and other mood states: a review. J Psychosom Res. 1993 Sep 37(6):565–574.
9. Crabbe JB, Dishman RK. Brain electrocortical activity during and after exercise: a quantitative synthesis. Psychophysiology. 2004; 41(4): 563–574.

10. de Aquino Lemos V, Antunes HK, dos Santos RV, Lira FS, Tufik S, de Mello MT. High altitude exposure impairs sleep patterns, mood, and cognitive functions. Psychophysiology. 2012; 49(9):1298–1306.
11. Ekkekakis P, Hall EE, VanLanduyt LM, Petruzzello SJ. Walking in (affective) circles: can short walks enhance affect? J Behav Med. 2000; 23(3):245–275.
12. Fulco CS, Rock PB, Trad L, Forte V, Jr., Cymerman A. Maximal cardiorespiratory responses to one- and two-legged cycling during acute and long-term exposure to 4300 meters altitude. Eur J Appl Physiol Occup Physiol. 1988; 57(6):761–766.
13. Hall EE, Ekkekakis P, Petruzzello SJ. Regional brain activity and strenuous exercise: predicting affective responses using EEG asymmetry. Biol Psychol. 2007; 75(2):194–200.
14. Hansen CJ, Stevens LC, Coast JR. Exercise duration and mood state: how much is enough to feel better? Health Psychol. 2001; 20(4):267–275.
15. Heine M, Subudhi AW, Roach RC. Effect of ventilation on cerebral oxygenation during exercise: insights from canonical correlation. Respir Physiol Neurobiol. 2009; 166(2):125–128.
16. Imray CHE, Myers SD, Pattinson KTS, Bradwell AR, Chan CW, et al. Effect of exercise on cerebral perfusion in humans at high altitude. J Appl Physiol. 2005; 99(2):699–706.
17. Li XY, Wu XY, Fu C, Shen XF, Wu YH, Wang T. Effects of acute mild and moderate hypoxia on human mood state. Space Med Med Eng (Beijing). 2000; 13(1):1–5.
18. McAuley E. Physical activity and psychosocial outcomes. In: Bouchard C, Shephard RJ, Stephens T, editors. Physical activity, fitness, and health:international proceedings and consensus statement. Champaign (IL): Human Kinetics; 1994:551–568.
19. McAuley E, Mihalko SL, Bane SM. Acute exercise and anxiety reduction: does the environment matter? J Sport Exerc Psychol. 1996; 18:408–419.
20. Mitchell RL, Phillips LH. The psychological, neurochemical and functional neuroanatomical mediators of the effects of positive and negative mood on executive functions. Neuropsychologia. 2007; 45(4):617–629.
21. Moss MC, Scholey AB, Wesnes K. Oxygen administration selectively enhances cognitive performance in healthy young adults: a placebo-controlled double-blind crossover study. Psychopharmacology (Berl). 1998; 138(1):27–33.
22. Perrey S. Non-invasive NIR spectroscopy of human brain function during exercise. Methods. 2008; 45(4):289–299.
23. Petersen SR, Dreger RW, Williams BE, McGarvey WJ. The effects of hyperoxia on performance during simulated firefighting work. Ergonomics. 2000; 43(2):210–222.
24. Petruzzello SJ, Landers DM, Hatfield BD, Kubitz KA, Salazar W. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise. Outcomes and mechanisms. Sports Med. 1991 Mar 11(3):143–182.
25. Roach RC, Loeppky JA, Icenogle MV. Acute mountain sickness: increased severity during simulated altitude compared with normobaric hypoxia. J Appl Physiol (1985). 1996; 81(5):1908–1910.
26. Ruby BC, Robergs RA. Gender differences in substrate utilisation during exercise. Sports Med. 1994; 17(6):393–410.
27. Scherr J, Wolfarth B, Christle JW, Pressler A, Wagenpfeil M, Halle M. Association between Borg's rating of perceived exertion and physiological measures of exercise intensity. Eur J Appl Physiol. 2013; 113:147–155.
28. Schneider S, Askew CD, Diehl J, Mierau A, Kleinert J, et al. EEG activity and mood in health orientated runners after different exercise intensities. Physiol Behav. 2009; 96(4-5):709–716.
29. Shukitt-Hale B, Banderet LE, Lieberman HR. Elevation-dependent symptom, mood, and performance changes produced by exposure to hypobaric hypoxia. Int J Aviat Psychol. 1998; 8(4):319–334.
30. Shukitt BL, Banderet LE. Mood states at 1600 and 4300 meters terrestrial altitude. Aviat Space Environ Med. 1988; 59(6):530–532.
31. Smith EE, Jonides J. Storage and executive processes in the frontal lobes. Science. 1999; 283(5408):1657–1661.
32. Steptoe A, Bolton J. The short-term influence of high and low intensity physical exercise on mood. Psychol Health. 1988; 2(2):91–106.
33. Steptoe A, Cox S. Acute effects of aerobic exercise on mood. Health Psychol. 1988; 7(4):329–340.
34. Subudhi AW, Dimmen AC, Roach RC. Effects of acute hypoxia on cerebral and muscle oxygenation during incremental exercise. J Appl Physiol (1985). 2007;103(1):177–183.
35. Swain RA, Berggren KL, Kerr AL, Patel A, Peplinski C, Sikorski AM. On aerobic exercise and behavioral and neural plasticity. Brain Sci. 2012; 2(4):709–744.
36. Terry P. The efficacy of mood state profiling with elite performers: a review and synthesis. TSP. 1995; 9(3):309–324.
37. Villringer A, Planck J, Hock C, Schleinkofer L, Dirnagl U. Near infrared spectroscopy (NIRS): a new tool to study hemodynamic changes during activation of brain function in human adults. Neurosci Lett. 1993; 154 (1-2):101–104.
38. Yeung RR. The acute effects of exercise on mood state. J Psychosom Res. 1996 Feb 40(2):123–141.
39. Young AJ, Cymerman A, Burse RL. The influence of cardiorespiratory fitness on the decrement in maximal aerobic power at high-altitude. Eur J Appl Physiol Occup Physiol. 1985; 54(1):12–15.