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Cover Page Footnote

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Response of Appetite and Appetite Regulating Hormones to Acute Hypoxia

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Abstract

AIM: To determine the acute response of appetite and appetite regulating hormones after exposure to simulated altitude. METHODS: Seven males and five females (height: 178.9 $\pm$ 2.3 cm; weight: 77.3 $\pm$ 7.2 kg; body fat: 18.4 $\pm$ 1.7%) participated in two, three-hour trials in a hypoxic (5000 m) and normoxic (350 m) environment. Blood samples were collected prior to and immediately following three hours of exposure for the measurement of leptin, adiponectin, and acylated ghrelin. Appetite, acute mountain sickness, heart rate, blood oxygenation, tissue oxygenation, respiration rate, and whole body gases were also measured. RESULTS: Leptin was not different between hypoxic (5.8 $\pm$ 1.8 ng ml^{-1}) and normoxic trials (6.2 $\pm$ 2.0 ng ml^{-1}; $p = 0.603$). Adiponectin was not different between hypoxic (9.0 $\pm$ 0.2 µg ml^{-1}) and normoxic trials (8.4 $\pm$ 0.7 µg ml^{-1}; $p = 0.216$). Acylated ghrelin was not different between hypoxic (15.0 $\pm$ 3.8 pg ml^{-1}) and normoxic trials (16.3 $\pm$ 4.6 pg ml^{-1}; $p = 0.285$). Appetite scores were not different between trials ($p > 0.05$) with the exception of fullness which was greater in the hypoxic condition ($p = 0.027$). Heart rate and symptoms of acute mountain sickness were higher while blood and tissue oxygenation were lower in the hypoxic trial ($p < 0.05$). No differences were noted in other metabolic parameters ($p > 0.05$). CONCLUSION: Appetite and appetite regulating hormones are not affected by three hours of hypoxic exposure, and thus some of these negative consequences of hypoxic exposure may not be evident with short exposure times.

Keywords: leptin, adiponectin, ghrelin, altitude, oxygenation, extreme environments

Introduction

There is a decline in body mass in response to extended exposure to altitude or hypoxia (Benso et al., 2007; Hamad & Travis, 2006; Rose et al., 1988; Westerterp & Kayser, 2006; Westerterp-Plantenga et al., 1999; Zaccaria, Ermolao, Bonvicini, Travain, & Varnier, 2004). When ascending to a higher altitude, the barometric pressure exerted on oxygen is decreased causing a decline in arterial oxygen saturation (Mazzeo, 2008). When the body cannot deliver an adequate supply of oxygenated blood to target tissues due to high altitude, this causes a state of hypoxia. Many factors may contribute to reductions in body mass at altitude, including appetite, exercise (Westerterp & Kayser, 2006), altered intestinal function, and overall disruption of energy balance (Hamad & Travis, 2006; Tschöp & Morrison, 2001) resulting in changes to both fat mass and muscle mass (Tschöp & Morrison, 2001).

Weight loss has been observed after ascending to a simulated altitude of 8848 m, where a reduction in energy intake was a key component for the loss of body mass over 31 days (Westerterp-Plantenga et al., 1999). Similar results were found during a 40-day stay in a hypobaric chamber simulating the altitude of Mount Everest. Subjects consumed fewer calories, lost weight, and lost body fat even when palatable food was provided ad libitum throughout the confinement (Rose et al., 1988). Therefore, hypoxic conditions independent of food availability may be at least partially responsible for the observed weight loss (Rose et al., 1988; Westerterp-Plantenga et al., 1999). Furthermore, the reduction in energy intake may be attributed to a loss of appetite. Indeed, subjects who ascend to high altitude (8848 m) note changes in the following aspects of an appetite profile: "deviations in hunger, desire to eat, estimation of how much one could eat, satiety, and fullness" (Westerterp-Plantenga et al., 1999). Acute mountain sickness (AMS) may also play a role in loss of appetite due to the associated nausea (Westerterp-Plantenga et al., 1999). However, it is unknown whether the loss of appetite occurs with hypoxia alone or if other conditions (exercise, temperature, food availability, etc.) inherent to these study designs are the major factors. Further monitoring in a controlled laboratory environment is needed to determine if hypoxia independently drives the loss of appetite. Several hormones regulate appetite and changes in these hormones with exposure to hypoxia may explain changes in appetite and body mass at altitude.

Leptin, adiponectin, and ghrelin work to regulate appetite and maintain energy homeostasis through receptors in the hypothalamus and pituitary gland. Increased levels of leptin work to keep one satiated after eating (Zhang et al., 1994), while decreased levels of adiponectin and ghrelin signal the hypothalamus that food intake is not necessary and energy

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balance has been met (Cummings, 2006; Kadowaki, Yamauchi, & Kubota, 2008). Current research is unclear in determining the effects of altitude exposure on leptin, adiponectin, and ghrelin plasma concentrations. These hormones appear to respond quickly to metabolic alterations. Leptin may decrease as early as 60 minutes following exposure to a simulated altitude of 4300 m (Kelly et al., 2010). During short-term exposures to hypoxia, ghrelin and acylated ghrelin levels appear to decrease after 48 hours of free-living conditions (Shukla et al., 2005) and after 7 hours of controlled environmental chamber conditions (Shukla et al., 2005; Wasse, Sunderland, King, Batterham, & Stensel, 2012). During extended exposures to altitude, leptin concentrations may decrease, increase, or remain unchanged during acute and chronic exposures to altitude (Barnholt et al., 2006; Debevec, Simpson, Macdonald, Eiken, & Mekjavic, 2014; Shukla et al., 2005; Tschop et al., 2000; Zaccaria et al., 2004). Few studies have investigated adiponectin's acute response to altitude; however, there appears to be no change in adiponectin levels after chronic exposure to free-living altitude (4300 m) conditions (Barnholt et al., 2006).

The limited research describing the acute response (within a few hours) of these hormones coupled with a lack of control in many field-based investigations warrant further study. Therefore, the purpose of this study is to determine the acute response of appetite and appetite regulating hormones after exposure to simulated altitude in well-controlled resting conditions. It is important to investigate the connection between appetite regulation and altitude exposure to understand if weight loss at increased altitude is attributed to appetite suppression or other factors such as temperature and exercise that tend to accompany exposure to hypoxia. This knowledge may provide information on possible strategies that could be incorporated early to help preserve body mass for those mountaineering or a possible novel treatment for overweight or obese individuals with increased appetite.

Materials and Methods

Participants

Twelve recreationally active (seven males and five females) subjects were recruited for this study. Recreationally active males and females were defined as anyone self-reporting structured exercise 2–5 times weekly. After describing the procedures and potential risks to the participants they were asked to sign an Institutional Review Board approved informed consent before participating.

Study Design

General overview

Subjects were exposed to normobaric hypoxia simulating 5000 m and ambient oxygen content (350 m) for three hours. The altitude of 5000 m was chosen as it is slightly higher than what has been used in previous studies that have demonstrated an effect on appetite regulating hormones and represents the higher limit of the experimental chamber capacity. Subjects were seated and rested during the three-hour duration. Trials were separated by no more than five days. Blood samples (4 mL) were collected from the antecubital vein before and after exposure and analyzed for the acute response in the key appetite regulating hormones, leptin, adiponectin, and acylated ghrelin. Perceived appetite and symptoms of AMS were also assessed. Heart rate, blood oxygenation, respiration rate, tissue oxygenation, and whole body gas exchange were analyzed throughout the trials.

Initial visit

Height, weight, and percent body fat were collected for descriptive purposes. Percent body fat was measured using an electronic load cell-based hydrostatic weighing system (Exertech, Dresbach, MN). Each subject performed six to ten trials, with the highest three underwater weights averaged and recorded. Body density was calculated from the underwater weight and was converted to percent body fat using the Siri equation (Siri, 1961). Residual lung and gastrointestinal volume was estimated and corrected for using an established prediction equation (Thomas & Etheridge, 1980).

Experimental trials

Experimental trials were conducted in a randomized and counterbalanced order. The experimental trials for women were completed between days 3 and 8 of the follicular phase of the menstrual cycle (Kelly et al., 2010) to control for fluctuations in leptin across the menstrual cycle (Lecke, Morsch, & Spritzer, 2011). The menstrual cycle has no effect on adiponectin or ghrelin concentrations (Dafopoulos, Sourlas, Kallitsaris, Pournaras, & Messinis, 2009). Thus, trials were separated by approximately five days. Men's experimental trials were also separated by five days for consistency. Subjects were required to record a 24-hour food log prior to the first experimental trial and repeat the 24-hour log before the second experimental trial. Subjects were not allowed to exercise 24 hours prior to the experimental trials. The day prior to the experimental trials, participants were provided with a small standardized breakfast and were instructed to eat the breakfast two hours before arrival atthe laboratory. The standardized meal consisted of a granola bar and orange juice (375 kcal, 75 g carbohydrates, 7 g fat, 6 g protein). All trials were conducted in the morning to account for diurnal variation in hormones (Bray & Young, 2007; Kotidis et al., 2006; Shetty, Kusminski, & Scherer, 2009).

The experimental trials consisted of two separate, three-hour, resting trials. One trial was conducted in a normobaric hypoxic condition (12.03% oxygen fraction simulating

5000 m) and the other in an ambient control condition (350 m). The trials were conducted in an environmental chamber (Darwin, St Louis, MO) that controlled for temperature (22° C), humidity (40%) and environmental oxygen concentration simulating altitude (Altitude Control Technologies, Lafayette, CO). Participants remained in a seated position for the duration of the trials, except when completing a clinical assessment of AMS (described in detail below).

Blood collection

Blood samples were collected immediately prior to entering the environmental chamber and immediately following the three hours of altitude exposure. Approximately 4 mL of blood was collected using venipuncture technique from the antecubital vein into an ethylenediaminetetraacetic acid-coated vacutainer (Greiner Bio-One, Monroe, NC). Within three minutes of the collection of blood the vacutainer was treated with 40 μL of HALT protease inhibitor (Thermo Fisher, Wilmington, DE) and inverted several times. Whole blood was transferred to two capillary tubes and centrifuged for 5 minutes for analysis of hematocrit in duplicate (ZIPOcrit, LW Scientific, Lawrenceville, GA). A separate whole blood sample was transferred to a microcuvette and analyzed for hemoglobin (HemoCue, Cypress, CA). The values of hematocrit and hemoglobin were used to correct for plasma volume shifts that may occur with altitude exposure (Miles, Bransford, & Horvath, 1981). The remaining blood samples were then centrifuged for 10 minutes at 3500 rpm at 4° C and the plasma was aliquoted into 200 μL volumes. The plasma samples were then frozen at −30° C for later analysis of leptin, adiponectin, and acylated ghrelin.

Circulating leptin, ghrelin, and adiponectin

Concentrations of the hormones were determined in triplicate using enzyme-linked immunosorbent assay (ELISA) kits. Manufacturer's protocols were followed for each leptin, adiponectin, and acylated ghrelin ELISA kit (Bertin Pharma, Montigny-le-Bretonneux, France). Plasma samples were diluted 1:3, 1:30, and 1:2 for leptin, adiponectin, and acylated ghrelin, respectively.

Appetite and mountain sickness

The Lake Louise Self-Report Questionnaire addressed symptoms associated with AMS—headache, appetite and nausea, fatigue, and lightheadedness (Roach, Bartsch, Hackett, & Oelz, 1993). Along with subjective feelings of the subject, clinical assessments related to AMS were performed. Subjects were asked to perform a physical task of tandem walking to assess ataxia or loss of full control of bodily movements. Changes in mental status or signs of peripheral edema were recorded. A combined score > 5 was defined as clinically significant AMS (Bartsch et al., 1993).

A 100 mm Visual Analogue Scale was used to assess the subjects' perceived appetite. The following questions were used for the assessment as recommended by Blundell et al. (2010): "how hungry are you?", "how full are you?", "how satiated are you?", "how strong is your desire to eat?" and "how much do you think you could eat right now?" A Composite Satiety Score (CSS) was also used to assess appetite. The CSS was calculated as follows: CSS = (fullness + (100 − desire) + (100 − hunger) + (100 − perceived food consumption (PFC)))/4 (Debevec et al., 2014).

Heart rate, oxygen saturation, and metabolism

Heart rate (Polar V800; Polar, Lake Success, NY), blood oxygenation ($WristOx_2$; Nonin Medical, Inc., Plymouth, MN), and tissue oxygenation (MOXY, Hutchinson, MN) were monitored continuously throughout the trials. The pulse oximeter was worn on each subject's index finger. Tissue oxygenation was measured using a near-infrared spectroscopy device that was adhered to a subject's skin over the vastus lateralis. This non-invasive device allows for continuous monitoring of tissue oxygenation throughout the trials. Heart rate, blood, and tissue oxygenation data were averaged for 1, 2, and 3 hours. Metabolic gases and respiration rate (TrueOne Metabolic System; ParvoMedics, Sandy, Utah) were measured by requiring subjects to breathe through a mouthpiece and wear a nose clip, while expired air was collected and analyzed. Measurements were collected in five-minute increments at 55-, 115-, and 175-minute time points. Carbohydrate oxidation (%) and fat oxidation (%) were calculated by a TrueOne Metabolic System using equations derived from Peronnet and Massicote (1991).

Statistical Analysis

A repeated measures two-way ANOVA (2 × 2, time by trial) was used to determine statistical significance. After running the ANOVA, if significance was found a Fisher's protected least significant difference *post hoc* test was used to determine where significance occurred. Significance was set at 5% error rate ($p < 0.05$). Statistical Package for Social Sciences software (SPSS 23.0, Chicago, IL) was used to analyze data.

Results

Participants

Twelve participants (24.6 ± 0.7 years, body mass of 77.3 ± 7.2 kg, 178.9 ± 2.3 cm, 18.4 ± 1.7%), namely seven males (25.1 ± 1.1 years, 83.0 ± 8.1 kg, 179.3 ± 2.0 cm, 15.3 ± 2.1%) and five females (23.8 ± 0.4 years, 69.3 ± 8.2 kg, 178.4 ± 5.2 cm, 22.6 ± 1.1%), completed the study. Eleven subjects' blood was analyzed due to

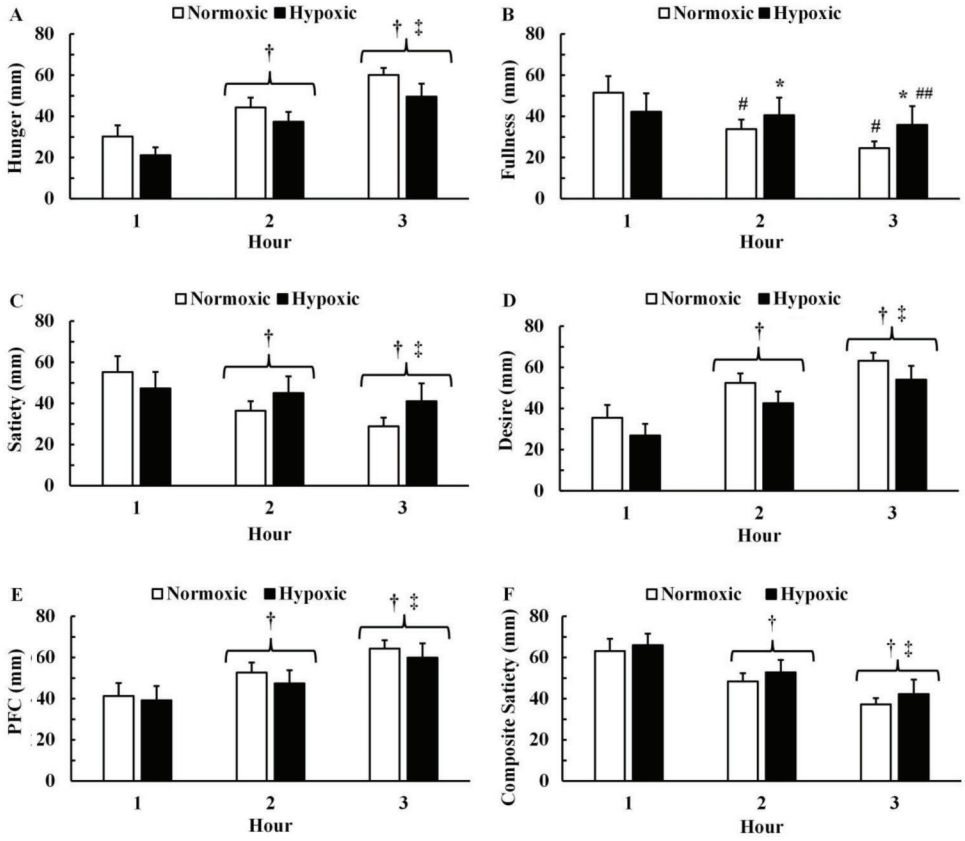

Figure 1. (n = 12) Hunger scores (A), fullness scores (B), satiety scores (C), desire scores (D), PFC scores (E), and CSS (F); $^{*}p < 0.05$ from normoxic, $^{\dagger}p < 0.05$ from 1 h, $^{\ddagger}p < 0.05$ from 2 h, $^{\#}p < 0.05$ from 1 h normoxic, $^{\#\#}p < 0.05$ from 2 h hypoxic. Data are mean ± SE.

inability to obtain post-experimental trial blood from one female subject.

Appetite

No differences occurred between trials for the following appetite scores: hunger (p = 0.094), satiety (p = 0.507), desire (p = 0.139), or perceived food consumption (p = 0.390). However, fullness was higher in the hypoxic condition compared to the normoxic condition (p = 0.027). Hunger, desire, and perceived food consumption increased from 1 h to 2 h ($p < 0.001$, p = 0.001, p = 0.009, respectively) and continued to increase from 2 h to 3 h (p = 0.001, p = 0.001, $p < 0.001$, respectively). Satiety decreased from 1 h to 2 h (p = 0.036) and continued to decrease from 2 h to 3 h (p = 0.016), regardless of trial. Fullness decreased from 1 h to 3 h (p = 0.005) and from 2 h to 3 h (p = 0.003), regardless of trial. CSS decreased from 1 h to 2 h (p = 0.002) and then continued to decrease from 2 h to 3 h (p = 0.001; Figure 1), regardless of trial.

Appetite Regulating Hormone Plasma Concentrations

Based on hemoglobin and hematocrit measures, plasma volume increased 1.9 ± 1.5% for the hypoxic trials and 3.3 ± 1.4% for the normoxic trials. Hormone concentrations were adjusted to account for this change. There were no differences between the hypoxic and normoxic trials in the appetite regulating hormones leptin (p = 0.603), adiponectin (p = 0.216), or acylated ghrelin (p = 0.285). No differences were observed pre- to post-trial for leptin (p = 0.060), adiponectin (p = 0.866), or acylated ghrelin (p = 0.481; Figure 2).

Acute Mountain Sickness

AMS scores were higher in the hypoxic condition (0.66 ± 0.91 points) than in the normoxic condition (0.07 ± 0.18 points; p = 0.021). No single subject had a clinically significant score of AMS, defined as having a score > 5.

Blood and Tissue Oxygenation

Blood oxygenation was lower in the hypoxic condition compared to the normoxic condition (p = 0.001) regardless of time (p = 0.196). Tissue oxygenation was lower in the hypoxic condition compared to the normoxic condition (p = 0.011). Tissue oxygenation increased from 1 h to 2 h (p = 0.042) and then continued to increase from 2 h to 3 h (p = 0.006; Table 1), regardless of trial.

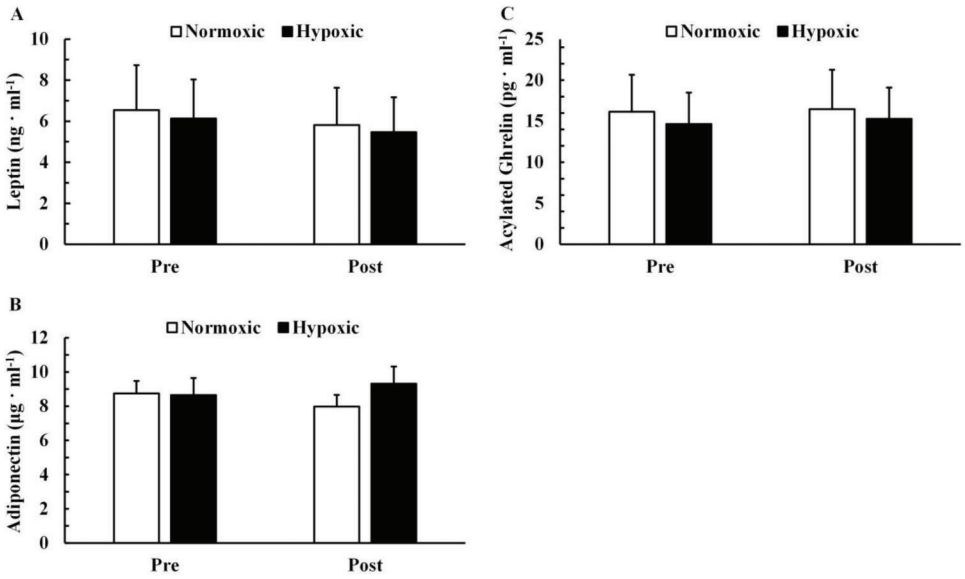

Figure 2. (n = 11) Plasma leptin concentrations (A), plasma adiponectin concentrations (B), and plasma acylated ghrelin concentrations (C). Data are mean ± SE.

Table 1
(n = 12) Study parameters measured during experimental trials.

	Hour 1	Hour 2	Hour 3	Grand mean
Heart rate (bpm)				
Normoxic	75±3	68±3†	67±3	70±2
Hypoxic	80±3	76±3†	76±3	78±1*
Blood oxygenation (%)				
Normoxic	97±0.4	98±0.5	98±0.3	98±0.3
Hypoxic	80±1	79±1	81±1	80±0.5*
Tissue oxygenation (%)				
Normoxic	59±3	63±3†	67±3†‡	63±2
Hypoxic	55±3	55±3†	58±4†‡	56±1*
VO_2 (L min^{-1})				
Normoxic	0.32±0.02	0.32±0.02	0.33±0.02	0.32±0.01
Hypoxic	0.32±0.03	0.36±0.05	0.36±0.05	0.35±0.01
Respiratory rate (bpm)				
Normoxic	15.4±0.7	15.5±0.9	15.4±0.7	15.4±0.0
Hypoxic	14.6±0.9	15.1±1	14.8±0.8	14.8±0.2
Carbohydrates (%)				
Normoxic	42±8	35±10	17±10†‡	31±7
Hypoxic	57±15	48±19	41±19†‡	49±4
Fat (%)				
Normoxic	59±8	66±10	83±10†‡	69±7
Hypoxic	44±15	53±19	59±19†‡	52±4

Note. Data are mean ± SE.
*$p < 0.05$ from normoxic; †$p < 0.05$ from 1 h; ‡$p < 0.05$ from 2 h.

Oxygen Utilization and Substrate Use

No differences occurred between hypoxic and normoxic trials in absolute VO_2 (p = 0.606), respiratory rate (p = 0.318), % CHO utilization (p = 0.281), or % fat utilization (p = 0.280). However, % CHO oxidation decreased from 1 h to 3 h (p = 0.004) and 2 h to 3 h (p = 0.025) while fat oxidation increased from 1 h to 3 h (p = 0.004) and from 2 h to 3 h (p = 0.025; Table 1), regardless of trial.

Heart Rate

Heart rate was higher in the hypoxic condition compared to the normoxic condition (p = 0.001). Heart rate decreased from 1 h to 2 h (p = 0.004), but remained similar between 2 h and 3 h (p = 0.464; Table 1), regardless of trial.

Discussion

This study aimed to determine the effect of acute hypoxic exposure on appetite and appetite regulating hormones. Although research is limited with acute exposure to hypoxia, it was hypothesized that leptin would be higher, and adiponectin and acylated ghrelin would be lower, resulting in lower appetite in hypoxia compared to normoxia. The main findings of this study were that there were no differences in appetite regulating hormones between hypoxia and normoxia in our subjects. Only the feeling of fullness was marginally lower in hypoxia compared to normoxia, but other aspects of appetite were not different between hypoxia and normoxia.

Previous research has focused primarily on extended stays in a hypoxic environment with little information regarding the acute response. The current study is one of very few to investigate the effects of acute exposure to hypoxia on appetite and appetite regulating hormones. When this body of literature is taken collectively, differences in appetite regulating hormones associated with

hypoxia may be related to longer durations than what was used in the current study. Reductions in body mass were observed along with increased leptin and decreased ghrelin resulting in a loss of appetite after seven days at 4300 m in free-living conditions (Shukla et al., 2005). Few human studies have investigated adiponectin in this context, but during free-living altitude (4300 m) exposure for three weeks, no changes in adiponectin levels were observed (Barnholt et al., 2006). Shorter exposures to hypoxia may also elicit changes. Leptin concentrations increased after 20 hours of being transported to an elevation of 4559 m. This increase was also accompanied by a loss of appetite (Tschop et al., 2000). However, appetite and hormones do not always have a strong agreement (Wasse et al., 2012). In contrast to that study and the current study, an acute stay in a well-controlled normobaric hypoxic chamber (simulating 4300 m) found leptin concentrations to decrease as opposed to increase within 60 minutes of hypoxic exposure after carbohydrate supplementation (Kelly et al., 2010). After seven hours in a normobaric hypoxic chamber, acylated ghrelin was found to decrease, potentially indicating the subjects were not as hungry (Wasse et al., 2012). The current study did not observe any differences in leptin, adiponectin, or acylated ghrelin. Thus, three hours of exposure to hypoxia may not be sufficient time to elicit meaningful changes in appetite or appetite regulating hormones. Furthermore, appetite regulation is likely much more complex than regulation by these three hormones alone (Kalra et al., 1999; Suzuki, Simpson, Minnion, Shillito, & Bloom, 2010).

An alternative interpretation of our results is that these hormones do not respond within three hours despite a sufficient stimulus. It is possible that these hormones did respond to the hypoxic stimuli but are not observable until some time in the future. This study did not look at these response kinetics, but these hormones do respond quickly with other stimuli. High-intensity exercise with durations as short as 20 minutes (Jürimäe, Purge, & Jürimäe, 2005), one hour (Broom, Stensel, Bishop, Burns, & Miyashita, 2007), and two hours (Duclos, Corcuff, Ruffie, Roger, & Manier, 1999) have shown changes in these hormone concentrations compared to resting values demonstrating the rapidity of response that these hormones have. Exercise results in shifts in substrate use from fat oxidation to more carbohydrate oxidation. Similar to exercise, hypoxic exposure can cause a similar shift in metabolism. Specifically, leptin is affected by these shifts in metabolism (Tuominen et al., 1997). When subjects were fed carbohydrate before acute exposure to hypoxia, not only did substrate use shift but leptin was also altered compared to a normoxic control (Kelly et al., 2010). Indeed, further research supports the regulation of blood glucose by leptin. Refer to the review by Morton and Schwartz (2011) for a detailed discussion on the role of leptin in glucose metabolism. Thus, an alteration in substrate use, metabolism, or blood glucose may be a result of circulating leptin as opposed to an outcome. The current study did not see a significant difference in leptin and therefore explain why substrate use, oxygen utilization, or respiratory rate were not altered. Additionally, high blood glucose by intravenous glucose infusion does not appear to affect appetite-related outcomes (Schultes et al., 2016). The current study did not directly measure blood glucose. However, the lack of difference in substrate use between trials would suggest that blood glucose was also not different. Perhaps longer or more extreme hypoxia would cause a change in leptin and be accompanied by a shift in metabolism or alteration in blood glucose.

An additional difference besides time course and metabolic alterations between the current study and those that found differences in leptin and ghrelin with hypoxic exposure is that many previous studies have been conducted in free-living conditions such as mountaineering expositions that do not allow specific control. The current study was conducted in a well-controlled normobaric hypoxia chamber. There has been some debate on the potential differences in physiological responses between normobaric and hypobaric hypoxia exposure (Coppel, Hennis, Gilbert-Kawai, & Grocott, 2015). AMS scores tend to be higher in hypobaric hypoxia compared to normobaric hypoxia (Loeppky et al., 2005; Roach, Loeppky, & Icenogle, 1996). Ventilation is also lower in hypobaric compared to normobaric hypoxia (Roach et al., 1996). We did not observe measurable AMS or alterations in respiratory rate, which may be related to incorporating a normobaric vs. hypobaric intervention. Higher AMS during hypobaric hypoxia may in part lead to a decrease in appetite during exposure. Aspects of nausea associated with hypobaric hypoxia may help explain decreased appetite and weight loss at extended stays in hypoxia. Despite these potential differences, we did observe a hypoxic response, decreased blood and tissue oxygenation, and increased heart rate. Research using mountaineering or free-living designs is also unable to control for factors such as temperature, exercise, and diet, which may influence hormone concentrations. Cold exposure and exercise are associated with decreases in leptin (Peino et al., 2000; Sierra-Johnson, Romero-Corral, Somers, & Johnson, 2008). Aerobic activity is associated with decreases in acylated ghrelin (Broom, Batterham, King, & Stensel, 2009). Longer term exercise training programs are related to changes in body mass, which may be responsible for deceases in leptin and increases in adiponectin (Fatouros et al., 2005). Not controlling for diet in free-living designs may also result in changes in hormones that are not specifically related to hypoxia (Sierra-Johnson et al., 2008). The current study controlled for temperature and exercise by conducting the experimental trials in an environmental chamber where subjects were seated at room temperature (22°C) throughout the three hours.

Conclusion

In conclusion, the current study indicates that appetite and appetite regulating hormones are not affected by three hours of hypoxic exposure. This finding has important implications for individuals who may be exposed to short periods of hypoxia. During these short periods, the negative consequences associated with hypoxia such as a loss of appetite and body mass do not appear to be evident. Longer durations or more intense hypoxia may be required in order to see differences in appetite, appetite regulating hormones, and overall energy imbalances—which may lead to decreases in body mass (Hamad & Travis, 2006; Tschöp & Morrison, 2001; Westerterp & Kayser, 2006). When the current study is interpreted along with previous research it contributes to the understanding of the time course of responses associated with hypoxia and may be considered in interventions designed to counter the negative outcomes associated with hypoxia.

Disclosure of Potential Conflicts of Interest

The abstract of this paper was presented at the Research and Creative Activities Fair as a poster presentation with interim findings. The poster's abstract was published in "Poster Abstracts" on the University of Nebraska at Omaha's library's online digital commons: digitalcommons.unomaha.edu/cgi/viewcontent.cgi?article=2075&context=srcaf.

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