

# Obesity: Associations with Acute Mountain Sickness

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**Background:** Although few retrospective studies of high altitude have reported that obesity might be associated with the development of acute mountain sickness (AMS), this association has not been studied prospectively.

**Objective:** To determine whether obesity is associated with the development of AMS.

**Design:** Obese and nonobese men were compared at a simulated altitude of 3658 m (12 000 ft).

**Setting:** 24 hours in a hypobaric environmental chamber.

**Participants:** 9 obese and 10 nonobese men.

**Measurements:** Percentage body fat (by hydrostatic weighing),

Lake Louise AMS score, and  $SaO_2$  level (by pulse oximetry) were measured.

**Results:** Average AMS scores increased more rapidly with time spent at simulated high altitudes for obese men than for nonobese men ( $P < 0.001$ ). The response of  $SaO_2$  with exposure differed between nonobese and obese men. After 24 hours in the altitude chamber, seven obese men (78%) and four nonobese men (40%) had AMS scores of 4 or more.

**Conclusion:** Obesity seems to be associated with the development of AMS, which may be partly related to greater nocturnal desaturation with altitude exposure.

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**R**apid ascent from low to high altitude (above 2500 m or 8200 ft) often causes acute mountain sickness (AMS), a syndrome characterized by headache and other systemic symptoms, such as nausea, lassitude, and difficulty sleeping. The prevalence and severity of AMS depend on the speed of ascent, the altitude attained, preacclimatization, age, sex, exertion levels while at altitude, and the ventilatory response to acute hypoxia (1, 2).

Few retrospective field studies of high altitude have reported that obesity, as evidenced by body mass index (BMI), might be associated with the development of AMS (3–7). However, this association has not been studied prospectively under controlled conditions at reasonably accessible altitudes or in individuals with mild to moderate obesity. We sought to determine whether obese individuals are more likely to develop AMS than nonobese individuals during decompression to a simulated altitude of 3658 m. We hypothesized that obese individuals were more susceptible to develop AMS than nonobese individuals during exposure to high altitude–induced hypobaric hypoxia.

## METHODS

### Participants

Volunteers were recruited through local advertisements and were selected for participation on the basis of percentage body fat. “Nonobese” was defined as percentage body fat less than 25%. “Obese” was defined as a BMI of 30 kg/m<sup>2</sup> or greater and percentage body fat of 30% or greater. None of the participants had a history of cardiovascular or respiratory abnormalities. No participant was taking long-term medications. All participants were non-smokers.

Nine obese men (mean age [ $\pm$ SD], 35  $\pm$  8 years) and 10 nonobese men (mean age [ $\pm$ SD], 34  $\pm$  8 years) were studied. All participants resided at sea level (100 m) in

Dallas, Texas. One obese and three nonobese participants previously had mild AMS. One obese participant was exposed to a 2500-m altitude 4 days before this study; no other participant was exposed to a 1500-m or higher altitude before participating in the study. Each participant received both written and verbal explanations of the experiment before giving written consent. The Institutional Review Board of the University of Texas Southwestern Medical Center and Presbyterian Hospital of Dallas approved this study.

### Study Protocol

The study was conducted in a large (40 ft long by 9 ft diameter) multiplace (room for >1 person) decompression chamber at the Institute for Exercise and Environmental Medicine in Dallas. The barometric pressure was held at 483 mm Hg, which is equivalent to an altitude of 3658 m (12 000 ft). The temperature (25  $\pm$  0.5 °C), humidity (28%  $\pm$  1%), and concentration of CO<sub>2</sub> (0.07%  $\pm$  0.02%) in the chamber were monitored continuously by trained medical staff. Four participants at a time were studied in the chamber during the 24 hours of exposure (Figure 3).

### Assessment of AMS

According to guidelines established by the Lake Louise AMS consensus report (8), each participant completed an AMS self-report questionnaire at sea level (before decompression) and during decompression to 483 mm Hg at 6 hours, 12 hours, and 24 hours. The questionnaire included items for symptoms of headache, gastrointestinal symptoms, fatigue or weakness, dizziness or lightheadedness, and difficulty sleeping. Each symptom was graded on a scale from 0 to 3, with 0 representing no symptoms; 1, mild symptoms; 2, moderate symptoms; and 3, severe symptoms. A score of 15 was the maximum score possible. A self-score of 4 or more was an indication of AMS (8).

**Context**

A few small retrospective studies show associations between obesity and acute mountain sickness.

**Contribution**

This 24-hour study involving 9 obese and 10 nonobese men was conducted in a decompression chamber that simulated a rapid ascent to an altitude of 3658 m (12 000 ft). Obese men more often developed symptoms of mountain sickness and had lower nocturnal oxygen saturation values than did nonobese men.

**Cautions**

Although this elegant, short experiment suggests that obese men were more susceptible to acute mountain sickness, the study involved few people, simulated a steady rate of ascent, and did not simulate physical activity with altitude exposure.

—The Editors

This scoring system has been validated against the U.S. Army Environmental Symptoms Questionnaire, demonstrating similar sensitivity and specificity (9).

**Measurements of SaO<sub>2</sub>**

Daytime SaO<sub>2</sub> was measured by pulse oximetry (Ohmeda 3700 Pulse Oximeter, Datex-Ohmeda, Boulder, Colorado) at sea level and at 6 hours and 24 hours of simulated altitude. Nocturnal SaO<sub>2</sub> in each participant was continuously recorded in the chamber from 10:30 p.m. to 6:30 a.m. The mean nocturnal SaO<sub>2</sub> was calculated from values obtained every 30 minutes. Heart rate was measured at sea level and at altitude during the daytime and during sleep.

**Other Measurements**

Body composition was determined by hydrostatic weighing, and percentage body fat, fat mass, and lean mass were calculated. At sea level, all participants underwent standard spirometry (measuring lung volumes, maximal flow-volume loop, and maximal voluntary ventilation) and diffusing capacity of the lung in a whole-body plethysmograph (Model 6200, SensorMedics, Yorba Linda, California). Pulmonary function testing was performed according to the guidelines of the American Thoracic Society.

**Statistical Analysis**

Data are expressed as means ( $\pm$ SD). The parameters of AMS score and SaO<sub>2</sub> were analyzed by a two-way analysis of variance (ANOVA) using SAS software, release 8.02 (SAS Institute, Inc., Cary, North Carolina), with repeated measures on one factor (altitude-time) and between-participant comparisons for the other factor (group, nonobese and obese). Comparisons were considered significant when the *P* value was less than 0.05.

**Role of the Funding Sources**

The funding sources had no role in the design, conduct, or reporting of the study or in the decision to submit the manuscript for publication.

**RESULTS****Participants**

The Table shows general characteristics of the participants. One participant in the obese group was removed from the chamber after 10 hours because of severe headache, nausea, and dizziness (AMS score, 8). As a result, this participant was not included in further analyses.

**AMS Scores**

There was a significant interaction between altitude-time and group ( $P < 0.001$ ) as a result of the two-way ANOVA. This indicated that the increase in AMS scores with altitude exposure was more pronounced in the obese participants (Figure 1). Overall, after 24 hours in the chamber, seven obese participants and four nonobese participants had an AMS score of 4 or more. The frequency of AMS symptoms at 24 hours in 18 participants was as follows: headache, 89%; gastrointestinal upset, 36%; fatigue and weakness, 36%; dizziness, 15%; and difficulty sleeping, 75%.

**SaO<sub>2</sub>**

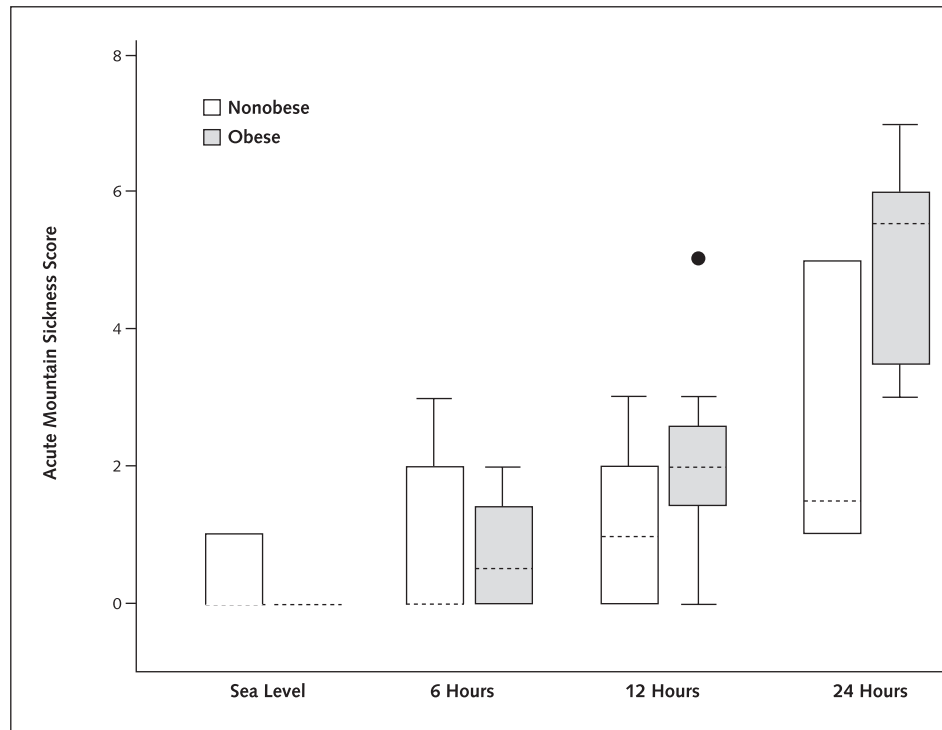
There was also a significant interaction between altitude-time and group ( $P < 0.001$ ) for SaO<sub>2</sub> as a result of the two-way ANOVA (Figure 2). This indicated that the decrease in SaO<sub>2</sub> with altitude exposure differed between the two groups.

**Table. General Characteristics and Pulmonary Function of Participants at Baseline\***

Characteristic	Nonobese Participants (n = 10)	Obese Participants (n = 9)
Race or ethnicity, n		
White	8	7
African American	2	1
Asian	0	1
Age, y	34.9 $\pm$ 7.8	34.7 $\pm$ 6.7
Height, cm	181.2 $\pm$ 3.7	179.8 $\pm$ 3.2
Weight, kg	82.4 $\pm$ 13.6	120.6 $\pm$ 15.0
Body fat, %	15.9 $\pm$ 5.4	37.5 $\pm$ 5.0
Body mass index, kg/m <sup>2</sup>	25.0 $\pm$ 4.0	36.9 $\pm$ 4.6
Hemoglobin level, g/L	152 $\pm$ 16	154 $\pm$ 11
FVC, L	6.07 $\pm$ 0.84	5.51 $\pm$ 0.65
FEV <sub>1</sub> , L	4.68 $\pm$ 0.44	4.19 $\pm$ 0.49
Peak expiratory flow, L/s	10.91 $\pm$ 0.97	9.60 $\pm$ 1.32
Total lung capacity, L	7.77 $\pm$ 1.27	7.03 $\pm$ 1.01
Functional residual capacity, L	3.95 $\pm$ 1.02	2.70 $\pm$ 0.38
Diffusing capacity of the lung, mL/min per mm Hg	34.4 $\pm$ 3.9	31.6 $\pm$ 1.9

\* All but five participants had completed college. Values expressed with a plus/minus sign are means  $\pm$  SD.

**Figure 1.** Comparison of the acute mountain sickness (AMS) score at sea level and at simulated altitude for 24 hours in nonobese ( $n = 10$ ) and obese ( $n = 8$ ) participants.



Data are displayed as boxplots, which indicate the distribution of AMS scores within each group. The bottom of the box represents the lower 25th quartile of scores, and the top of the box represents the 75th quartile of scores. The dashed line represents the median. The vertical lines show the largest or lowest value observed outside the box. The black circle shows the position of extreme outlier. The interaction between AMS score and group with exposure was significant ( $P < 0.001$ ).

## DISCUSSION

Our principal finding was that obese participants have higher AMS scores than nonobese participants during a 24-hour exposure to simulated altitude of 3658 m. Thus, obesity seems to be associated with the development of AMS. Also, the response of  $SaO_2$  with exposure differed between nonobese and obese men; obese men had lower values than nonobese men. These findings suggest that impaired breathing during sleep may be an important pathophysiologic mechanism for the increased levels of AMS in obese individuals.

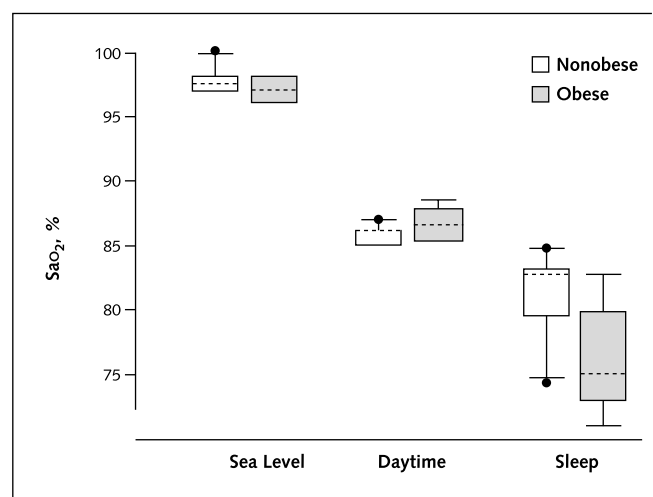
### Limitations

Although our results suggest that obese individuals may be more susceptible to AMS, these results must be interpreted with caution. Possible limitations to generalization include the small sample size, the selected nature of the study sample, the narrow spectrum of obese participants studied, the steady rate of ascent, the lack of physical activity during altitude exposure, and the simulated environment in which the participants were studied.

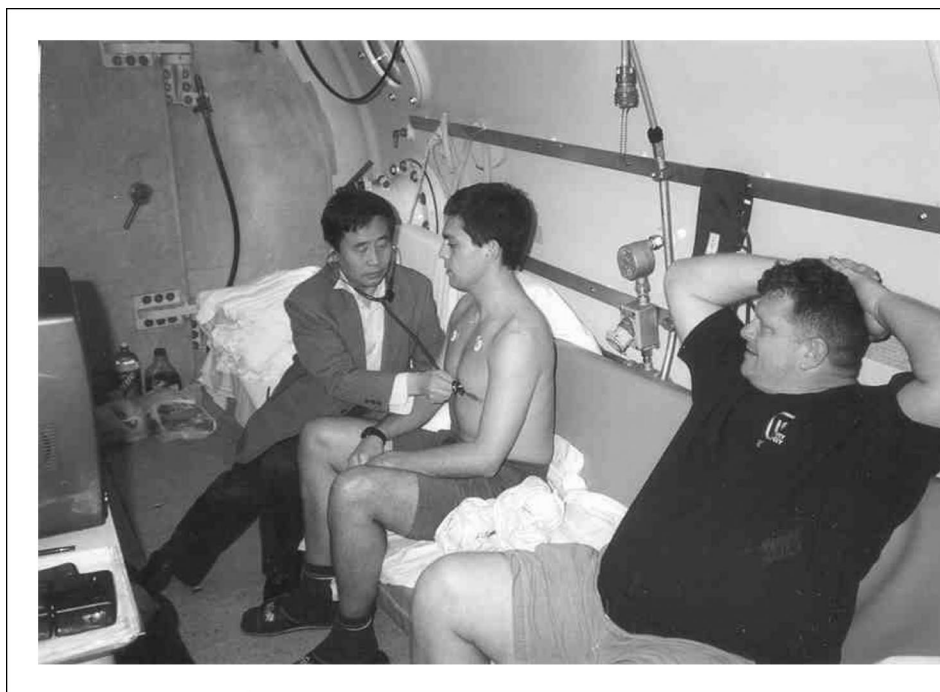
### Obesity and AMS

Obesity is characterized by an abnormally large adipose tissue mass. In particular, excess weight leads to the development of various pathophysiologic disorders and, specifically, cardiovascular and respiratory abnormalities.

**Figure 2.** Comparison of  $SaO_2$  at sea level, during the daytime, and during sleep at night in nonobese ( $n = 10$ ) and obese ( $n = 8$ ) participants.



Data are displayed as boxplots, which indicate the distribution of acute mountain sickness scores within each group. The bottom of the box represents the lower 25th quartile of scores, and the top of the box represents the 75th quartile of scores. The dashed line represents the median. The vertical lines show the largest or lowest value observed outside the box. Black circles show the position of extreme outliers. The interaction between  $SaO_2$  values with exposure and group was significant ( $P < 0.001$ ).

*Figure 3. Participants during simulated altitude exposure in decompression chamber.*

Obesity-related respiratory function abnormalities, such as sleep-disordered breathing and nocturnal hypercapnia and hypoxia, place obese individuals at risk for illness at higher altitudes (10–13). In addition, the prevalence of obesity in western society, especially in the United States (where 22% of the population has a BMI > 30 kg/m<sup>2</sup> and roughly 30% of the population is overweight [14, 15]), further increases the potential for altitude-related difficulties at easily accessible high altitudes during recreational activities.

A review of the literature revealed no prospective data on the effect of obesity on high-altitude illness. In our study, AMS scores increased with time during altitude exposure in both nonobese and obese participants, which is consistent with previous data demonstrating that AMS symptoms are common after 24 hours of rapid ascent to high altitude (1, 2). The severity of symptoms, however, significantly differed between nonobese and obese men, suggesting that the occurrences of AMS at high altitude may be closely related to increased body weight.

Acute mountain sickness frequently occurs in travelers who rapidly ascend to an altitude of 2500 m without acclimatizing; the incidence and severity depend on the speed of ascent, the altitude attained, and susceptibility of the individual. Only a few studies have reported an association between the incidence of AMS and heavier men and women (3, 16). No study controlled for the speed of ascent or absolute altitude achieved, thus preventing the establishment of a clear link between AMS and obesity. In our study, 78% of obese participants and 40% of nonobese participants developed AMS after 24 hours of exposure to the simulated altitude. Thus, this prospective, carefully

controlled study strongly indicates that obese persons develop more severe symptoms of AMS than do nonobese persons after rapid ascent to high altitude.

#### Nocturnal Desaturation and AMS

A previous study has documented that hypoxia at altitude occurs to the greatest degree and lasts longest during sleep (17). In our study, the SaO<sub>2</sub> level was significantly lower in obese participants than in nonobese participants ( $P < 0.001$ ), with the lowest values occurring at night. We speculated that those who have a lower SaO<sub>2</sub> during sleep might have had sleep-disordered breathing, such as periodic breathing with apnea (7, 18). West and colleagues (19) pointed out that the magnitude of hypoxia at extreme altitude during sleep can be related to the intensity of periodic breathing with apnea. However, it is uncertain whether the periodic breathing is associated with the cause of high-altitude illness.

#### Possible Prophylactic Considerations

On the basis of these results, obese persons traveling to high altitude should consider specific prophylactic measures to reduce their risk for AMS. First, they should follow standard guidelines to ensure a slow, gradual ascent. Second, such persons, even those without a history of AMS, should consider using prophylactic acetazolamide, which increases ventilation at altitude and facilitates acclimatization (1).

#### Summary

To determine whether obese men were more susceptible to AMS than nonobese men, we compared the Lake Louise AMS score and SaO<sub>2</sub> values in 9 obese and 10

nonobese men after rapid ascent to a simulated altitude of 3658 m. The results indicated that there were higher AMS scores and lower SaO<sub>2</sub> values in obese participants than in nonobese participants. We conclude that obesity is an important, previously unrecognized factor in the development of AMS.

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