

Altitude Decompression Sickness Susceptibility: Influence of Anthropometric and Physiologic Variables

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Introduction: There is considerable variability in individual susceptibility to altitude decompression sickness (DCS). The Air Force Research Laboratory Altitude DCS Research Database consists of extensive information on 2980 altitude exposures conducted with consistent procedures and endpoint criteria. We used this database to quantify the variation in susceptibility and determine if anthropometric and/or physiologic variables could be used to predict DCS risk. **Methods:** There were 240 subjects who participated in at least 4 of 70 exposure profiles in which between 5 and 95% of all subjects tested developed DCS symptoms. A subject/study ratio (SSR) was calculated by dividing the DCS experienced by a subject during all their exposures by the DCS incidence for all subjects who participated in the identical exposures. The SSR was used to identify the relative susceptibility of subjects for use in analyzing possible relationships between DCS susceptibility and the variables of height, weight, body mass index, age, percent body fat, and aerobic capacity. **Results:** The DCS incidence was 46.5% during 1879 subject-exposures by subjects exposed at least 4 times. A significant relationship existed between higher DCS susceptibility and the combination of lower aerobic capacity and greater weight ($p < 0.05$). **Discussion:** Despite a correlation, less than 13% of the variation in DCS susceptibility was accounted for by the best combination of variables, weight and $\text{VO}_{2\text{max}}$. **Conclusion:** Differences in DCS susceptibility cover a wide range and appear to be related to some anthropometric and physiologic variables. However, there was insufficient correlation to allow prediction of an individual's susceptibility.

Keywords: DCS, age, weight, height, body mass index, aerobic capacity, body fat.

DECOMPRESSION sickness (DCS) symptoms may develop during exposure to reduced atmospheric pressure. Such environments are experienced during unpressurized high altitude flight, extravehicular activity from a spacecraft, or depressurization in an altitude chamber. The chance of developing DCS depends mostly on the level of nitrogen in the tissues (including blood), the specific environmental factors of the decompression, and individual variables (6). The risk of DCS can be reduced by breathing 100% oxygen before exposure to lower the nitrogen content in the tissues and blood. This denitrogenation process is called prebreathing.

Without adequate denitrogenation, decompression-induced supersaturation of tissues with nitrogen may result in formation of a nitrogen gas phase. The gas phase is in the form of bubbles of nitrogen with some

carbon dioxide, oxygen, and water vapor. The bubbles interact with the surrounding tissue and blood by exerting pressure and potentially resulting in biochemical and hematologic responses. The pressure can slow or block blood flow and stimulate responses by sensory nerves. The resulting pain and other symptoms of DCS can cause distraction and may interfere with optimal function (3,14). The symptoms can also be more serious, involving respiratory or neurologic function, resulting in severe loss of performance, abort of a flight mission, and the need for hyperbaric oxygen therapy (8).

Many environmental conditions have been tested to determine the degree of DCS risk in a population (2,5,7,10-13,18,20). The four primary factors that determine DCS risk are altitude, time at altitude, prebreath time, and level of activity (exercise at altitude) (11). However, knowing an individual's susceptibility to a specific environment may be important for successful completion of a particular mission by specific individuals. An individual's susceptibility to DCS is defined here as the response of an individual as compared with the mean response of all subjects exposed to the identical set of conditions. Several individual variables have been reported to have an influence on DCS incidence: level of hydration (5,16); physical fitness (18); age (6,9,15,18); body mass index (BMI, weight in kg/height in m^2 ; 18); weight (9,18); and % body fat (1,18).

The Air Force Research Laboratory Altitude DCS Research Database (DCS Database) contains extensive information on 2980 altitude exposures conducted under consistent procedures and endpoint criteria. The purpose of this study was to retrospectively define the range of susceptibility and to determine the influence of some anthropometric and physiologic variables on DCS

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susceptibility using information from the DCS Database.

METHODS

The altitude exposures were conducted at the Air Force Research Laboratory's Brooks City-Base facilities in San Antonio, TX, during the past 20 yr. While any single exposure was limited to a response of 'DCS' or 'no DCS,' analyzing data from subjects who experienced multiple exposures allowed mean responses; e.g., four exposures could yield a mean of 0, 25, 50, 75, or 100% DCS. The conditions for inclusion in this study were as follows:

1. Subjects participated in at least four of the altitude exposure profiles.
2. Each exposure profile resulted in at least 5%, but not more than 95% DCS during all subject-exposures, including those by subjects not included in the study. In cases where subjects were exposed more than once to identical exposure conditions, the exposures were divided into separate subsets by date of exposure to control for any exposure order effect.
3. At least 10 subjects participated in each selected profile.

There were 2210 subject-exposures by 407 subjects experiencing 47.5% DCS during completion of the 70 profiles which met the above criteria. Of the 2210 exposures, 1879 were accomplished by the 240 subjects with at least 4 exposures. The mean anthropometric and physiologic values for the selected 47 women's and 193 men's 1879 exposures were: women 29.6 yr, 63.9 kg, 1.65 m, BMI 23.3, 22.7% body fat, 35.9 ml · kg⁻¹ · min⁻¹ $\dot{V}O_{2max}$; men 30.4 yr, 83.2 kg, 1.78 m, BMI 26.1, 17.9% body fat, 41.6 ml · kg⁻¹ · min⁻¹ $\dot{V}O_{2max}$. These values are very close to those shown in Webb et al. (18), largely because many of the records were coincident. The voluntary, fully informed consent of the subjects used in this research was obtained, and the protocols were approved by an Institutional Review Board. All subjects passed an appropriate subject physical examination, and were otherwise representative of the USAF rated aircrew population. They were not allowed to participate in scuba diving, hyperbaric exposures, or flying for at least 72 h before each scheduled altitude exposure. Prior to each altitude exposure, a medical monitor conducted a short physical examination of that day's subjects to identify any signs of illness or other problem which would endanger the subject or bias the experimental results.

Since the issues discussed here relate to incidence of DCS within and among individuals, detailed descriptions of the 70 exposure scenarios were omitted in the interest of space. However, descriptions of most of the profiles may be found in other publications from this laboratory (11,13,18,20). These descriptions reveal considerable diversity of exposure conditions, e.g., from zero prebreathe to 4-h prebreathe, from 18,000 ft to 40,000 ft altitude of exposure, from 90 min to 480 min of exposure time, and from rest to heavy exercise during exposure.

Chamber facilities, equipment, and procedures have been described in Webb et al. (18). Breathing gas for preoxygenation (when accomplished), ascent, and altitude exposure was usually 100% oxygen (aviator's breathing oxygen; normal analysis 99.7–99.8% oxygen). To reach the scheduled altitude, subjects were usually decompressed at 1524 m · min⁻¹ and remained at that altitude [6462 m (21,200 ft) to 12,192 m (40,000 ft)] for 1.5 to 8 h or until another endpoint (see below) was obtained. During each exposure, the subjects performed exercises (10,17–21) or remained at rest.

During the exposures, subjects were monitored for, and data collected on, DCS symptoms. Endpoints of the exposures were: 1) completion of the scheduled exposure (1.5 to 8 h); 2) development of DCS signs or symptoms; or 3) observation of left ventricular gas emboli. A more complete description of the endpoints used may be found in Pilmanis et al. (13).

Subject/Study Ratio (SSR)

The ratio of each subject's response to the response of all subjects exposed in those studies was calculated and defined here as the SSR of DCS susceptibility. This metric was developed to classify individual subjects by their relative susceptibility to DCS. The SSR represents the susceptibility of each subject relative to peers who accomplished identical exposures. For example: subject # 85 developed DCS during 5 of 8 exposure profiles (63% DCS), whereas 189 of all 379 subjects exposed to those same profiles developed DCS (50% DCS), yielding an SSR of 1.25 (63%/50%). This means that under this set of profile conditions, subject #85 was 1.25 times as likely to develop DCS as his/her peers.

To study individual relationships between DCS risk (SSR) and height, weight, BMI, age, $\dot{V}O_{2max}$, and body fat, simple linear regression analyses were accomplished. Stepwise multiple regression analyses were then accomplished to find the combination of these independent variables that would provide the best relationship with SSR.

RESULTS

The 70 exposure profiles involved 2210 subject-exposures which resulted in 47.5% DCS. The 240 subjects exposed at least 4 times during these 70 exposure scenarios account for 1879 of these exposures and yielded a very similar 46.5% DCS incidence. Of these 240 subjects, 99 completed at least 4 additional exposures. The mean DCS incidence during the second 4 exposures by all subjects in those profiles was only 1.1% higher (n.s.). The SSRs for their first four exposures were compared with their second four exposures to determine if the first four could have been used as a predictive tool to estimate response to the second four exposures. The mean SSR during the second 4 exposures of all 99 subjects was 0.018 lower (n.s.) than the during the first 4 exposures (range -3.52 to +1.43). The standard deviation of the differences between the SSRs during the first vs. second four exposures was 0.79, indicating considerable variance within individual subjects.

In Fig. 1, the test subjects' ratios were grouped by 0.20

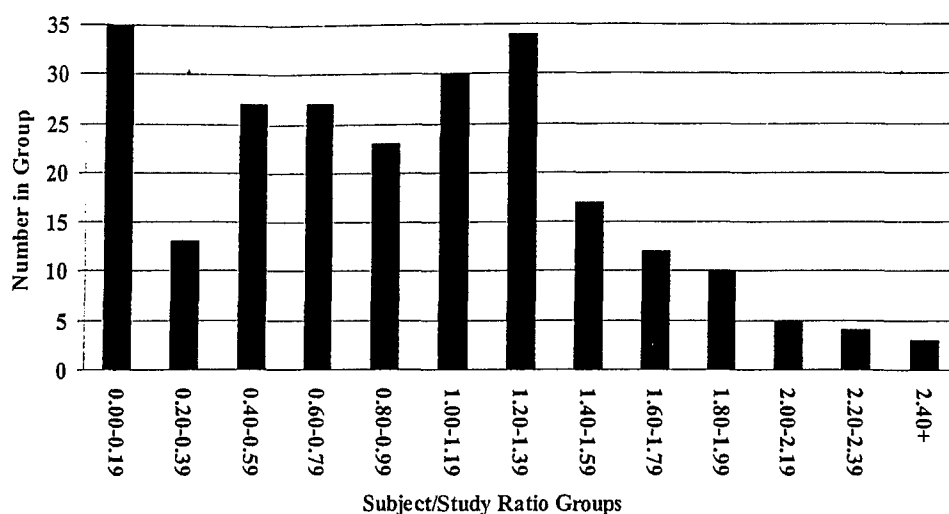


Fig. 1. Distribution of subject-to-study ratios (SSR) for the 240 subjects who participated in ≥ 4 exposures.

increments of SSR, i.e., 0.00–0.19, 0.20–0.39, etc., and the number of subjects within a given SSR group plotted on the vertical axis. The largest of these groups of subjects had an SSR of 0–0.19 ($n = 35$; 15% of the 240 subjects), demonstrating a very high resistance to DCS. There was a wide range of susceptibility (Fig. 1), including subjects with SSRs over 2.00, averaging double the incidence of DCS compared with their peers.

To address the question of whether DCS susceptibility (i.e., SSR) could be predicted from the anthropometric and/or physiologic variables recorded in the database, least-squares regression analyses were employed. Initially a simple regression line was calculated, defining the relationship between SSR and each independent variable (age, weight, height, BMI, percent body fat, and $\dot{V}O_{2\max}$). This allowed us to investigate the impact of each variable as a potential predictor of susceptibility independent of all others. It also allowed us to take advantage of the full amount of data available for each variable since complete data was not available for %

body fat or $\dot{V}O_{2\max}$. The regressions were calculated separately for men and women and for the combined sample. Table I shows that $\dot{V}O_{2\max}$ was significantly related to SSR for both men and women. For the men, significant relationships were also evident for age, weight, and BMI. For this study, what is more important than the significance tests are the R-squared (R^2) values. R^2 defines the percentage of the variation in SSR that is accounted for by the independent variable. $\dot{V}O_{2\max}$ clearly accounts for more of the variation in female susceptibility than any of the other variables (12.6%). For men, $\dot{V}O_{2\max}$ and weight each account for about 4.9% of the variation in susceptibility. Thus, even for these cases where there is a significant relationship between susceptibility to DCS and a variable, the magnitude of R^2 is very small. Indeed, it cannot even be suggested that an individual's DCS susceptibility could be predicted from any one of these variables. Fig. 2 illustrates this point by showing the large amount of

TABLE I. RESULTS OF SIMPLE LINEAR REGRESSION ANALYSES.

Gender	Independent Variable	Sample Size	Slope	R^2	p-value*
Women	Age	47	-0.012	0.012	0.465
	Weight	47	0.014	0.033	0.220
	Height	47	-0.000	0.000	0.988
	BMI	47	0.061	0.055	0.113
	% Body Fat	42	0.039	0.076	0.076
	$\dot{V}O_{2\max}$	43	-0.037	0.126	0.020
Men	Age	193	0.019	0.027	0.024
	Weight	193	0.013	0.049	0.002
	Height	193	0.010	0.011	0.138
	BMI	193	0.048	0.040	0.005
	% Body Fat	145	0.009	0.008	0.299
	$\dot{V}O_{2\max}$	130	-0.019	0.048	0.013
All	Age	240	0.014	0.015	0.060
	Weight	240	0.010	0.041	0.002
	Height	240	0.007	0.010	0.132
	BMI	240	0.047	0.043	0.001
	% Body Fat	187	0.009	0.008	0.216
	$\dot{V}O_{2\max}$	173	-0.018	0.048	0.004

* $p < 0.05$ in bold.

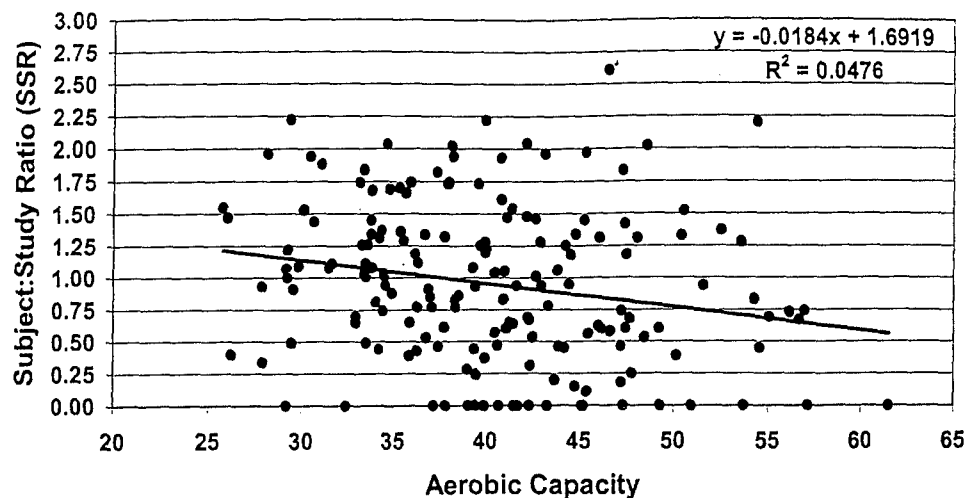


Fig. 2. $\dot{V}O_{2\max}$ vs. subject to study ratio. Note: Data from both male and female subject exposures; $n = 173$.

variation about the regression line relating $\dot{V}O_{2\max}$ to SSR.

In the second stage of analysis, stepwise multiple regressions were performed for women and men, separately, as well as for the complete data set. Because of missing data, the sample sizes for these analyses were reduced to 41 women and 122 men. For women, only $\dot{V}O_2$ was significantly related to SSR ($R^2 = 0.13$). For men, the combination of weight and age provided the best stepwise fit ($R^2 = 0.11$). When all 163 male and female subjects' data including both percent body fat and $\dot{V}O_{2\max}$ were used, the stepwise procedure selected $\dot{V}O_{2\max}$ and weight as the best set of predictors ($R^2 = 0.085$), with the equation defined as: $SSR = 0.85 - 0.018(\dot{V}O_{2\max}) + 0.010(\text{weight})$. In summary, even though there were sets of independent variables that were significantly related to SSR, in each case less than 13% of the variation in SSR could be explained by the relationship, indicating that prediction of an individual's SSR from the set of independent variables studied here is unreliable.

Finally, because of concerns about normality of the SSR data, regression analyses were also performed after applying various transformations to SSR (e.g., square root, log, etc.). In none of these attempts was there any real improvement in the degree of fit of the relationship. Polynomial regressions were also attempted, but no improvement in fit was found. Consequently, these results are not reported.

DISCUSSION

The SSR allows a normalization of results from subject-exposures by accounting for the severity of the exposure. If only the incidence of DCS by a subject exposed to several altitude profiles is used to identify resistant or susceptible subjects, it could reflect an artificially high or low value. For instance, if a subject developed DCS during all five exposure scenarios where 95% of all subject developed DCS, that subject could be falsely identified as highly susceptible. Use of the SSR provided an ability to observe a spectrum of susceptibilities and analyze the data with a greater diversity of statistical methods.

The existence of some highly susceptible subjects in this study may help explain the occurrence of DCS during mild altitude profiles, such as hypobaric chamber exposures for hypoxia demonstration where the DCS incidence is less than 0.3%. Identification of such highly susceptible or resistant subjects could also explain the diversity of research results, especially those studies with a low number of subjects. Even with only 4 very resistant subjects in a control group of 20 subjects compared with a test group of 20 different subjects including 4 very susceptible subjects, differences could be shown where none actually exist. Such susceptibility variations also tend to reduce the validity of stating incidence levels that imply accuracy to less than about 5% about a mean.

The existence of a relationship ($p < 0.05$) between SSR and some anthropometric and/or physiologic variables (Table I) is in agreement with previous findings of relationships between these variables and DCS incidence (1,4,6,7,9). However, the very low R^2 values imply a lack of DCS risk prediction capability using these variables. Further work on the extremes of susceptibility is needed to determine if these or other factors strongly influence susceptibility. A larger sample size would enhance the ability to correlate susceptibility with any specific anthropometric and/or physiologic variable, particularly in female subjects who have completed four or more exposures. However, correlation still may not yield a prediction capability of operational interest.

It appears that some resistant and susceptible subjects could be identified with as few as four exposures by comparing their response to the response of a peer-group of subjects. Identifying individuals as resistant or susceptible to DCS may be useful in situations where DCS risk is relatively high or where preventive measures such as long prebreathe times cannot be accomplished. In such cases, using individuals with demonstrated resistance may be a way to help ensure successful mission completion.

CONCLUSIONS

Test subject susceptibility to altitude DCS can be quantified by calculating the ratio of each subject's re-

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sponse to the responses of their peers during several exposure scenarios. The ratio reflects an estimate of each subject's susceptibility and can be used to evaluate the influence of individual variables on susceptibility. In this study, a wide range of susceptibility was observed. In addition, relationships were found between increased susceptibility to DCS (SSR) and higher weight or lower aerobic capacity. However, individual susceptibility could not be predicted as a function of the anthropometric and/or physiologic variables studied.

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