Nitrogen Load in Rats Exposed to 8 ATA from 10-35°C Does Not Influence Decompression Sickness Risk

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Introduction: Environmental temperature is commonly thought to modulate decompression sickness (DCS) risk, but the literature is mixed regarding which conditions elicit the greatest risk. If temperature is a risk factor, then managing thermal exposure may reduce DCS incidence. We analyzed whether hot or cold conditions during or immediately after a hyperbaric exposure altered DCS incidence in a rat model. **Methods:** Rats (eight groups of five animals in each of nine conditions; mean body mass $\pm$ SD = 259.0 $\pm$ 9.2 g) were placed in a dry chamber that was pressurized with air to 70 m (8 ATA) for 25 min, followed by rapid ($<$ 30 s) decompression under a series of temperature conditions (35°, 27°, or 10°C during compression; 35°, 20°, or 10°C post-decompression). Animals were observed for 30 min post-decompression for signs of DCS. DCS incidence in the 27°C compression/20°C post-decompression group was 50% by design. Data from all nine groups of paired temperature conditions were compared with each other using analysis of variance, Chi-square tests, and logistic regression. **Results:** No significant differences in DCS incidence were found among the groups (30–52.5% DCS incidence per group, 42% DCS incidence overall). **Discussion and Conclusions:** This animal model emphasized potential temperature effects attributable to tissue N_2 load acquired during compression; there was no evidence that environmental temperature from 10–35°C during or post-dive modulated DCS incidence. It remains to be determined if temperature modulates DCS risk as a function of variable N_2 elimination rates.
Keywords: diving, hyperbaria, compression, decompression illness.

DECOMPRESSION sickness (DCS) has been well characterized as a phenomenon associated with elevated gas tensions in tissues of people who breathed inert gas in hyperbaria and then reduced their pressure exposure (2). Rapid exposure to hypobaria, as in high altitude flight or extravehicular activities in space, may also lead to DCS (5). Tables of timed pressure exposures that were considered to be free of DCS risk for divers were generated empirically by the U.S. Navy (7), and came to be used by divers worldwide. The risk of DCS increases rapidly outside these tabled guidelines. However, some divers develop symptoms of DCS from dive exposures that were well within these exposure limits (9,25). A diver may face permanent disability or death from DCS if left untreated, and sometimes even when treated to the best of current medical ability (6).

Improving the dive tables through increased empirical testing of dive exposures and mathematical modeling has been successful at reducing DCS incidence (23), but has not completely solved the problem. Over the years, it has become apparent that DCS risk cannot be entirely accounted for using dive times and depths alone; this residual risk can be mathematically modeled probabilistically as a random event (26). The idea of managing residual DCS risk by more than just the probabilistic approach is nevertheless appealing. Phenomena that appear to occur randomly may contain underlying variables that could be experimentally controlled if adequately identified. Systematic experimentation is a powerful approach to the discovery of causal relationships among variables or events (20). It may be possible through systematic experimentation to identify interventions that can be manipulated in diving protocols to exert some additional level of control over DCS risk.

Temperature is potentially one of these residual risk factors for DCS. One can easily make a case that environmental temperature may alter inert gas uptake and removal. Extreme temperatures or radical temperature changes can lead to changes in metabolic rate, and are usually accompanied by significant blood flow shunts between the peripheral and core circulation, or changes in cardiac output. These changes could in turn have an impact on inert gas flux and, therefore, on DCS risk. However, logic alone does not dictate which temperature conditions promote DCS. Any condition that creates a faster gas wash-in rate could in theory increase DCS risk. However, the same condition should also increase gas washout, which could by itself reduce DCS risk. These opposing effects of the same temperature condition would render the net effect on DCS risk unpredictable (16,18).

Many people associated with diving report the perception that DCS risk depends on environmental temperature (8,10,15,16,25). However, on closer inquiry, the evidence to support these notions is often anecdotal,

contradictory, or at best suggestive that more investigation is in order. A standard reference book on diving medicine states in one chapter (Ref. 25, p. 40) that warm diving is higher risk because a warm diver will absorb additional N_2 at depth. In another chapter of the same text (Ref. 10, p. 172), cold diving is stated to be higher risk because a cold diver will absorb additional N_2 through hyperventilation. During the diving recovery operation following the crash of TWA Flight 800, the medical officers in charge reported that there was a marked increase in DCS incidence once heated dive suits were brought into use; they attributed this effect to warm divers acquiring inert gas more rapidly than they had in the preceding weeks of cold diving (15,16). All of the above statements lack references to controlled studies designed specifically for analyzing temperature effects with supporting data and statistical analysis.

Temperature change immediately following decompression is also reported within the diving community as a potential factor that may increase DCS risk. Some authorities have stated that there is a greater incidence of DCS if divers are cold after decompression because they eliminate inert gas less efficiently (25). A hot bath shortly after surfacing, on the other hand, is often included in DCS case reports as an elevated risk factor (21), presumably because the diver vasodilates and releases inert gas too rapidly (18). Again, these statements, despite their logic based on physical and physiological principles, are contradictory and have been offered without reference to supporting data from controlled experiments.

Ruterbusch et al. (22) reported preliminary results of a study using a range of dive durations in which divers were exposed either to warm conditions (36°C) during a dive and cold (27°C) during decompression, or vice versa. Their results showed that the warm dive/cold decompression condition had a higher risk of DCS than the reverse. However, with this experimental design one could not say if the higher risk was attributable to the warm dive, the cold post-dive, or the combination.

If a systematic analysis of temperature and DCS risk indicated which temperature conditions were optimal during and immediately following a dive, this might be a relatively easy means of reducing residual DCS risk without prolonging decompression time. Understanding the underlying physiology of these phenomena may reveal even more critical information regarding DCS. We performed a study with rats in which animals were exposed to either hot (35°C), moderate (27°C), or cold (10°C) conditions while under compression in air for 25 min, which is not sufficient time to saturate them with N_2 (17). The animals were then decompressed as rapidly as possible, followed by a hot (35°C), moderate (20°C), or cold (10°C) post-decompression period during which the animals were observed closely for signs of severe DCS. This gave us a matrix of nine paired temperature conditions to analyze. This model sequence allowed us to examine potential temperature effects and DCS risk primarily as a function of N_2 uptake kinetics (17).

METHODS

Animals

Rats (*Rattus norvegicus*, all adult males, n = 360 total, body mass range = 239–283 g) were examined on receipt by a member of the veterinary staff. They were housed in pairs in Thoren units and polycarbonate cages. Standard rat chow and water were available to the animals to consume ad libitum. A 12:12 light-dark cycle was maintained. All aspects of husbandry and care were performed in accordance with SOP DVM 230 "Rodent Husbandry." Standard length of holding of animals prior to experiments was 7–10 d. All procedures were approved by the Institutional Animal Care and Use Committee. The experiments were conducted in accordance with National Research Council guidelines on laboratory animal use (19). The institutional animal care facility is fully AALAC certified. Animals were selected randomly for inclusion in experimental groups.

Dive Protocol

Five naïve rats were used per experiment. The rats were placed in a small (140 L) hyperbaric chamber and experienced a simulated dive breathing air. While inside the chamber, rats were housed inside a drum mill made of wire mesh, with compartments that kept the animals from contact with each other. Throughout the simulated dive, the mill turned at 3.6 m · min^{-1}, which obligated the animals to walk at a moderate pace. The mill motion standardized the posture and activity level of the animals and prevented the chamber gas from thermally stratifying.

Based on past research, a compression and decompression sequence was selected that caused rats to have a 50% incidence of DCS when using a thermally moderate dive temperature of roughly 27°C when tested for subsequent DCS at a room temperature of roughly 20°C (the warmer temperature in hyperbaria takes into account the greater thermal conductivity of compressed gases). Chamber conditions simulated a dive to 70 m (231 ft of sea water equivalent pressure; 8 ATA, atmospheres absolute pressure) for 25 min. Compression rate was 1.8 ATA · min^{-1}. Decompression rate was as rapid as possible for the chamber plumbing, returning to 1 ATA in 25 s or slightly less. This rapid decompression rate was chosen as a model that minimizes tissue N_2 elimination during chamber decompression, and thus reflects as much as possible the differences between animals in the volume of gas acquired while they were compressed (17).

Post-decompression time, during which the rats were observed for DCS, was 30 min. Prior experience with rats (12,13) has indicated that in this severe model of DCS, rats can be reliably diagnosed for DCS symptoms by observing them walking (3.6 m · min^{-1}) on a treadmill for 30 min, beginning immediately on returning to 1 ATA. More than 95% of all animals that survive 30 min post-decompression remain alive and free of DCS symptoms after 24 h (12,13). Time of onset of symptoms was recorded for each animal to the nearest quarter-minute. Symptoms of DCS included labored breathing,
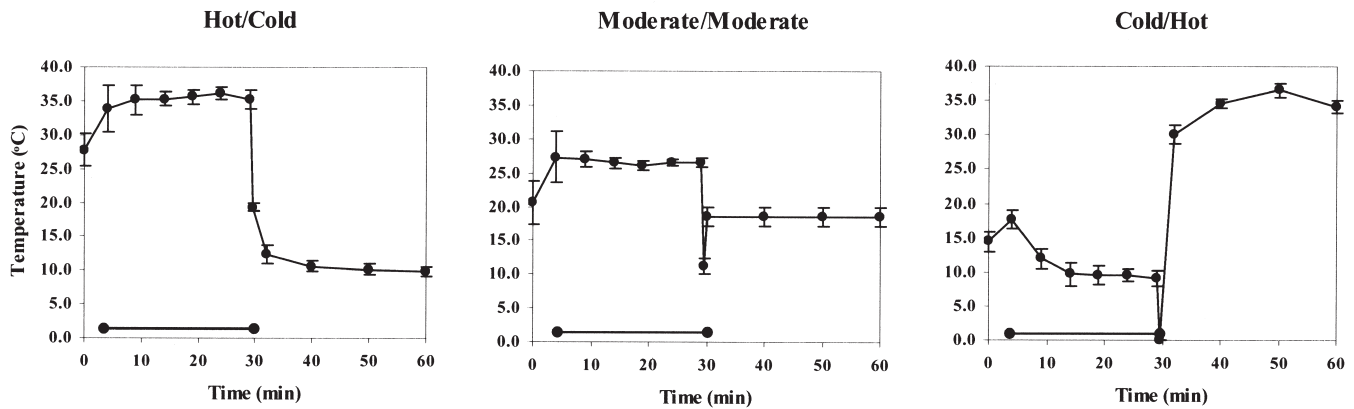
Fig. 1. Temperature variations for the compression/post-decompression conditions of this study in three temperature groups. Horizontal line along the x-axis represents time at 8 ATA. Data points represent means of eight repetitions of the experimental conditions; error bars represent ± 1 SD.

limb numbness, weakness, paralysis, seizures, and death. The severe symptoms of neurological DCS may be distressing to animals, but humans with similar symptoms do not report pain; consequently, the rats were not anesthetized at any time during or after their dives. Some rats had DCS of such severity that they died within one or a few minutes of symptom onset. Other rats had less severe manifestations of DCS that may or may not have reversed in 30 min. All animals surviving for 30 min, including those that had no manifestations of DCS, were euthanized as soon as the 30-min observation period was over by inhalation of CO_2 followed by surgical puncture of the diaphragm.

Once the 50% DCS risk of this dive sequence was established, other rats were tested following the same sequence, but were kept at either 10°C (cold, C), 27°C (moderate, M), or 35°C (hot, H) during the dive, followed by testing for DCS post-dive at either 10°C (C), 20°C (M), or 35°C (H). With these chosen temperatures, animals in the cold condition were clearly shivering and blue in the snouts and paws (peripherally vasoconstricted); animals in the hot condition had bright pink snouts and paws, and descended and red scrota (peripherally vasodilated); and animals at the moderate temperatures had no apparent temperature-related attributes.

The hyperbaric chamber was equipped with a heat-exchanger heat pump with a reversing valve for heating and cooling that operated on R-12 Freon expansion. The chiller function was able to compensate for some of the adiabatic heating of compression, and the chamber arrived at the pre-determined temperature of an experiment within 5–10 min of arrival at maximum pressure. Once at the chosen temperature, the chamber typically remained within 0.5°C for the hot and moderate settings, and within 1–2°C for the cold settings (**Fig. 1**). Adiabatic cooling (10–15°C) of the chamber during the rapid decompression was unavoidable, but of less than a minute duration including time to open the chamber hatch (Fig. 1). Immediately on opening the hatch, the rats were removed from the chamber while still inside the drum mill. The mill was placed in a controlled environment box at one atm for the 30-min post-decompression observation phase. This clear-walled box was also provided with a Freon-expansion heat pump for

heating and cooling. For the moderate post-decompression temperature conditions, the box was left at room temperature. For the hot and cold conditions, the experimentally selected temperature for the box was typically reached within 5–10 min of placing the animals inside, and was then maintained within 1–3°C (Fig. 1). There was no overlap in the three temperature categories among the experiments. Animals walked inside the drum mill for the duration of the observation phase, with momentary stops as needed to retrieve animals that were clearly at or near death from DCS.

Data Analysis

On completion of all experiments, there were nine combinations of paired dive temperature and post-dive temperature conditions, with 40 animals in each group, for a total of 360 animals analyzed. We had predicted that groups of 40 animals each would be adequate to demonstrate a significant ($p < 0.05$, Chi-square test with at least 75% power) change in DCS risk by 50% (i.e., if DCS risk dropped from 50% to 25% or less, or if DCS risk increased from 50% to 75% or more).

The DCS outcomes from the nine combinations of temperature conditions were tested by analysis of variance (ANOVA) and the Chi-square test for homogeneity. This homogeneity test looked for any overall differences in DCS incidence among the nine groups that were unlikely to be due to chance alone. Logistic regression and likelihood ratio tests in the manner described by Hosmer and Lemeshow (11) were used to construct a dose-response function in which the incidence of DCS (the independent variable) was attributed to animal mass and temperature during and after the dive. A survival analysis, using a log-rank test, was used to compare the time to DCS symptom onset among the treatment groups.

RESULTS

The mean body mass per group of rats varied by a maximum of 10 g among the nine groups (range 254 to 264 g). Although these differences in mass per group were small, an ANOVA indicated that there were statistically significant differences in mass among the groups ($p < 0.01$). However, logistic regression analysis

showed that body mass did not significantly correlate with DCS outcome ($p > 0.2$).

Time to onset of DCS symptoms among all individual animals ranged from 2.5 to 21 min. For those animals with fatal DCS cases, time to death ranged from 2.75 to 29 min. Although mean time to onset of DCS symptoms or death per group differed by only roughly 4 min among the nine groups (range 4.5 to 8.4 min mean time to DCS onset; range 6.5 to 10.4 min mean time to death), an ANOVA indicated that there were statistically significant differences among the groups for these times ($p < 0.01$). However, a log-rank test showed that there were no differences in time to DCS symptom onset or death between groups as a function of temperature ($p > 0.2$).

Among the 360 animals in the 9 temperature treatment groups, there were 152 (42.2%) cases of DCS observed (**Table I**). DCS incidence per treatment group of 40 animals ranged from 30% to 52.5% (Table I). Among these DCS cases, 107 (29.7%) animals died from their post-dive complications (Table I). Severe DCS was the only experimental cause of death. The Pearson Chi-square test for homogeneity indicated that there were no statistically significant differences among the nine groups for DCS incidence ($\chi^2 = 7.68$, 8 d.f., $p > 0.4$; **Fig. 2A**) or death ($\chi^2 = 9.2$, 8 d.f, $p > 0.20$; **Fig. 2B**). Since binomial data (DCS/no DCS; death/no death) have no means or standard errors of means, we have presented these data with their 95% confidence limits for comparisons (Fig. 2). Our experimental design was set to identify conditions as significant only if they produced a DCS incidence lower than 25% or greater than 75%, given that the moderate temperature condition had a 50% incidence. Thus, the differences among the nine temperature treatment groups were sufficiently small that we cannot rule out the possibility that the differences were due to chance alone.

We also considered our data in the form of continuous variables of body mass and temperature. In a logistic regression analysis using actual temperatures per experiment (rather than the H, M, and C categories) as dependent variables, there was no significant correlation between DCS incidence and either dive or post-dive temperatures ($p > 0.40$). A logistic regression analysis of death incidence vs. actual temperatures showed no significant correlation between death incidence and post-dive temperature ($p > 0.30$). There was a trend ($p = 0.07$) toward death incidence increasing with
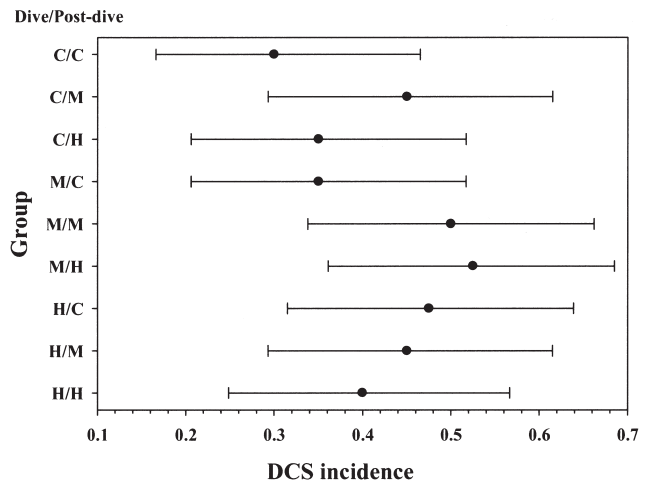
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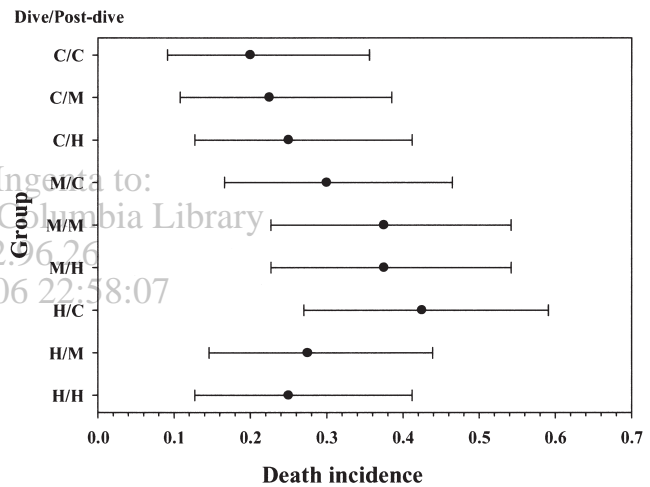
B.

Fig. 2. A) Incidence of or B) fatality from DCS with 95% confidence limits in rats exposed to 8 ATA for 25 min followed by rapid decompression within 30 s to 1 ATA. Rats (n = 40 per treatment group) were exposed to a hot (H = 35°C), moderate (M = 27°C), or cold (C = 10°C) environment during the dive exposure, and to a hot (H; 35°C), moderate (M; 20°C), or cold (C; 10°C) environment immediately post-decompression.

warmer dive temperature, but this correlation was not robust; minor test changes in the death incidences in the highest and lowest temperature groups had a large impact on the significance of this regression. The ratio of number of deaths to DCS incidents per treatment group did not vary significantly with dive temperature (ANOVA, F = 1.11, 8 d.f., $p = 0.39$).

DISCUSSION

Several studies have been performed in which operational diving databases have been analyzed in an effort to determine retrospectively if temperature effects

TABLE I. INCIDENCE OF DCS (AND OF FATALITY) IN RATS (n = 40 PER GROUP) EXPOSED TO 9 POSSIBLE PAIRED CONDITIONS OF TEMPERATURE, GROUPED AS HOT (H), MODERATE (M), AND COLD (C) DURING A HYPERBARIC EXPOSURE AND IMMEDIATELY POST-DECOMPRESSION.

| | | Post-Dive Temperature | | |
		H	M	C
	H	16 (10)	18 (11)	19 (17)
Dive Temperature	M	21 (15)	20 (15)	14 (12)
	C	14 (10)	18 (9)	12 (8)

Dive exposure was to 8 ATA for 25 min, followed by rapid decompression within 30 s to 1 ATA.

could explain a component of DCS risk (3,16,24). However, it is problematic to perform these studies well. It is difficult to control mathematically for the known DCS risk factors of dive depth and duration, which may not be recorded with the same precision in an operational database as they would be ideally in a laboratory-controlled database. Air and water temperatures are seldom recorded in operational settings, in which such information is usually not deemed important.

Using dives for which time and depth had been statistically controlled, Leffler (16) concluded from a retrospective analysis of U.S. Navy diving data that divers in heated suits had nearly twice the risk of any manifestation of DCS compared with divers with unheated suits. Leffler also found that each 10°C increase in water temperature increased DCS risk by a factor of nearly two. However, when the DCS manifestations were divided into Type 1 (pain only, skin lesions) and Type 2 (neurological and cardiopulmonary) symptoms, the dependence of DCS risk on temperature disappeared for Type 2 manifestations. All of the divers in the historical data set used by Leffler decompressed in a chamber on the surface, but the temperature of this post-dive environment was not reported.

A retrospective analysis of diving data from the British Navy was performed by Broome (3), in which dives were grouped as either "safe" or "risky" in order to control for dive duration and depth. In that study, dive temperature alone was not found to be a risk factor for DCS, but a high temperature differential between a relatively warm water dive, and a cold, windy air exposure post-decompression was found to increase DCS risk. Thus there is some discrepancy between the results reported by Leffler (16) and Broome (3). This discrepancy may be attributable at least in part to the limitations inherent in analyzing data retrospectively that were not collected to test a hypothesis. The discrepancy may also be due to considering dive temperature alone (16) vs. dive and post-dive temperature combinations (3).

The outcomes reported by Ruterbusch et al. (22), in which the DCS incidence for warm (36°C) dives followed by cold (27°C) decompression was higher than for cold dives followed by warm decompression, corroborate the findings of Broome (3) and are also consistent with those of Leffler (16). Our data comparing H/C to C/H dives (Fig. 2) do not provide statistical support for a temperature effect on DCS risk in this rat model. However, it must be understood how our model differs from the human dives examined in the other studies (3,16,22) in order to place our work in a perspective that may be relevant to them.

We used an animal model that is easily managed and well characterized for decompression research (12,13,17). It is clear that dry, compressed gases are not the same thermal stressor as water, and that a rat and human do not have similar quantitative thermoregulatory responses. Rather than mimic human divers directly, we were creating model conditions in which rats were clearly cold or hot enough to qualitatively elicit the general kinds of cardiovascular and respiratory shifts believed to be relevant to DCS research. The signs of

DCS associated with a 50% incidence in a rat model are far more severe than any that would be condoned in human trials. This severity reduces considerably the ambiguity in identifying symptoms of DCS from manifestations of thermal or other stressors. The high DCS incidence also allows us to maintain statistically useful sample sizes at levels that are more practical than those typically found in human trials. Our objective in this study was to determine if DCS risk was either increased or decreased by environmental temperature during the dive or immediately after decompression specifically as a function of gas load acquired during the time the animals were compressed. The compression and decompression sequence selected here was thus not one that any human trial or planned dive mission would ever use, but rather an experimental means of teasing apart any differential DCS risks associated with temperature in gas uptake phenomena from those of gas release phenomena. That is to say, in the current study we have intentionally examined only half of the problem of inert gas flux in DCS.

To examine temperature effects on DCS risk rigorously, this study should be followed by at least two others in which rats are tested within a similar matrix of nine temperature conditions, but using new compression and decompression sequences. In the next series, rats should be tested for temperature effects and DCS risk primarily as a function of N_2 elimination kinetics. This can be accomplished by leaving rats under pressure for sufficient time to saturate them with N_2 (90 min; 17) followed by a slow decompression rate (≤ 2 ATA · min^{-1}) that would allow for any differential gas release rates as a function of temperature to be manifest. In another series, the pressure profiles should be combined to study rats that are subsaturated and slowly decompressed in order to combine potential differences in gas uptake and gas elimination as a function of temperature simultaneously.

The data from our current study neither confirm nor refute the studies of Leffler (16), Broome (3), or Ruterbusch et al. (22), but the combination of the current and proposed studies may complement them. It may be that the higher residual DCS risks reported in these three human studies are reflective either of temperature effects associated primarily with N_2 elimination, or with a specific pairing of N_2 uptake and elimination phenomena. Ultimately a larger animal model that can be fully instrumented will be needed to determine what physiological events are correlated with these risk-enhancing gas fluxes. It is not at all clear whether DCS risk would be reduced if N_2 were eliminated more rapidly or more slowly than the rate for divers at moderate temperatures, and by what mechanisms. Doppler ultrasound detection of vascular bubbles may be a technique that could be useful in these studies with a larger animal model, although the link between Doppler bubble score and DCS manifestation is controversial (1,4,14). The current and proposed series of rat studies should reduce the number of conditions needed for such a study with a larger, more expensive, and more time-consuming animal model. A complete collection of information from these proposed studies could have a

major impact on understanding the natural history of DCS and mitigating residual DCS risk in the future.

We conclude that changes in environmental temperature from 10–35°C during and immediately after diving, using a model dive profile that emphasizes differential tissue N_2 uptake in rats, does not affect DCS risk. It remains to be determined if environmental temperature affects DCS risk when using other model dive profiles that examine N_2 elimination rates.

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