

## The impact of exertional heat stress induced hypohydration on Human cardiovascular function

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### ABSTRACT

Hypohydration, commonly known as dehydration refers to a decline in body water level below normal. Sportsmen, athletes, soldiers and other occupational workers exhibit predisposition towards hypohydration due to rigorous physical activity under elevated temperature. We have investigated the effect of 2% and 4% body weight loss due to exertional heat stress induced hypohydration on cardiovascular response in Human body to contribute to the development of approaches to combat the morbidity and mortality associated with hypohydration. Six healthy military personnel performed sub-maximal exercise at 45 °C and 30% Relative Humidity (RH) in Human Climatic Chamber (HCC) till the desired levels of hypohydration were achieved. Cardiovascular parameters were evaluated pre and post exercise. Post 2% and 4% hypohydration, a significant difference was observed in cardiovascular and thermoregulatory responses compared to the euhydrated state (pre exercise) indicating that hypohydration has a pronounced effect on cardiovascular system and thermoregulation in Human body.

**Key words:** Dehydration, Heart Rate Variability, cardiac output, stroke volume, Core body temperature, Skin mean temperature, Heart Rate, Human Climatic Chamber

## 1. INTRODUCTION

When the body water level falls below the normal threshold, the condition is referred to as hypohydration. Performing tasks involving rigorous physical activity for extended durations under high temperature predisposes certain occupational workers such as athletes, soldiers, commercial kitchen workers and fire fighters to being hypohydrated. Hypohydration has been shown to adversely affect the physical work capacity (Consolazio et al., 1968; Shirreffs & Sawka, 2011; Pichan et al., 1988). Soldiers when deployed to areas with restricted food and water supply experience heightened susceptibility to hypohydration, which takes a toll on their work efficiency (Consolazio et al., 1968).

We have investigated the effect of 2% and 4% body weight loss due to exertional heat stress induced hypohydration on cardiovascular response in Human body with an aim to facilitate reduction in morbidity and mortality associated with hypohydration.

Prolonged physical activity under the conditions of elevated temperature results in a rise in body temperature caused due to a cumulative effect of factors including energy expenditure, environmental conditions and clothing. Sweating is the fundamental mechanism by which the metabolic heat is dissipated from Human body, which results in a reduction in body fluid volume, inflicting a strain on the cardiovascular function (Monero et al., 2013).

The aim of this study is to evaluate the effect of moderate hypohydration (upto 4%) exertional heat stress induced hypohydration on cardiovascular function in Human. Six healthy military personnel performed sub-maximal exercise at 45 °C and 30% RH in the HCC till the desired levels of hypohydration were achieved. Various cardiovascular parameters such as HR, SV, SO and HRV along with Tc and Tm were evaluated before and after the heat exposure in human climatic chamber. A significant difference in the levels of Tc, Tm, CO, SV, HR, LF: HF, total power, R-R interval, sweat loss and sweat rate was observed post 2% and 4% hypohydration compared to control indicating that moderate hypohydration upto 4% has a pronounced effect on cardiovascular function.

## 2. MATERIALS AND METHODS

### 2.1. Ethical approval

Prior to the study, the experimental procedure including the risks involved was explained to each participant and written consent obtained. All protocols and procedures were approved by Institutional ethical committee for Humans, Defence Institute of Physiology and Allied Sciences (DIPAS), Defence Research and Development Organization (DRDO). The studies conformed to the standards set by the latest revision of the *Declaration of Helsinki*.

### 2.2. Participants

Six healthy male Defence personnel [age  $31.33 \pm 2.5$  years, height  $170.4 \pm 7.5$  cm, body mass  $76.85 \pm 5.1$  kg, body surface area  $1.87 \pm 0.1 \text{m}^2$ ] participated in the study. Inclusion criteria consisted of a medical history free of musculoskeletal, cardiac, endocrine, and heat-related illnesses. They consumed the appropriate amount of water before the commencement of exercise to attain euhydrated state after which they performed submaximal exercise (Åstrand & Rodahl, 1986) till the desired weight reduction was achieved.

### 2.3. Experimental design

To assess the effects of exertional heat stress induced hypohydration on cardiovascular function, the participants were made to perform sub-maximal exercise (Åstrand & Rodahl, 1986) in HCC simulated at 45 °C and 30% RH. The exercise was performed at two separate occasions to attain two different hydration states: 2% body weight reduction or 2% hypohydration and 4% body weight reduction or 4% hypohydration. The two exercise sets were separated by at least two weeks to ensure the re-establishment of normal physiological status by nullification of the effect of previous exposure. All the participants were instructed to consume balanced diet and not to engage in any vigorous physical activity, smoking and alcohol consumption for at least 24 hours prior to each exercise session. All the sessions were conducted approximately at the same time for each participant. 4% hypohydration represents a state of significant body water loss and the value was chosen to facilitate a comprehensive assessment of potential implications of severe hypohydration on Human body. We couldn't evaluate the effect of more severe hypohydration due to the volunteers' safety considerations.

### 2.4. Experimental procedure

The subjects were instructed to ingest 5ml of water per kg body weight 2 hours before reporting to the laboratory to attain a euhydrated state (Montain & Coyle, 1992). After reporting to the laboratory, the subjects rested for 60

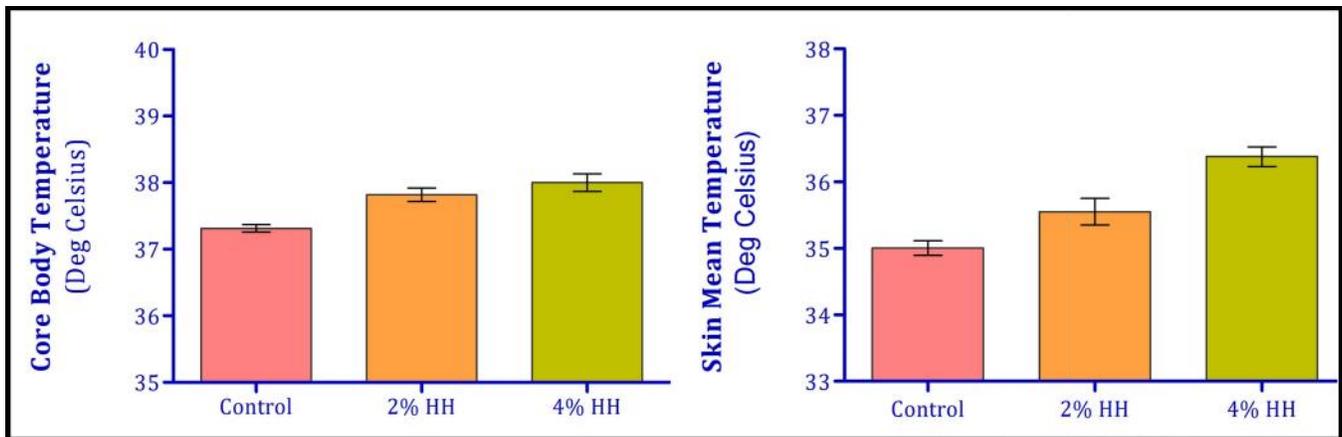


Figure 1

Hypohydration and body temperature

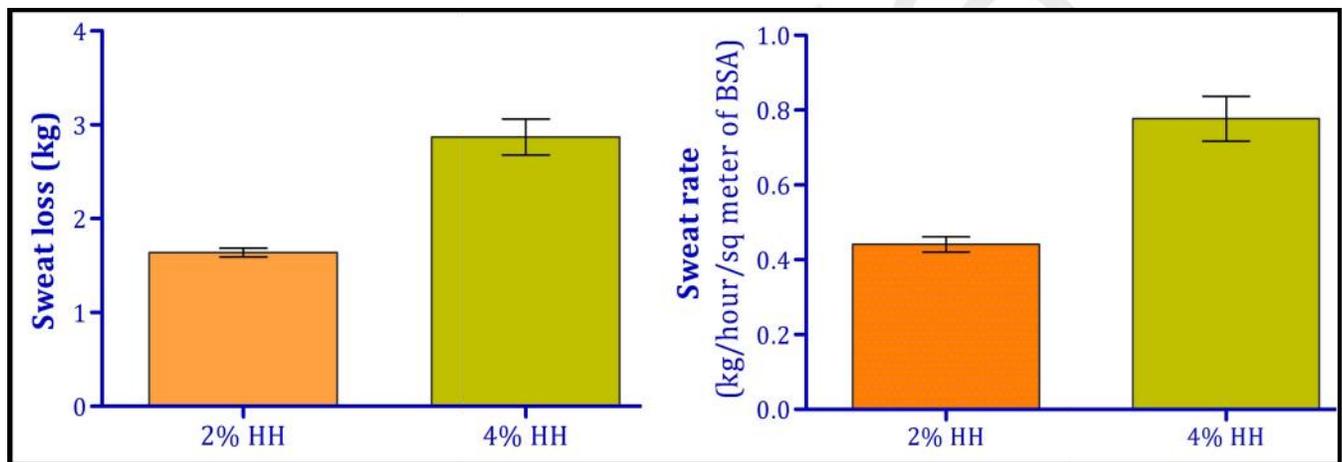


Figure 2

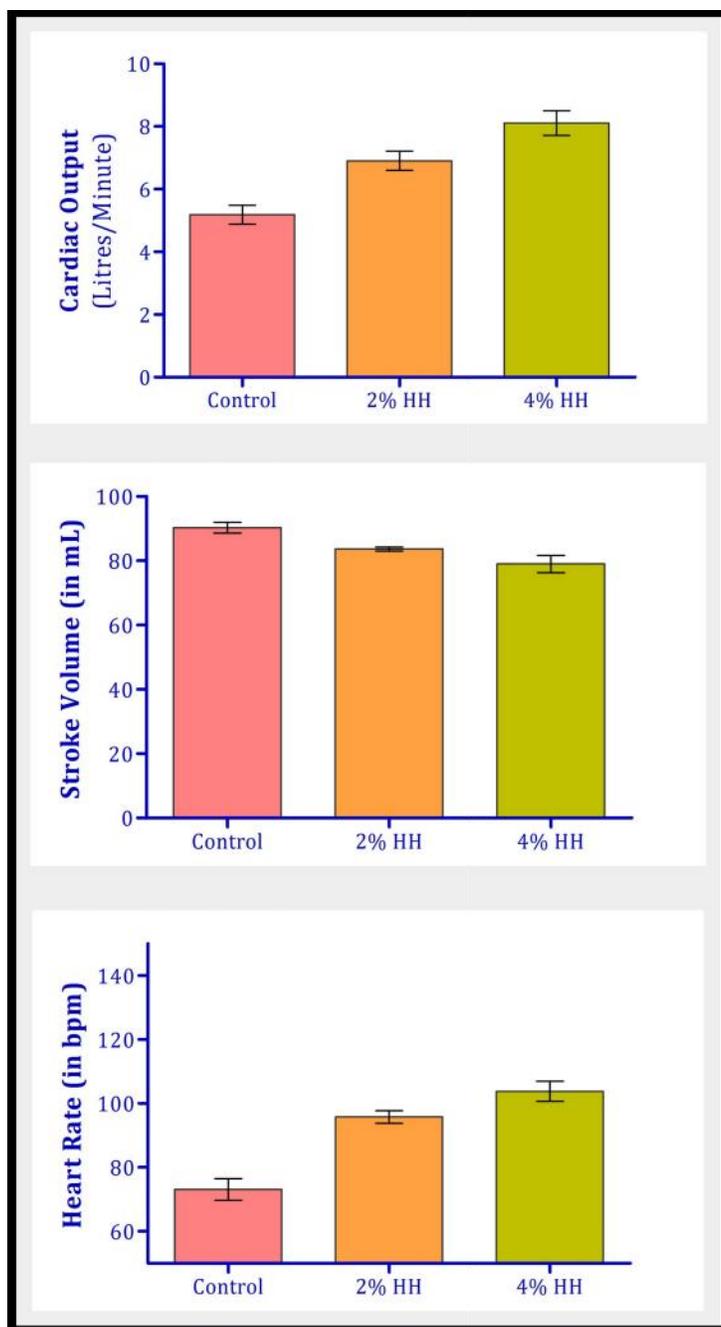
Sweat rate changes with graded hypohydration

minutes. HRV measurement was carried out for 15' at rest using standard limb lead II by telemetric physiological monitoring system (Nexus-10, Netherlands) and data was collected after getting stable electrocardiographic baseline. R-R interval was used for HRV analysis. These intervals were processed using Kubois HRV analysis 2.0 software (University of Kuopio, Finland) (Schultz et al., 2011). Electrocardiography (esaote my lab 30 gold cardiovascular) was performed and oral temperature was recorded using YSI electrodes from which Tc was deduced and Tm was calculated using Ramanathan equation (Ramanathan, 1964). Initial nude body weight was measured using digital human weighing machine, model PPF 100K and make PERFECT. One participant entered the HCC simulated at 45°C and 30% RH at a time and performed sub-maximal exercise (Standardized step test, 15 steps/min), (Åstrand & Rodahl, 1986) until targeted hypohydration level was attained. After the completion of exercise in the HCC, the subject was instructed to exit the chamber. The post exposure data was obtained. Sweat loss\* was calculated by measuring the difference in weight before and after the exposure and sweat rate was calculated (sweat loss/hour/m<sup>2</sup> of the body surface area).

\* Sweat loss = [(Nude body weight pre exercise-nude body weight post exercise) + volume of water consumed if any-volume of urine generated if any]

### 2.5. Statistical analysis

All variables were analysed using one way ANOVA. At least a 95% confidence level (p<0.05) was used as a threshold for statistical significance.



**Figure 3A**  
Hypohydration and cardiovascular changes

physiological status by nullification of the effect of previous exposure. It is acknowledged that the hypohydration in Military and sports settings can at certain occasion be much more severe than 4%, but due to the concerns of subject safety, hypohydration above 4% was not included in the experimental design.

Tc and Tm were found to increase with the degree of hypohydration in our study. This is in accordance with the observation made by Sawka et al. (1985), who investigated the effect of body water loss on hyperthermia during exercise. In their study, the subjects were hypohydrated by 0%, 3%, 5%, and 7% of body weight through a cumulative effect of restricted food and fluid intake coupled with heat exposure and exercise. During exercise, a linear relationship was observed between the level of hypohydration and hyperthermia with an elevation of 0.15°C with 1% reduction in body weight when compared with euhydrated state temperatures with same degree and conditions of exercise.

### 3. RESULTS

#### 3.1. Body temperature

Tc was calculated from oral temperature after application of a correction factor. It was found to be  $37.31 \pm 0.14$ ,  $37.82 \pm 0.25$ ,  $p < 0.01$  (0.008) and  $38.00 \pm 0.33$ ,  $p < 0.01$  (0.008) in euhydrated state, Post 2 % hypohydration and Post 4% hypohydration respectively (Figure 1). Tm was calculated using Ramanathan formula using 4 point measurement i.e.  $0.3 * (T_{chest} + T_{biceps}) + 0.2 * (T_{calf} + T_{thigh})$  which was found to be  $35 \pm 0.2$ ,  $35.55 \pm 0.5$ ,  $p < 0.05$  (0.018) and  $36.38 \pm 0.4$ ,  $p < 0.001$  (0.0003) in euhydrated state, Post 2 % hypohydration and Post 4% hypohydration respectively.

#### 3.2. Sweat loss and Sweat Rate Analysis

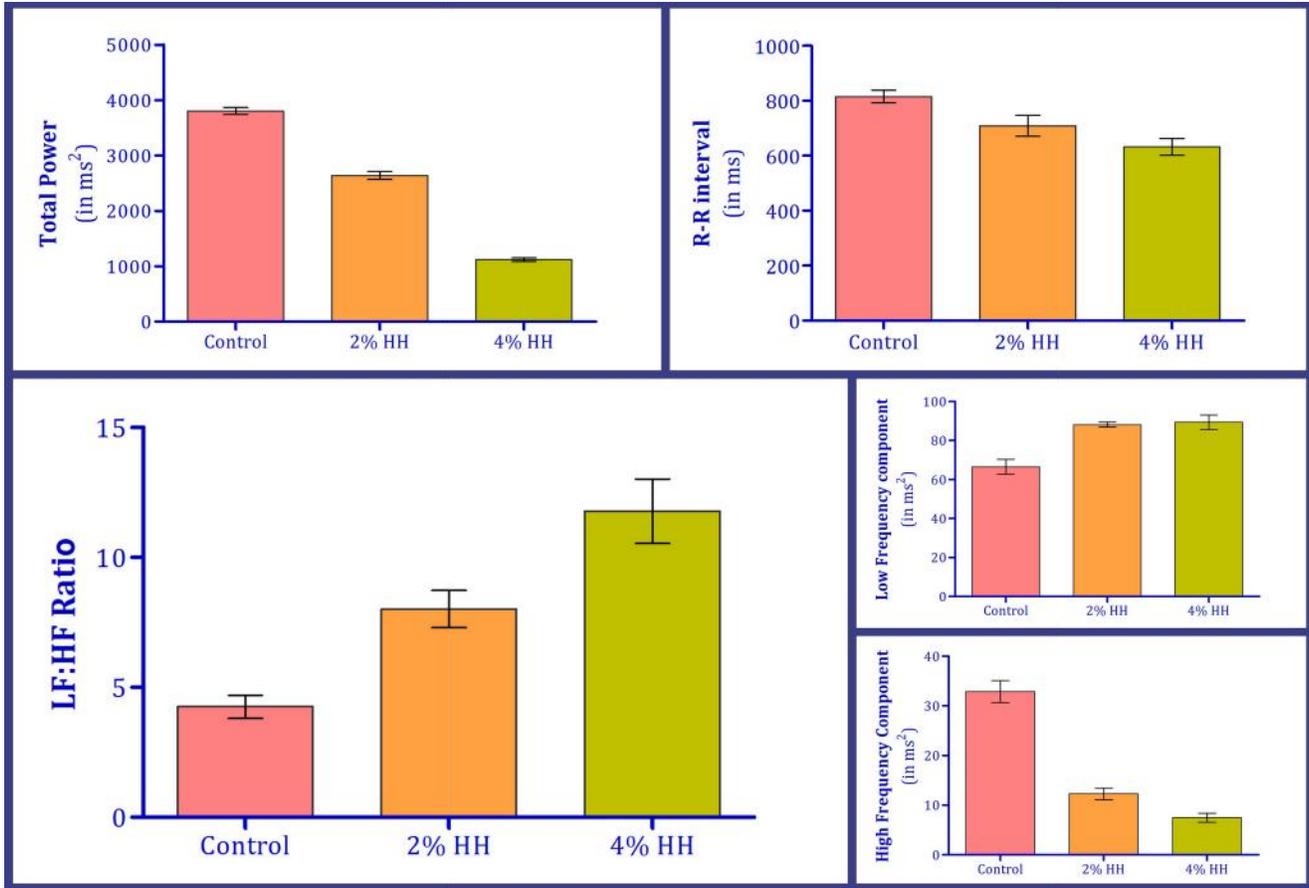
Sweat rate (in kg) and sweat loss (in kg/hour/m<sup>2</sup> Body Surface Area) were found to increase with the duration of exercise (Figure 2). It was found to be  $1.64 \pm 0.11$  kg,  $p < 0.001$  (3.44144E-07) and  $2.87 \pm 0.47$  kg,  $p < 0.001$  (2.34493E-05) post 2 % hypohydration and post 4% hypohydration respectively. Sweat rate was found to be  $0.44 \pm 0.05$ ,  $p < 0.001$  (3.44144E-07) and  $0.78 \pm 0.15$ ,  $p < 0.001$  (2.34493E-05) post 2 % hypohydration and post 4% hypohydration respectively.

#### 3.3. Cardiovascular Responses

Heart rate was noted as  $73.1 \pm 7.2$ ,  $95.8 \pm 5.3$ ,  $p < 0.001$  (0.00018) and  $113.8 \pm 6.3$ ,  $p < 0.001$  (0.0003) under conditions of euhydration, post 2 % hypohydration and post 4% hypohydration respectively (Figure 3A & 3B). SV was recorded to be  $90.30 \pm 3.66$ ,  $83.68 \pm 1.42$ ,  $p < 0.05$  (0.005) and  $79 \pm 6.04$ ,  $p < 0.05$  (0.007) under euhydration, post 2 % hypohydration and post 4% hypohydration respectively. CO was found to be  $6.60 \pm 0.7$ ,  $8.01 \pm 0.7$ ,  $p < 0.05$  (0.002) and  $8.99 \pm 0.9$ ,  $p < 0.001$  (0.0005) under euhydration, Post 2 % hypohydration and Post 4% hypohydration respectively.

### 4. DISCUSSION

This study determined the impact of exertional heat stress induced hypohydration on Human cardiovascular function. The participants were made to perform sub-maximal exercise (Åstrand & Rodahl, 1986) in HCC simulated at 45 °C and 30% RH. The exercise was performed at two separate occasions to attain two different hydration states: 2% body weight reduction or 2% hypohydration and 4% body weight reduction or 4% hypohydration. The two exercise sets were separated by at least two weeks to ensure the re-establishment of normal



**Figure 3B**

Hypohydration and cardiovascular changes

Heart rate was found to increase significantly upon 2% and 4% hypohydration compared to the euhydrated state. Also, the heart rate recorded post 4% hypohydration was higher as compared to that recorded for 2% hypohydration. SV decreased with an increase in the magnitude of hypohydration whereas the CO was elevated upon increase in the degree of hypohydration, which could be a result of increased heart rate compensating for the decrease in SV to increase the CO. This increase in CO upon dehydration is contrary to the results obtained in the study by Montain and Coyle (1992) in which Tc and heart rate were found to increase linearly and SV and CO declined significantly with an increase in the magnitude of dehydration.

HRV analysis is a reliable tool for estimating the autonomic nervous system balance with the opposed oscillatory effects of the sympathetic and parasympathetic systems on the sinus node of the heart HRV, undermined by the contributions from vagal and sympathetic tone. The high-frequency band (0.15–0.40 Hz) power spectrum is primarily determined by vagal activity and the low-frequency band (0.04–0.15 Hz) power spectrum reflects the sympathetic nerve activity. Therefore, the absolute power ratio in the LF and HF bands (LF/HF ratio) indicates sympathetic:parasympathetic balance (Liu et al., 2008). In our study, an increase in the LF and LF:HF ratio was observed upon hypohydration, with a decrease in HF and total power indicating increased sympathetic activity upon 2% and 4% hypohydration. This finding is in contradiction with the results obtained in a study by Carter et al. (2005) who attempted to evaluate the combined effect of heat stress and hypohydration on HRV in Humans. An overall decrease in HRV was observed upon both the hypohydration and exercise heat stress. A reduction in total power, LF, very low frequency, and LF:HF ratio was observed upon hypohydration with a significantly higher HF.

These results indicate that moderate hypohydration up to 4% drastically influences the cardiovascular function in Human body. These changes become more pronounced with an increase in the intensity of hypohydration rising to prominence upon 4% hypohydration as compared to 2% hypohydration.

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