



# Medical Science

The International Weekly Journal for Medicine

## The effect of acute exertional heat stress and heat acclimation on cardiovascular responses and stability in Human

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### Publication History

Received: 14 April 2014

Accepted: 19 May 2014

Published: 28 May 2014

### Citation

Laxmi Prabha Singh, Medha Kapoor, Amir Chand Bajaj, Abhishek Bhardwaj, Rajender K Guta, Thiruthara P Baburaj, Shashi Bala Singh. The effect of acute exertional heat stress and heat acclimation on cardiovascular responses and stability in Human. *Medical Science*, 2014, 8(31), 71-76

### ABSTRACT

Heat acclimatization refers to the phenomenon of adaptation to the external heat acquired through the natural and gradual exposure to elevated temperature. Heat acclimation, in principle, is quite similar to heat acclimatization, the major difference being that heat acclimation is attained in environmentally controlled simulation chambers. The current study investigates the effect of acute exertional heat stress on cardiovascular function in Human and the impact of heat acclimation in minimizing the cardiovascular strain imposed upon acute exertional heat stress. Six human volunteers participated in the study. They were subjected to acute exertional heat stress and heat acclimated in Human Climatic Chamber simulated at 45°C and 30% RH. Cardiovascular stabilization was observed upon heat acclimation to the conditions of exertional heat stress as measured upon evaluation of Heart Rate Variability, Cardiac Output and stroke Volume.

**Key Words:** Heart Rate Variability, Cardiac Output, Stroke Volume, Core body temperature, Skin mean temperature, Human Climatic Chamber

## 1. INTRODUCTION

During military operations, the soldiers are often deployed to hot regions. This renders them predisposed to what are known as the heat related illnesses, which include conditions as mild as heat cramps to as severe as heat stroke. It is very important to acclimatize soldiers in advance to heat stress to reduce the risk of heat related illnesses. The process of acclimatizing individuals artificially in controlled climatic chambers is referred as acclimation. We have developed a heat acclimation schedule of eight-day duration. The current study investigates the effects of acute exertional heat stress on cardiovascular parameters and heat acclimation in conferring improved tolerance and adaptation to acute heat stress.

Heat acclimatization refers to the phenomenon of adaptation to the external heat acquired through the natural and gradual exposure to elevated temperature. Heat acclimation, in principle, is quite similar to heat acclimatization, the major difference being that heat acclimation is attained in environmentally controlled simulation chambers (Dantzler, 1997; Magalhães et al. 2010; Nadel et al., 1974).

Heat acclimation is crucial for combating the morbidity and mortality associated with heat related illnesses. Although acclimatization for heat is crucial for the inhabitants of hot regions, it particularly of a greater relevance for certain occupational workers such as Military personnel, athletes, farmers, and commercial kitchen workers etc. as they undergo rigorous physical activity under the conditions of elevated temperature for extended durations (Carter et al., 2005; Jackson & Rosenberg, 2010; Mirabelli et al., 2010). Six healthy male participated in the study. They performed Sub-maximal exercise (Standardized step test) in HCC simulated at 45°C, 30% RH for 100 minutes/ day, eight days in a row to attain heat acclimation. Cardiovascular and thermoregulatory parameters were recorded at three occasions: before beginning of the exercise on day 1(control), after exercise on the first day (acute exertional heat stress) and after exercise on the 8th day (post-acclimation).

Cardiovascular stabilization was observed upon heat acclimation to the conditions of exertional heat stress as measured upon evaluation of HRV, CO and SV.

## 2. MATERIAL AND METHODS

### 2.1. Participants

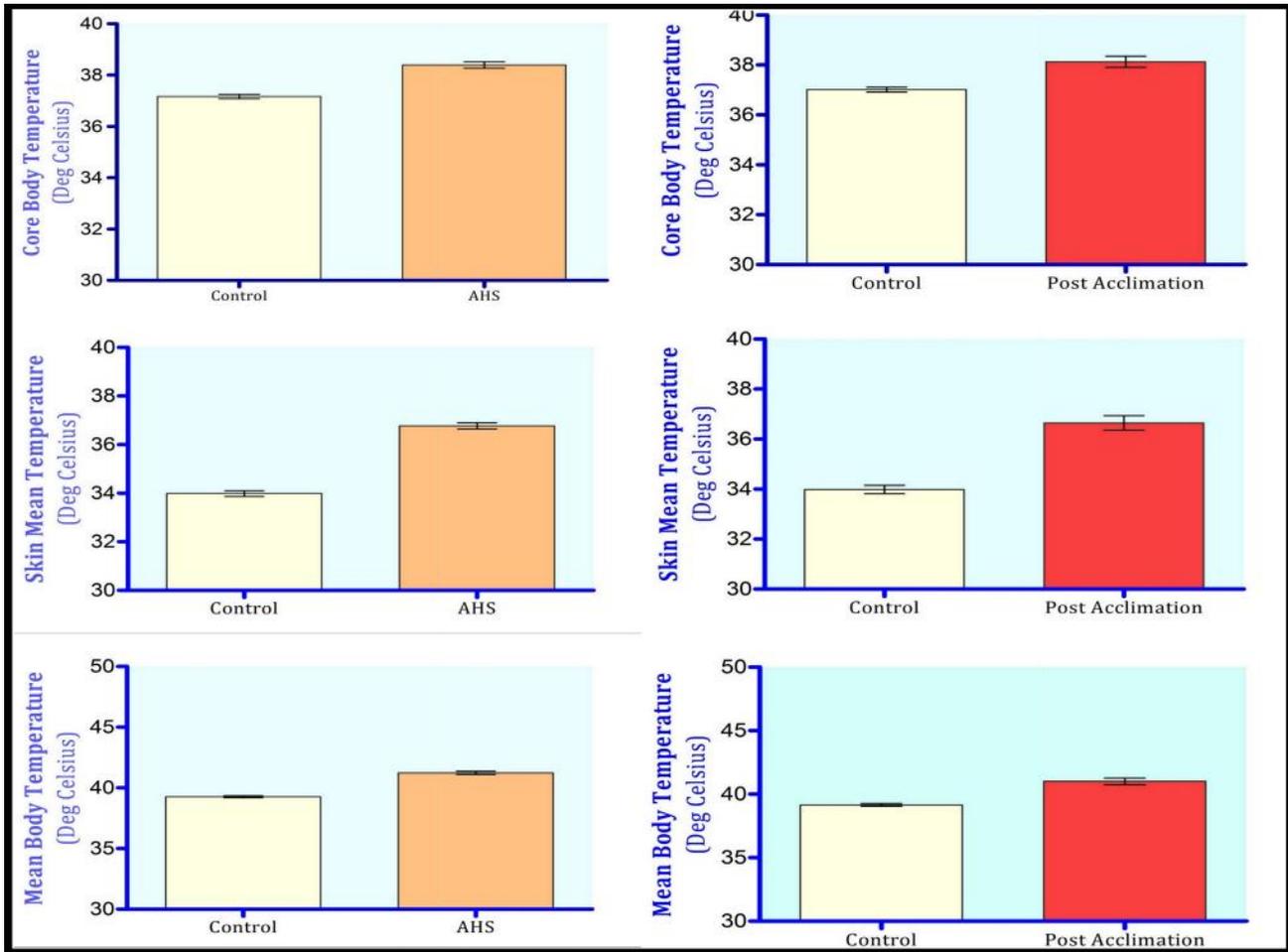
Six healthy male volunteered to complete this study [age  $31.5 \pm 1.2$  years, height  $165.8 \pm 0.9$  cm, body mass  $73.7 \pm 3.2$  kg, body surface area  $1.82 \pm 0.1$  meter square]. Inclusion criteria consisted of a medical history free of musculoskeletal, cardiac, endocrine, and heat-related illnesses. Prior to the study, all of the experimental procedure, including the risks involved, was explained to each participant. Written consent was then obtained. All protocols and procedures were approved by Institutional Human ethical committee.

### 2.2. Experimental design

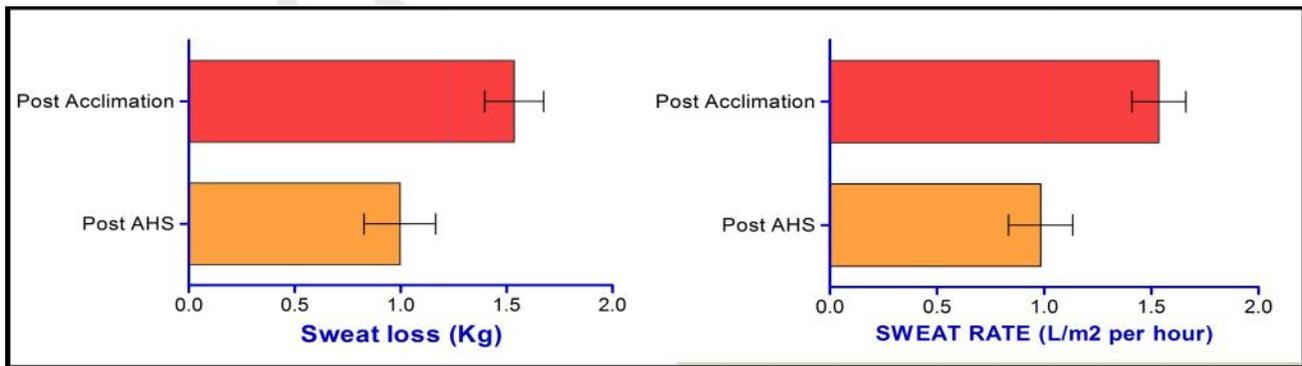
We aim to assess the influence of heat acclimation in providing tolerance towards exertional heat stress on physiological and biochemical level. The standardized step test (sub maximal exercise) was performed by the participants at 45°C, 30% RH simulated in Human Climatic Chamber (HCC) for 100 minutes for eight days to attain heat acclimation. The exercise test was separated by 10 minutes rest period. All participants were instructed not to engage in any vigorous physical activity for at least 24 h prior to each experimental test, but were to follow a normal balanced diet throughout and also, to refrain from alcohol and smoking. All studies were conducted approximately at the same time of the day for each participant to avoid variations due to circadian influences.

### 2.3. Experimental procedure

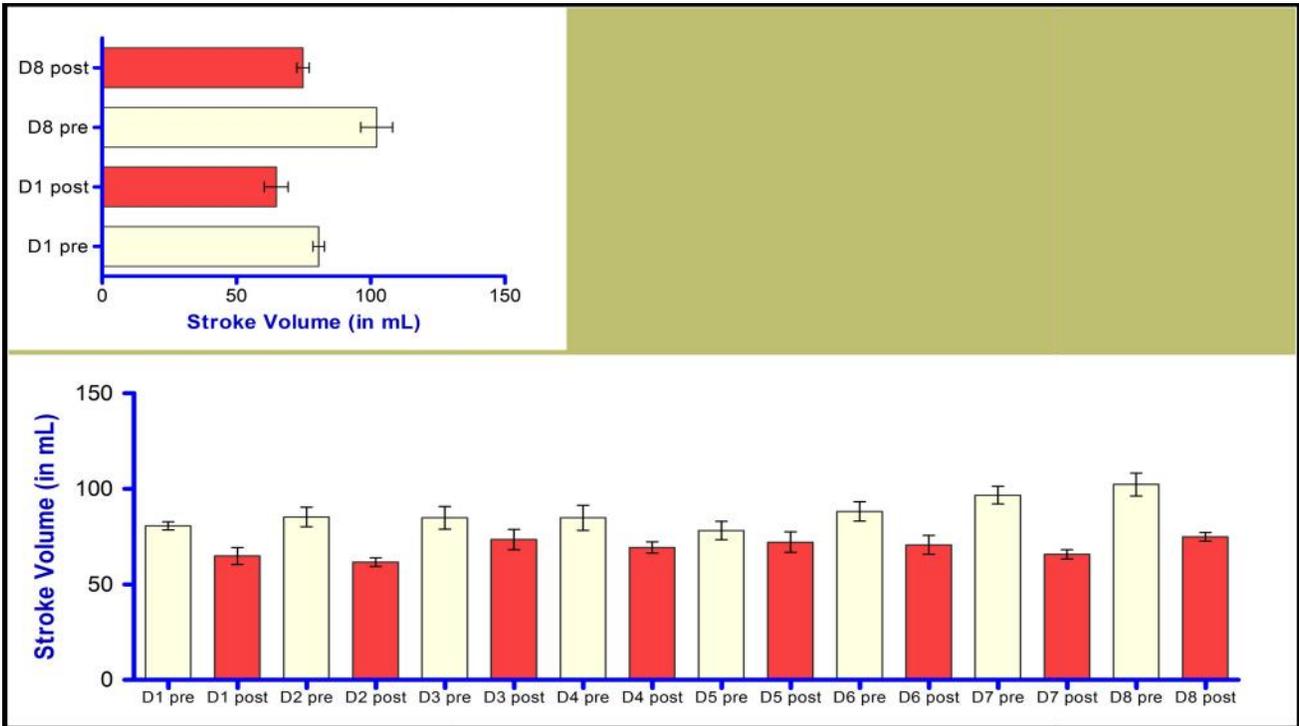
Following arrival at the laboratory, the participants were made to rest for 60 min after which electrocardiography (esaote my lab 30 gold cardiovascular) was performed. After this, the core body temperature was measured using oral temperature with correction using YSI electrodes YSI electrodes from which the core body temperature was deduced. Skin mean temperature was calculated using Ramanathan equation. Initial nude body weight was measure using digital human weighing machine model PFPF 100K and make PERFECT. After noting the control readings, the participant was sent inside the HCC which was maintained at 45°C and 30% RH and were made to do sub maximal exercise using standardized step test (15 steps/min) for 50 minutes followed by 10 minutes of rest and again 50 minutes of sub maximal exercise for eight continuous days. The same schedule as before was repeated to get post exposure data. Sweat loss was calculated by the difference in weight before and after the exposure and sweat rate was determined sweat loss per hour per square meter of the body surface area to nullify the effect of individual body structure.



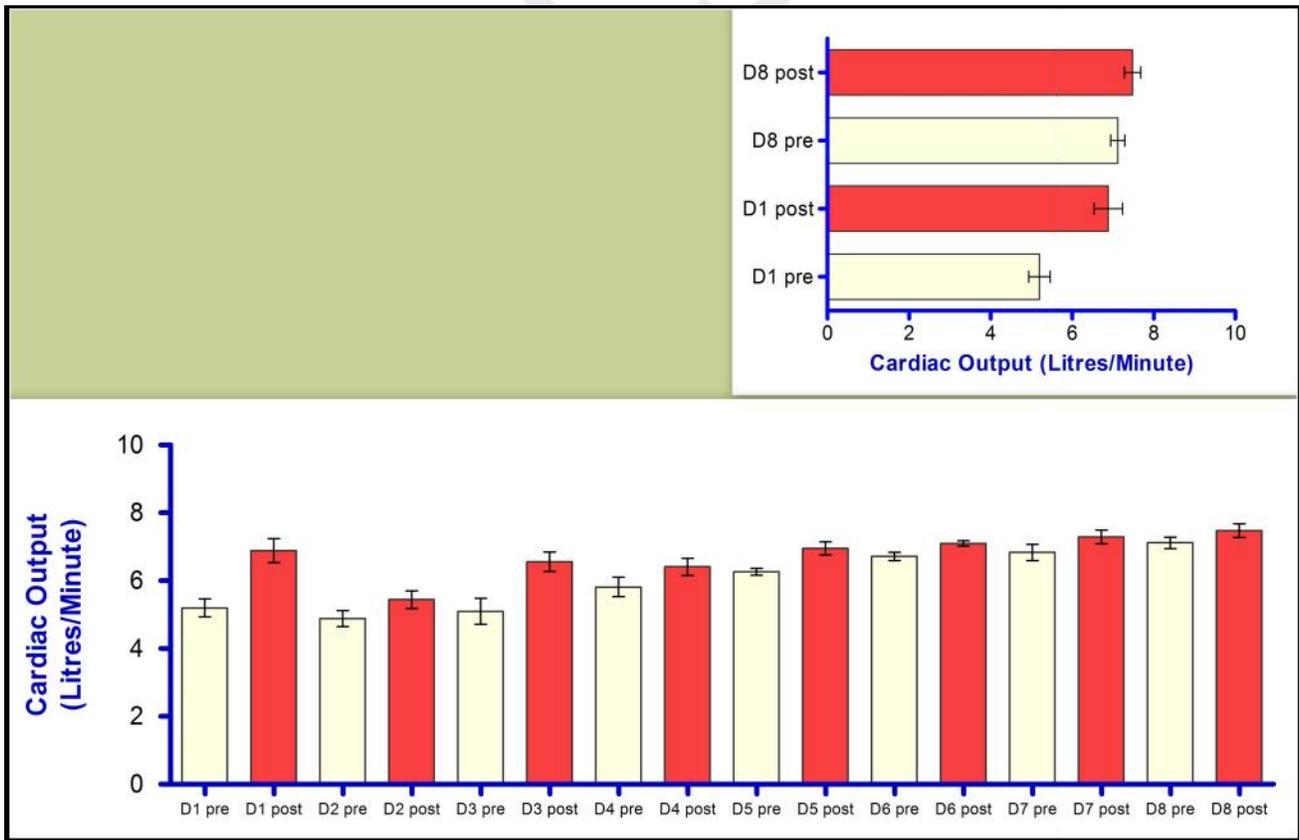
**Figure 1**  
Variation in Core Body Temperature (CBT), Skin Mean Temperature (SMT) and Mean Body Temperature (MBT) in control, acute heat stress (AHS) and Heat Acclimation



**Figure 2**  
Degree of sweat loss and change in sweat rate post acute heat stress and acclimation



**Figure 3**  
 Stroke Volume Changes



**Figure 4**  
 Cardiac Output Changes

## 2.4. 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.

## 3. RESULTS

### 3.1. Body Weight

Pre exercise and post exercise weights were  $73.89 \pm 3.3$  and  $72.83 \pm 3.2$  kg respectively with a loss of  $1.06 \pm 0.2$  kg body weight on day 1 and after acclimation i.e. pre and post exposure body weights were found to be  $73.96 \pm 3.1$  and  $72.43 \pm 3.0$  kg respectively with a loss of  $2.87 \pm 0.4$  kg of body weight.

### 3.2. Body Temperature Responses

Core body temperature was measured using oral temperature with correction factor which was found to be  $37.17 \pm 0.08$  and  $38.39 \pm 0.13$ ,  $p < 0.0001$  in normal state and after acute heat stress whereas it was found to be  $37.01 \pm 0.09$  and  $38.13 \pm 0.22$ ,  $p < 0.001$ ,  $p = 0.0008$  in normal state and Post acclimation respectively. Mean skin temperature was calculated using Ramanathan formula (Ramanathan, 1964) using 4 point measurement i.e.  $0.3 * (T_{\text{chest}} + T_{\text{biceps}}) + 0.2 * (T_{\text{calf}} + T_{\text{thigh}})$  which is found to be  $33.98 \pm 0.12$ ,  $36.77 \pm 0.12$ ,  $p < 0.001$  in normal state and after acute heat stress whereas it was found to be  $33.98 \pm 0.17$  and  $36.65 \pm 0.29$ ,  $p < 0.001$  in normal state and Post acclimation respectively. Mean body temperature is found to be  $39.26 \pm 0.08$ ,  $41.23 \pm 0.13$ ,  $p < 0.001$  in normal state and after acute heat stress whereas it was found to be  $39.15 \pm 0.10$  and  $41.00 \pm 0.26$ ,  $p < 0.001$  in normal state and Post acclimation respectively (Figure 1).

### 3.2. Sweat loss and Sweat rate analysis

Sweat loss (in kg) and sweat rate (in  $\text{kg}/\text{hour}/\text{m}^2$  BSA) were found to increase with increase in duration of exercise. Sweat loss was measured using the formula:  $\text{Sweat loss} = [(\text{nude body weight pre exercise} - \text{nude body weight after exercise}) + \text{volume of water consumed if any} - \text{volume of urine generated if any}]$  formula which was found to be  $0.99 \pm 0.16$  kg and  $1.53 \pm 0.13$  kg,  $p < 0.05$  ( $0.03$ ) post acute heat stress and post acclimation respectively (Figure 2). Similarly, sweat rate was calculated and found to be  $0.98 \pm 0.15$  kg and  $1.53 \pm 0.12$  kg,  $p < 0.05$  ( $0.018$ ) post acute heat stress and post acclimation respectively.

### 3.3. Cardiovascular Responses

Heart rate increased post acute heat stress and post acclimation which was noted as  $71.8 \pm 4.4$ ,  $147.2 \pm 9.5$  and  $141.0 \pm 5.1$ ,  $p < 0.001$  in normal state, Post acute heat stress and Post acclimation respectively. Baseline stroke volume was found to increase in a graded manner from 1<sup>st</sup> day to 8<sup>th</sup> day of the protocol. A prominent difference was observed between the stroke volume recorded post exercise 8<sup>th</sup> day in reference baseline for the same as compared to that observed for the baseline and post exercise values on the first day of the protocol (Figure 3). Similar results were found for cardiac output. Significant increase in baseline cardiac output was observed. On day 1 and 8<sup>th</sup>, baseline reading for cardiac output was found to be  $4.9 \pm 0.15$  and  $6.9 \pm 0.18$ ,  $p < 0.001$  (Figure 4). Also, the difference in cardiac output pre and post exercise on the first day was more pronounced as compared to 8<sup>th</sup> day.

## 4. DISCUSSION

The current study investigates the effect of acute exertional heat stress on cardiovascular function in Human and the impact of heat acclimation in minimizing the cardiovascular strain imposed upon acute exertional heat stress. Six human volunteers participated in the study. They were subjected to acute exertional heat stress and heat acclimatized in Human Climatic Chamber simulated at  $45^\circ\text{C}$  and 30% RH. Cardiovascular stabilization was observed upon heat acclimation to the conditions of exertional heat stress as measured upon evaluation of Heart Rate Variability, Cardiac Output and stroke Volume.

The sweat rate and sweat loss was found to increase upon heat acclimation as compared to unacclimated state, when the same magnitude and intensity of exercise was performed. Increased sweating increases body's cooling efficiency. The increase in sweating results from an increase in the sensitivity of the sweat glands for thermal stimuli after acclimation which may result from an increase in receptor density stimuli of neural or hormonal nature and/or an increase in the size and number of the number of activated sweat glands (Nielsen, 1998).

Heat acclimation resulted in an increase in baseline CO in a graded fashion as the acclimation protocol progressed day by day with an increase in stroke volume and decrease in heart rate. The increase in SV observed upon heat acclimation is attributed to an increase in plasma volume expansion, which further results in an increased cardiac output. This reflects an increased cardiac efficiency manifested in increased skin vasodilatation ability. The plasma

volume expansion is the mainstay for increased cardiovascular effectiveness. The plasma volume expansion is brought about due to greater vascular protein retention. It compensates for an increase in heart rate and a decline in SV that occurs at the time of exercise in hot environmental conditions to maintain CO (Horowitz, 2003).

The cardiovascular system, which is responsible for heat dissipation from the skin, upon acclimation, increases the volume of circulating blood with skin vasodilatation/splanchnic vasoconstriction temperature threshold when subjected to heat stress (Senay et al., 1976).

## ACKNOWLEDGMENTS

We would like to thank Dr. Pooja Chaudhary for her help and support. We would also like to thank Defence Institute of Physiology and Allied Sciences for facilitating our research work. We would like to extend sincere gratitude towards the Council of Scientific and Industrial Research (CSIR) for providing financial support.

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