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Original Research Article

The acute effects of passive heat exposure on arterial stiffness, oxidative stress, and inflammation

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ABSTRACT

Background and objective: The aim of the study was to determine the acute effect of passive heat exposure (PHE) on arterial stiffness, oxidative stress (OxS) and inflammatory parameters.
Materials and methods: Subjects were studied in thermoneutral conditions before and after PHE in a climatic chamber. Pulse wave analysis was used for assessment of central hemodynamic and arterial stiffness parameters. Venous blood samples were obtained to measure OxS and inflammatory parameters.
Results: Rectal temperature increased after PHE exposure compared to baseline: 37.01 °C ± 0.19 °C and 36.4 °C ± 0.31 °C, respectively ($P < 0.001$). There was a 17% ($P < 0.05$) decrease in large artery elasticity index (from 24.68 ± 5.53 to 20.42 ± 2.65 mL/mmHg*10), which was predicted upon normothermic value ($r = -0.878$, $P < 0.01$). However, no significant changes were found in others arterial stiffness parameters. A 30% ($P < 0.05$) increase occurred in blood IL-6 concentration (from 0.43 ± 0.15 to 0.56 ± 0.23 pg/mL), but OxS parameters remained significantly unchanged.
Conclusions: This study describes for the first time acute PHE effects on arterial stiffness, inflammation and OxS. PHE significantly decreases large artery elasticity index and increases inflammatory IL-6 level. However, further larger investigations are needed for clarifying acute PHE effects on arterial function and biomarkers.

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1. Introduction

Exercising in a hot environment is a stressful challenge for the human body. In our previous studies, we have shown the positive effects of heat acclimation on arterial stiffness [1], oxidative stress (OxS) and inflammation parameters [2]. However, the heat acclimation protocol employed in these studies [1,2] combined both exercise and heat stress. Therefore, it was not possible to evaluate the potential impact of heat exposure independently from exercise on the changes observed in arterial stiffness and the markers of OxS and inflammation.

To our knowledge, there are very few reports to date, which provide information regarding the effect of passive heat stress on arterial stiffness and the data are contradictory. It has been demonstrated in vitro that heating of isolated arteries produces thermal-induced decreases in vessel stiffness [3]. Furthermore, it has been shown in a study conducted in humans that acute local thermal therapy may result in a decrease in arterial stiffness in both healthy young and older women [4].

On the contrary, Moyen et al. [5] showed slightly increased central arterial stiffness with heating and found that baseline stiffness appears to mediate the magnitude of heating-induced changes in arterial stiffness, while peripheral arterial stiffness remained unchanged.

Furthermore, Ganio et al. [6] studied the effect of passive heat stress on arterial stiffness and concluded that an increase in core temperature induced by passive heating does not affect arterial stiffness.

However, Ganio et al. [6] showed that the magnitude by which heat stress non-significantly decreased individual arterial stiffness was predicted upon the normothermic value – individuals who had stiffer arteries were more responsive to heat exposure induced improvements.

There is a lack of information regarding the impact of heat stress without exercise on OxS in humans. Heat stress is suggested to be an environmental factor responsible for stimulating reactive oxygen species production. We have previously shown that exhausting endurance exercise in the heat increases OxS level [2]. Data from an animal study by Yang et al. [7] showed that acute exposure to high temperatures may result in increased OxS and others [8] suggested that OxS should be considered a part of the stress response to heat exposure.

There is also a lack of information regarding the impact of heat stress on inflammation.

During prolonged exercise with or without heat stress, the level of inflammatory cytokines increases, heat exposure tends to stimulate the release of IL-6 [9]. Human studies have shown that heat stress increases plasma IL-6 values and plasma IL-6 concentrations have been previously shown to be positively correlated with increase in core temperature [10].

Thus, considering the paucity of relevant literature and the discrepancy in the data available, the aim of this study was to determine the acute effects of passive heat exposure (PHE) on arterial stiffness, OxS and inflammatory indices in young healthy men.

2. Materials and methods

2.1. Ethical approval

The study was approved by the Research Ethics Committee of the University of Tartu. Prior to the beginning of the study, all subjects gave their written informed consent. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

2.2. Subjects and study design

The present study constitutes one part of a complex experimental heat acclimation study [1,2].

The sample consisted of 9 physically active men (age 28.78 ± 3.35 year; height 182.33 ± 4.64 m; weight 78.88 ± 11.15 kg; peak oxygen uptake 53.58 ± 7.92 mL/kg/min; heart rate 48.83 ± 9.93 beats/min; systolic blood pressure 117.22 ± 8.69 mmHg; diastolic blood pressure 66.00 ± 7.35 mmHg).

None of the subjects took any medication, none of them were smokers nor had a history of heat illness. Two months prior to participation in this study, the subjects were instructed to avoid any use of additional food supplements, to follow a healthy diet and keep it stable. The study was conducted during the winter period in Estonia, which is situated between 57°37′ and 59°49′ of north latitude. Parameters of arterial stiffness, OxS and inflammation were measured in thermoneutral conditions (22 °C; relative humidity 35%) twice: before and after PHE in a climatic chamber.

During PHE the subjects stayed in a sitting position for 110 min in a climatic chamber. After the exposure, they walked out of the chamber (distance 10 m) after which the body mass of the subjects was measured. In thermoneutral conditions the procedures of measurement of arterial stiffness were conducted after a period of 10 min in supine position in order to ensure the standardization of physiological parameters. After the measurement of arterial stiffness, the blood samples for parameters of oxidative stress and inflammation were taken.

2.3. Passive heat exposure

The subjects stayed in a sitting position for 110 min in a climatic chamber (Design Environmental Ltd., Gwent, South Wales, UK) maintained at a high temperature (42 °C; relative humidity 18%), which was set up accordingly to the previous study where combined heat and exercise regimen was used [1,2]. It is suggested that core body temperature is a factor participating in the induction of OxS and inflammation and has an effect on arterial stiffness. Subjects' core body temperature was monitored in real time using a rectal probe (TX-2, Columbus Instruments, Columbus, OH, USA) and the values before and immediately after they entered/exited the climatic chamber were recorded by means of an electronic data logger (Iso-Thermex 256, Columbus Instruments, Columbus, OH, USA). Body mass of the subjects was measured to the nearest 0.001 kg before and after passive heat exposure, using an electronic scale (CH3G-150I Combics, Sartorius AG, Göttingen, Germany).