

# THE BENEFICIAL EFFECTS OF HEAT ACCLIMATION AND EXERCISE TRAINING ON CARDIAC PERFORMANCE AND CARDIOVASCULAR EFFICIENCY IN STRESSFUL ENVIRONMENT

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## INTRODUCTION

Exposure to chronic adverse environmental conditions, or to chronic physiological insults changes the body responses to further insults. Depending on the adaptive requirements of each of the individual stressors, upon subjection to a multimodal stress, expression of an adaptation is either reinforcement or underexpression of each feature. A call for opposite responses, which produces adaptive "conflicts", may also lead to exclusion of features from the acclimatization/ acclimation repertory. Taken together, however, adaptations lead to a better performance under the stress. It is now clear that both intrinsic and extrinsic (e.g. intracellular vs extracellular) adjustments play key roles in the adaptation process (1).

For example, **heat acclimation and exercise training**, individually, as well as in conjunction, improve cardiovascular performance in both normothermic and hot environments. This is manifested by decreased resting heart rate (2), increased cardiac reserves and greater peripheral blood flow and volume (2). However, while both stressors induce bradycardia and a larger blood volume, heat acclimation leads to improved cardiovascular reserves in the face of decreased metabolic rate whereas exercise training results in improved cardiovascular reserves coinciding with increased metabolic rate (3). It is difficult, therefore, to interpret whether the improved cardiac/cardiovascular performance obtained following the combined chronic stress of heat acclimation and exercise training is attributable to both or to either one of the individual stressors. Furthermore, since both heat acclimation and exercise training induce changes in the cardiac muscle and blood vessels, as well as in body fluids volume (4), upon cross adaptation, it is difficult to evaluate 1. the relative importance of the intrinsic and extrinsic factors in the improvement of cardiovascular performance (e.g. changes in ventricular preload-namely Starling low, vs changes in myocardial contractility) 2. the mode of interaction between the two stressors and whether there additive, interference or exclusion effects.

The major goal of this presentation is to discuss these issues. Based on animal experimental models and data derived from human studies, emphasis will be given to cardiac performance.

## **INCREASED CARDIAC EFFICIENCY - A LESSON FROM WHOLE BODY RESPONSES**

Although the integrative response to heat acclimation/exercise training is well documented, the circulatory mechanisms leading to this complex adaptation and the individual impacts of each stressor are not well understood; particularly in light of newly accumulating evidence on the roles played by cellular processes in this adaptation.

A manifestation of the outcome of heat acclimation and exercise training on cardiac work during exercise at various levels of heat loads in the rat model is illustrated in Fig. 1. It is evident that at the same work load, both exercise training and heat acclimation attenuated the increase in cardiac work (double product) compared to the untrained/non-acclimated rats. This suggests an increased efficiency of the system. In the experimental paradigm employed it was hard to pinpoint whether changes in the myocardium *per se* have occurred. While greater efficiency in the trained groups was achieved primarily by changes in heart rate, in the heat acclimated groups this effect was obtained by a decrease in blood pressure (decreased total peripheral resistance) and heart rate (3). Collectively, in this model effects of heat acclimation predominates.

There are differences between rats and humans in their response to heat stress/heat acclimation and exercise training. Nevertheless, the global stressors-induced outcome - increased efficiency of cardiac performance is similar for both species. For human subjects this was clearly demonstrated by Rowell et al., (5), in a group of subjects subjected to 14 days of heat acclimatization. Acclimatization led to a marked decrease in heart rate. In turn, stroke volume increased, leading to maintenance/increase in cardiac output at a greater efficiency. Similar findings were observed by other investigators.

It is well documented that stroke volume rises during acclimation whether or not plasma volume increases. This suggests that factors other than increased cardiac filling pressure are part of the adaptation repertoire. Indeed, improved intrinsic contractile properties have been inferred in human subjects (6) as well as in some animal models (7) upon exercise training. Although previous reviews (2,8) stated that during exercise training "there is little to be gained in overall cardiac performance from any further increase in myocardial contractility". The relative importance of these changes are not yet evaluated. Little is known about the adaptation of the myocardium to chronic heat and exercise training. The data, mostly from animal experimental models, suggest that improved myocardial contractility contributes to cardiac efficiency. Such an adaptive feature might be of prime importance upon endurance training in adverse environments.

Taken together, the newly accumulating data on exercise/heat-induced intrinsic changes leading to improved cardiac contractility requires reconsideration of their relative importance in the adaptive repertoire.

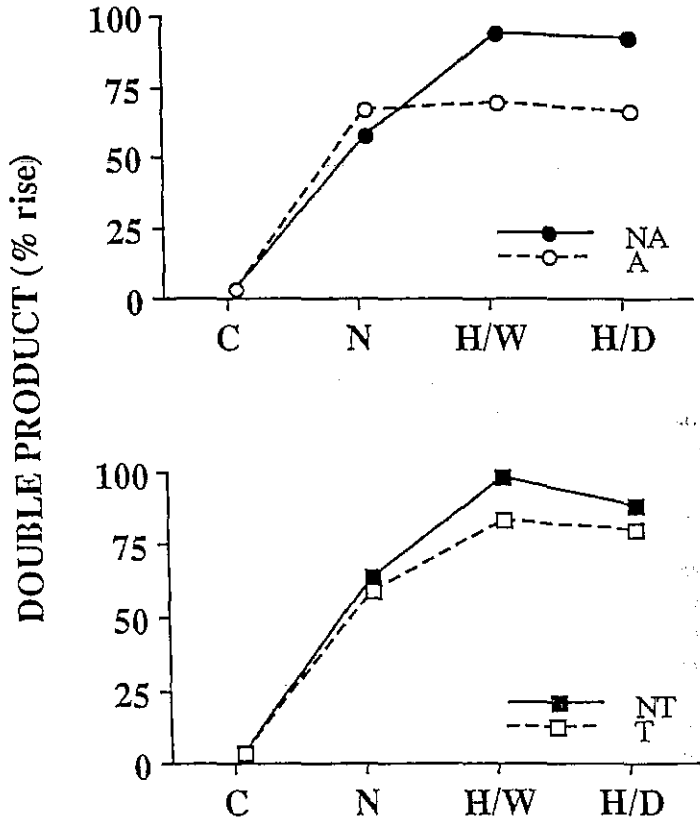


Figure 1. Cardiac work, expressed as the double product, in mixed groups of heat acclimated and exercise trained rats during 20 min of mild treadmill exercise (15m/min,  $VO_2$  50 ml/min·kg<sup>-1</sup>) at two different heat loads: hot/wet: 35°C, 70%, R.H. hot/dry 40°C, 20% R.H. Normothermic conditions were 24°C.

Upper panel: Non-acclimated (NA) and heat acclimated (A) rats.  
 Lower panel: Untrained (NT) and trained (T) rats.

Group A contained both trained and untrained heat acclimated rats whereas the trained group T contained normothermic and heat acclimated trained rats. Likewise, group NA contained sedentary and trained normothermic rats whereas NT contained sedentary normothermic and heat acclimated rats. Data of groups A and T differed significantly from their matched controls, NA and NT respectively ( $p < 0.05$ ). Data were adapted from reference 3.

## **INCREASED MYOCARDIAL EFFICIENCY - A LESSON FROM ISOLATED HEART PREPARATIONS**

Isolated hearts and single myocyte models may be of significant value in gaining deeper insight into variety of mechanisms underlying training or heat acclimation induced improvement in cardiac function. When taken together with data derived from whole body studies, the gained knowledge assists us to evaluate the relative importance of these adaptations in the overall improved cardiovascular reserves. A detailed review of this aspect is beyond the scope of this article. Briefly, both heat acclimation or exercise training induce greater pressure generation due to improved handling of cytosolic  $Ca^{2+}$  required for contraction. For example, augmented peak calcium transients due to modulation of several sarcolemmal and sarcoplasmic reticulum transporting proteins take place (7,9,10). There are, however, cardinal differences between hearts from heat acclimated and exercise trained animals. A major manifestation of this difference is different rates of pressure development and relaxation. Heat acclimation attenuates whereas exercise training accelerates these rates, due to different myosin isoenzymes profile. Heat acclimated hearts with the slow myosin ( $V_3$ ) fail to reconstitute pressures at high beating rates whereas exercised trained hearts with the fast myosin ( $V_1$ ) show higher energy expenditure compared to the heat acclimated hearts (11). Despite these differences, heat acclimation, when combined with exercise training, produces a favorable additive effect on the mechanical and metabolic properties of rat hearts, compared with the effects of exercise training or heat acclimation alone. This is expressed by greater pressure generation and increased mechanical efficiency compared to that observed by hearts from heat acclimated or exercise trained animals [heart weight]/[body weight] ratio does not differ significantly from non-acclimated hearts indicating that the increased contractile performance was not due to a greater muscle mass. It is likely that greater pressure generation is associated with improved handling of cytosolic  $Ca^{2+}$ , yet, neither of these mechanisms were studied. The improved pressure generation is accompanied also by greater efficiency which is exemplified by a left shift of the rate pressure product -  $O_2$  consumption relations (11). Increased efficiency together with a wider range of pressure augmentation due to intrinsic adaptations then, appear to be a principal adaptation to a combined stress of heat acclimation and exercise training.

### **IN SUMMARY**

Some evidence is now available that heat acclimation in combination with exercise training provides beneficial additive effect in terms of improved cardiac contractility coincidentally with greater efficiency. This leads to increased cardiovascular performance and increased endurance upon hyperthermia resulted by environmental and metabolic heat stress.

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