HEAT ACCLIMATION: PHENOTYPIC PLASTICITY AND CUES TO THE UNDERLYING MOLECULAR MECHANISMS

M. Horowitz, Department of Physiology, The Hebrew University, Hadassah Medical School, Israel.

Acclimation, in contrast to evolutionary adaptation, is a within life-time phenotypic adaptation, although it may involve a genetic basis. The phenotypic expression of heat acclimation comprises lowered metabolic rate, body temperature (Tb), and heart rate, concomitantly with greater cardiovascular reserves, increased efficiency and capacity of the evaporative cooling system and elevated temperature threshold for thermal injury. Collectively these lead to an “expanded regulatory capacity” within the range of safe Tb. Our current data imply that reprogrammed gene expression and changes in cellular signaling underlie the acclimatory phenomenology(4). An important adaptive feature associated with elevated threshold for thermal injury is enhanced cytoprotection. Among cytoprotective mechanisms, the inducible heat shock protein (HSP 72 kDa) was the most thoroughly studied. Acclimation leads to 200% elevation of the constitutive level of this protein, thus providing protection with out the need for *de novo* HSP synthesis upon stress. Acclimation also predisposes the signaling pathway for HSP synthesis to respond faster to heat stress, at the transcription level (3). Whether these two phenomena are interdependent is not yet understood, although other gene products follow similar pattern. There is evidence that the time window for the changes observed in the machinery of HSP induction is at the early phase of heat acclimation (1-2 acclimation days), involving accelerated excitability of the sympathetic system. The increased cardiovascular reserves constitute intrinsic changes both in the vasculature, e.g. augmented nitric oxide synthase (eNOS) level and altered G proteins function, and in the heart, e.g. altered expression of the contractile, E-C coupling and calcium regulatory proteins (1,2, Cohen and Horowitz, in preparation). These lead in the acclimated heart to greater pressure generation at lowered oxygen consumption, enhanced positive inotropic response and improved chamber compliance, thus matching cardiac function to greater venous return occurring upon heat acclimation. A heat acclimation-induced drop in plasma thyroxin level is responsible for many of the changes observed. An important consequence of thermal acclimation is the development of cross-tolerance between heat acclimation and impaired oxygen demands/oxygen supply balance. The beneficial implications of this feature will be discussed.

Eynan, M., Palmon, A., Hasin, Y., Horowitz, M. (1998) Heat acclimation induces changes in cardiac mechanical performance: the role of thyroid hormone. Am. J. Physiol. 276, R550-558.

Eynan M, Gross, H., Hasin, Y., Palmon, A., Horowitz, M. (2000) Temporal changes in heart mechanics with heat acclimation: Adrenergic signaling and SR-Ca regulatory proteins Am. J. Physiol. 279: R77-R85.

Maloyan, A., Palmon, A., Horowitz, M. (1999) Heat acclimation increases basal HSP72 level and alters its production dynamicsduring heat stress. Am J. Physiol. 276, R1506-R1515

Horowitz M (2001) From Molecular and Cellular to Integrative Heat Defense during Exposure to Chronic Heat. Comp Biochem Physiol.

horowitz@cc.huji.ac.il