DECREASED ACTIVE CUTANEOUS VASODILATATION IN AGED SKIN: MECHANISMS, CONSEQUENCES, AND INTERVENTIONS

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Despite early reports to the contrary, it is now well recognized that aged men and women respond to heat stress with an attenuated increase in skin blood flow (SkBF). While other human thermoregulatory adjustments and responses to hyperthermia are highly dependent on other factors -- aerobic capacity (VO2max), acclimation status, hydration, diseases and medications -- the relative inability of aged skin to vasodilate appears to be a primary consequence of advanced age. For example, while a healthy acclimated 65-year old athlete will respond to exercise in a hot environment with a similar increase in body core temperature and sweating rate as a VO2max-matched 30-year old, SkBF may be 30-50% lower. Over the past 10 years we have examined the neural and cardiovascular mechanisms underlying and supporting this age-related decrement, the potential health-related consequences of the integrated cardiovascular response of older individuals exposed to heat stress, and interventions which have been shown to increase cutaneous vasodilatation in the elderly. Elimination of sympathetic cutaneous vasoconstriction via either alpha-adrenergic receptor blockade or by preventing local release of norepinephrine does not selectively increase SkBF in heat-stressed older human subjects. Rather, structural changes in aged skin coupled with a decreased active vasodilator sensitivity account for the altered control of SkBF. The potential role of nitric oxide-mediated mechanisms is currently under investigation. On the supply side, the lower SkBF response of the elderly is accompanied by both a smaller increase in cardiac output and a lesser redistribution of flow from splanchnic and renal circulations. The relative inability to maintain stroke volume in light of a falling central venous pressure and attenuated increase in cardiac output may be secondary to the decreased beta-adrenergic sensitivity which accompanies aging. In the healthy older population, aerobic conditioning, heat acclimation, and, in the case of postmenopausal women, unopposed exogenous estrogen have all been shown to increase SkBF at a given core temperature. While the primary purpose of increasing human SkBF in hyperthermic conditions is to transfer and dissipate heat, the principle challenge of aged individuals exposed to heat stress is not to thermal homeostasis, but rather to cardiovascular homeostasis.

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