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HUMAN RESPONSES TO EXERCISE-HEAT STRESS

by

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This report provides a comprehensive overview of the normal human physiological responses to environmental and exercise-related heat stress, with emphasis placed on acute heat exposure and acclimation produced by repeated heat exposure. In addition, the report reviews biomedical factors that modify exercise-heat tolerance such as aerobic fitness, dehydration, circadian patterns and sleep loss and medications. Also, the report reviews thermoregulatory responses of special populations such as women, blacks, children, older adults and spinal cord injured populations.

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EXECUTIVE SUMMARY

This technical report provides a comprehensive overview of the normal physiological responses to environmental and exercise-related heat stress, with emphasis placed on acute heat exposure and acclimation produced by repeated heat exposure. Environmental heat stress increases the requirements for sweating and circulatory responses to dissipate body heat; an environment that is warmer than the skin also causes the body to gain heat from the environment, and thus increases the amount of heat that the body must dissipate. In addition, muscular exercise increases metabolic rate, and thus also increases the rate at which heat must be dissipated to the environment to keep core temperature from rising to dangerous levels. Environmental heat stress and muscular exercise therefore interact synergistically, and may push physiological systems to their limits. To regulate body temperature, heat gain and loss are controlled by the autonomic nervous system through adjustment of: a) heat flow from the core to the skin via the blood; and b) sweating. The hypothalamic thermoregulatory center receives information from thermoreceptors in the skin and body core, and processes this information via a proportional control system to generate a signal for heat loss, by the thermoregulatory effector responses of sweating and skin blood flow. Repeated heat exposures that are sufficient to raise core temperature, result in adaptations which reduce the physiological strain produced by such exposure. The primary biomedical factors influencing effectiveness of the thermoregulatory system in defending body temperature are the individual's acclimation state, aerobic fitness, hydration level, sleep status, circadian timing, health and medication. In addition, such factors as gender, race, age, and certain medical disorders—including skin disease and spinal cord injury—affect thermoregulatory responses.

INTRODUCTION

Environmental heat stress increases the requirements for sweating and circulatory responses to dissipate body heat; when the environment is warmer than the skin, it also causes the body to gain heat from the environment, and thus increases the amount of heat that the body must dissipate. In addition, muscular exercise increases metabolic rate above resting levels, and thus also increases the rate at which heat must be dissipated to the environment to keep core temperature from rising to dangerous levels. Environmental heat stress and muscular exercise therefore interact synergistically, and may push physiological systems to their limits. This technical report provides an overview of the normal physiological responses to environmental and exercise-related heat stress, with emphasis placed on acute heat exposure and acclimation produced by repeated exposure to heat stress. More complete reviews of some material covered in this technical report can be found in other recent reviews (4,129,168,195,197,198,219,240,254).

HEAT STRESS

Humans are tropical animals, and it is primarily through cultural and behavioral means that they have adapted to life in temperate and cold environments (60,123,137). This generalization is based on the following evidence: (a) humans rely primarily on physiological thermoregulation in the heat, but primarily on behavioral thermoregulation in the cold; (b) the thermoneutral ambient temperature for nude humans and the temperature necessary for undisturbed sleep are relatively high (≈27°C); and (c) humans demonstrate substantial heat acclimatization, but only modest cold acclimatization.

Hot climates are present over large areas of the earth and are tolerated well by humans. **Figure 1** is a global map of Wet Bulb Globe Temperature (WBGT)[1] during July, the hottest month in the northern hemisphere (4). During July much of North America, South America, Europe and Asia have WBGT values above 29°C (85°F).

[1] WBGT is a computed index of environmental heat stress. Outdoors WBGT = 0.7 wet bulb + 0.2 black globe + 0.1 dry bulb; indoors WBGT = 0.7 wet bulb + 0.3 black globe

Depending on the climate, such high WBGT values can be achieved either through high humidity, as reflected in high wet bulb temperature, or through high air (dry bulb) temperature and solar load, as reflected in black globe temperature.

[INSERT FIGURE 1 HERE]

Given access to shade and adequate water, healthy persons can tolerate extended exposure to virtually any climatic heat stress. By contrast, many occupational situations involve heat-stress conditions so severe they cannot be tolerated for extended periods. Such conditions typically involve WBGT values above 35°C (115,136) and are encountered in deep mining, boiler room work and fire fighting, as well as when wearing protective clothing in hot environments. The safety of persons working in these conditions depends on either observing strict limits on exposure time (154), or the use of microclimate cooling (215).

THERMOREGULATORY CONTROL

In contrast to the broad temperature range in places of human habitation, human body temperature is usually regulated within a narrow range (35° to 41°C). This is accomplished through two parallel processes: behavioral temperature regulation and physiological temperature regulation. Behavioral temperature regulation operates largely through conscious behavior, and may employ any means available. Physiological temperature regulation operates through responses that do not depend on conscious voluntary behavior, and includes control of: (a) rate of metabolic heat production, (b) heat flow via the blood from the core to the skin, and (c) sweating.

Physiological control systems can produce graded responses according to the disturbance in the regulated variable, in this case core temperature. Usually, the magnitudes of changes in responses (e.g., sweating, skin blood flow) are approximately proportional to displacement of the regulated variable from some basal value, and such control systems are called "proportional-control systems". For convenience body thermal receptors can be grouped into those located in core and those in skin, but it should be remembered that neither the core nor the skin has a uniform temperature The thermal receptors in the core are unevenly distributed, and

have been shown in laboratory mammals to be concentrated especially in the hypothalamus, where much of the integration also occurs. Temperature changes of only a few tenths of 1°C in the anterior preoptic area of the hypothalamus elicit substantial changes in the thermoregulatory effector responses (21). Outside the hypothalamus, core sites that may have thermal receptors include the heart, major blood vessels, and spinal cord (20,48,231). **Figure 2** shows how reflex control of three heat-dissipating responses—sweating, skin blood flow, and forearm venous volume—depends on body core temperature (represented by esophageal temperature) and mean skin temperature (189,241,243). At any given skin temperature, each response is approximately proportional to esophageal temperature; and increasing the skin temperature lowers the threshold level of esophageal temperature for the response, thus increasing the response at any given core temperature.

[INSERT FIGURE 2 HERE]

The most accurate mathematical representation of reflex control of thermoregulatory effector responses is probably a complicated function of different tissue temperatures with some non-linear elements; however, it is convenient to represent the control in terms of a linear combination of core temperature (T_c) and mean skin temperature ($\bar{T}_{sk}$), and to represent the control of each thermoregulatory effector response by an equation like the following:

$$R - R_o = \underline{a}_1 \cdot T_c + \underline{a}_2 \cdot \bar{T}_{sk} + \underline{b}$$

where R is a thermoregulatory response;

R_o is a basal value of R;

and $\underline{a}_1$, $\underline{a}_2$, and $\underline{b}$ are constants.

In terms of Figure 2, $\underline{a}_1$ is the slope of a particular R:T_c relation and $\underline{a}_2$ is the effect of a unit change in $\bar{T}_{sk}$ on the R:T_c relation. The ratio $\underline{a}_1$:$\underline{a}_2$ is about 9:1 for the heat-dissipating responses of sweating (130), "active" vasodilation (see below) as represented by forearm blood flow (243), and vasodilation through release of vasoconstriction as represented by finger blood flow (242). For control of forearm venous volume, reported values of the ratio $\underline{a}_1$:$\underline{a}_2$ are substantially smaller than 9:1 (241), but the physiological significance of this difference is not clear.

The coefficients $\underline{a}_1$ and $\underline{a}_2$ represent the sensitivity of R to changes in T_c and $\bar{T}_{sk}$, respectively, and the ratio of 9:1 means that a change of 1°C in core temperature elicits about nine times as great a change in thermoregulatory response as does a 1°C change in mean skin temperature. However, since skin temperature generally varies over a wider range than does core temperature, the importance of skin temperature in thermoregulatory control is greater than what the 9:1 ratio might suggest. The responsiveness of the thermoregulatory system to core temperature changes is necessary if the thermoregulatory system is to keep core temperature relatively constant, and the system's sensitivity to mean skin temperature allows the system to respond appropriately to large changes in environmental temperature while permitting little change in body core temperature.

The function of the human thermoregulatory system is shown schematically in **Figure 3** (198). This scheme presumes that there are thermal receptors in the core and skin that send information about their temperatures to some central integrator, which generates a "thermal command signal" that participates in the control of sweating, vasodilation, and vasoconstriction. The notion of such a thermal command signal is supported, first, by observations that for the thermoregulatory control of any one of the heat-dissipating responses, the ratio of the contributions of core and skin temperature inputs is the same as for the others; secondly, by observations that thresholds for different thermoregulatory responses are shifted simultaneously, and to a similar degree, by factors such as circadian rhythms, fever, phase of the menstrual cycle, and heat acclimation (77). It is useful to think of such similar and simultaneous shifts in thresholds for a number of different thermoregulatory responses as representing (or as being the result of) a shift in thermoregulatory "set point". The notion of such a thermoregulatory set point is made more intuitively comprehensible by the fact that changes in set point are reflected in similar-size changes in core temperature at rest.

[INSERT FIGURE 3 HERE]

Since skin temperature generally is not uniform, the contribution of skin temperature to thermoregulatory control is usually expressed in terms of an appropriately weighted mean skin temperature. Neurophysiological studies show three

types of thermal receptors in skin (82,90), one type responding to heating with a transient burst of activity and an increase of static activity; one type responding to cooling with a transient burst of activity and an increase of static activity, and one type responding to several stimuli, including warming, with a continuous discharge. The transient discharges that occur at the beginning of heating or cooling give the central integrator information about changes in skin temperature as they are occurring, and this feature may account for the sensitivity of sweating not only to skin temperature, but also to the rate at which skin temperature is changing (131)

CORE TEMPERATURE

Measurement of Core Temperature

Fundamental to the experimental study of human temperature regulation is the measurement of body core temperature. Core temperature is measured either to estimate average internal temperature to compute changes in heat storage in the core, or to provide an estimate of the core temperature input to thermoregulatory control. There is no one "true" core temperature because of temperature difference among different sites in the core. However, temperatures at all core sites are close to (within about 1°C) central blood temperature at thermal steady state. An ideal site to measure core temperature should be convenient and unbiased by environmental conditions, and should rapidly and quantitatively reflect small changes in central arterial blood temperature. Core temperature is often measured at the esophagus, rectum, mouth, tympanum, and auditory meatus.

Esophageal temperature is obtained with a sensor passed through the nose and throat into the esophagus to the level of the left atrium, where the heart and esophagus are in contact and virtually isothermal for several centimeters (22). Esophageal temperature responds rapidly (time constant ~ 1 min) and quantitatively to changes in central blood temperature (41,158,208), and most thermal physiologists consider esophageal temperature to be the best non-invasive index of core temperature for humans. Rectal temperature is measured from 5 to 20 centimeters past the anal sphincter, since temperature is relatively uniform in that region (16,101,139). Rectal temperature typically is higher than temperatures at other core

sites, and responds more slowly (time constant ≈ 12 min) to rapid transients in core temperature, e.g., during exercise (7,158,176,177), probably because of a comparatively low rate of blood flow (78,127). However, steady-state rectal temperature is a good index to assess body core heat storage (177,225). Oral (sublingual) temperature is widely used clinically, but less commonly in physiological research (222). The tongue's high blood flow per gram of tissue (85) makes it an effective heat exchanger with central blood. However, oral temperature is sometimes biased by head and face skin temperatures (126), and may be artificially lowered by evaporation if the subject breathes through the mouth.

Tympanic temperature is obtained with a sensor resting against the tympanic membrane. Some subjects find the sensor, when properly placed, to be uncomfortable (22), and occasionally, the sensor may perforate the tympanic membrane (226,235). Because of these problems, some investigators have instead chosen to measure the temperature of the external auditory meatus. Placement of the sensor is important since there is a substantial (≈0.5°C) temperature gradient along the wall of the meatus (40). To avoid the direct influence of ambient air temperature on the sensor, the meatus should be plugged or covered when obtaining either tympanic or auditory meatus temperatures. Tympanic and auditory meatus temperatures respond to core temperature transients more rapidly than rectal temperature (time constant for tympanic temperature ≈ 4 min), though not so rapidly as esophageal temperature (158). A major attraction of tympanic temperature has been the notion that, because of the proximity of the external auditory meatus to the cranium, tympanic temperature is uniquely suited to serve as a noninvasive measure of intracranial temperature (17).

The importance of this supposed advantage for tympanic temperature is increased, in the view of some investigators, in light of the notion that the brains of hyperthermic humans are selectively cooled, as blood enroute to the brain is cooled in the head and neck by heat exchange with cool venous blood (29). If this latter notion is correct, then the brains of hyperthermic humans must be cooler than the arterial blood in the trunk, so that measurements of trunk core temperature, such as rectal or esophageal temperature, are not valid indices of brain temperature under such conditions. It is proposed that tympanic temperature reflects brain temperature rather

than trunk core temperature, and therefore, under these conditions, is superior to rectal or esophageal temperature as an index of brain temperature (see Reference #28, for a discussion). Evidence cited in support of the concept of selective brain cooling in hyperthermic humans consists of measurements of tympanic temperature that are lower than measurements of trunk core temperature taken at the same time. Such an interpretation depends critically on the assumption that when tympanic temperature differs from trunk core temperature in approximately steady-state thermal conditions[2], tympanic temperature is reliably the better index of brain temperature. The truth of this assumption has never been established. In fact, direct measurement in one surgical patient indicates that esophageal temperature reflects brain temperature considerably better than does tympanic temperature (209). In addition, tympanic and auditory meatus temperatures may, depending on environmental conditions, be either lower or higher than simultaneously measured steady-state rectal (80,132) and esophageal (124,125,126) temperatures. This is because local heating or cooling of the head surface affects the temperature of the tympanic membrane (98) and external meatus (124,125,126). Brengelmann (22) provides a critical review of tympanic temperature as a measure of brain and core temperature.

Core Temperature Responses to Exercise

During exercise, the rate of heat production increases above its level at rest because of heat produced as a by-product of skeletal muscle contraction. The increase in heat production occurs almost immediately at the onset of exercise, so that during the early stages of exercise, rate of heat production exceeds rate of heat dissipation, and the difference is stored as heat, primarily in the core, causing core temperature to rise. As core temperature rises, heat-dissipating reflexes are elicited; as the rate of heat dissipation increases, the rate of heat storage decreases and core temperature rises more slowly. Finally, as exercise continues, heat dissipation

[2]The condition of thermal steady state is included here to avoid artifacts due to differences among sites in the speed with which their temperatures respond to rapid changes in central blood temperature.

increases sufficiently to balance heat production, and essentially steady-state values are achieved (198).[3]

During exercise, the magnitude of core temperature elevation at steady state is largely independent of the environmental condition over a wide range, and is proportional to the metabolic rate (221), as first reported by Nielsen (141). Nielsen's subjects exercised at oxygen uptakes up to 3.0 $L \cdot min^{-1}$ in ambient temperatures between 5° and 36°C. **Figure 4** presents heat-exchange data for one subject during an hour of cycle exercise at a metabolic rate of approximately 650 watts in each ambient temperature. The difference between metabolic rate and total heat loss represents the energy used for mechanical work and the heat storage. Note that total heat loss, and therefore heat storage and elevation of core temperature, were essentially the same in all environments. The relative contributions of dry heat exchange (i.e., by convection and radiation) and evaporative heat exchange to the total heat loss, however, varied with the environmental conditions. At 10°C the large skin-to-ambient temperature gradient facilitated dry heat exchange, which accounted for ~70% of the total heat loss. As the ambient temperature increased, the gradient for dry heat exchange diminished and there was a greater reliance upon evaporative heat exchange.

[INSERT FIGURE 4 HERE]

Nielsen's finding that the steady-state level of core temperature is independent of the environmental conditions, conflicts with the personal experience of most athletes. For example, a runner will experience greater hyperthermia competing in a 35°C than in a 20°C environment. Lind (116) showed that core temperature elevation during exercise is independent of the environment only within a range of conditions or

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[3]Reaching such a steady state depends on two conditions, first, that it is possible to achieve sufficient heat dissipation to restore heat balance in the given environmental conditions; and second, that the control characteristics of the heat-dissipating responses to core and skin temperatures do not change with time. Under some circumstances, however, the second condition is not met, and core temperature continues to rise throughout exercise, though more slowly than during earlier stages. This may occur as a result of changes in fluid and electrolyte balance, hidromeiosis, or other factors.

a "prescriptive zone". **Figure 5** presents steady-state core temperature responses during exercise performed at three metabolic intensities in a broad range of environmental conditions. Environmental conditions are represented by the "old" effective temperature, an index that combines the effects of air temperature, humidity and air motion. Note that the greater the metabolic rate during exercise, the lower is the upper limit of the prescriptive zone.

[INSERT FIGURE 5 HERE]

Within the prescriptive zone, the proportional relationship between metabolic rate and steady-state core temperature elevation holds for a given individual, but does not, in general, hold well for comparisons between different individuals. Åstrand (5) first reported that use of relative exercise intensity, expressed as a percent of maximal oxygen uptake ($\dot{V}O_2max$), rather than actual metabolic rate (absolute intensity), removes most of the inter-subject variability in the relation of core temperature elevation to exercise intensity. Davies and his colleagues (43,45) explored this relationship further, in laboratory and field studies. Their subjects exercised at intensities between 20% and 90% of their $\dot{V}O_2max$, at dry-bulb temperatures ranging from 5° to 25°C, with relative humidity <50%. **Figure 6** is redrawn from their data. In **Figure 6a**, note the curvilinear relationship between steady-state core temperature and relative intensity (45). **Figure 6b** presents two subjects' steady-state core temperature values during exercise, at 65% and 85% $\dot{V}O_2max$, in relation to the ambient dry-bulb temperature (43). Core temperature was independent of dry-bulb temperature from 5 to 20°C at 65% $\dot{V}O_2max$; but at 85% $\dot{V}O_2max$, core temperature was influenced by dry-bulb temperature over the entire range of dry-bulb temperatures.

[INSERT FIGURE 6 HERE]

Since core temperature changes are related to the relative exercise intensity, it would seem logical to expect that any condition that lowers maximal oxygen uptake (and thus increases relative intensity) would also elicit a greater core temperature response at a given absolute (thus higher relative) intensity. Evidence from several studies, in which subjects' $\dot{V}O_2max$ was reduced with simulated high altitude in hypobaric chambers (81,110) or induced carboxyhemoglobinemia (138), suggests that

this is, in fact the case. However, contrary results have also been reported (172). Interpretation of such data is complicated by the fact that some experimental conditions decreased core temperature at rest, so that exercise would need to cause a greater net increase in core temperature to produce the same steady-state core-temperature level. Conversely, data from two studies in which autologous erythrocyte infusion was used to increase subjects' $\dot{V}O_2max$ suggest that conditions which increase $\dot{V}O_2max$ (thus lowering relative intensity) may lower the core temperature elicited by exercise at a given metabolic rate (187,192).

METABOLISM

Metabolic Rate

Most investigators find that $\dot{V}O_2max$ is lower in hot than in temperate environments (107,171,175,205). For example, $\dot{V}O_2max$ was 0.25 L·min^{-1} lower at 49°C than at 21°C in one study (199), and the state of heat acclimation in this study did not alter the size of the decrement in $\dot{V}O_2max$. However, some investigators report no effect of ambient temperature on $\dot{V}O_2max$ (169,245).

What physiological mechanisms might be responsible for such a reduction in $\dot{V}O_2max$? Thermal stress, by dilating the cutaneous vascular beds, might divert some of the cardiac output from skeletal muscle to skin, thus leaving less blood flow to support the metabolism of exercising skeletal muscle. In addition, dilation of the cutaneous vascular bed may increase cutaneous blood volume at the expense of central blood volume, thus reducing venous return and cardiac output. For example, Rowell et al. (174) reported that during intense (≈73% $\dot{V}O_2$) exercise in the heat, cardiac output can be reduced by 1.2 L·min^{-1} below control levels. Such a reduction in cardiac output during heat exposure could account for a 0.25 L·min^{-1} decrement in $\dot{V}O_2max$, assuming each liter of blood delivers ≈0.2 L of oxygen (1.34 ml O_2·gHb^{-1} x 15 gHb·100 ml^{-1} of blood).

Acute heat stress increases resting metabolic rate (38,39,46), but the effect of heat stress on an individual's metabolic rate for performing a given submaximal exercise task is not so clear. Many investigators report that to perform a given submaximal exercise task, the metabolic rate is greater in a hot than a temperate

environment (38,39,46,56). Some investigators, however, report lower metabolic rates in the heat (24,157,245,255). The state of heat acclimation does not account for whether individuals demonstrate an increased or decreased metabolic rate during submaximal exercise in the heat. However, other mechanisms may explain this discrepancy. Most investigators have only calculated the aerobic metabolic rate during submaximal exercise ignoring the contribution of anaerobic metabolism to total metabolic rate.

Dimri et al. (46) attempted to account for both aerobic and anaerobic metabolism in a study in which six subjects performed six-min exercise bouts at three intensities in each of three environments. **Figure 7** presents their subjects' total metabolic rate (bottom) and the percentage of this metabolic rate which was contributed by aerobic and anaerobic metabolic pathways. The anaerobic metabolism was calculated from the post-exercise oxygen uptake that was in excess of resting baseline levels. The aerobic metabolic rate at a given power output decreased with increasing ambient temperature. However, calculated anaerobic metabolic rate increased more than aerobic metabolic rate decreased, and they concluded that the total metabolic rate required to perform exercise at a given power output increases with higher ambient temperature. Their calculations of anaerobic metabolic rate may be challenged, and it is difficult to extrapolate these observations to predict what would occur during longer-term exercise, in which anaerobic metabolism would presumably be a smaller fraction of the total. Nevertheless, this study illustrates the pitfalls of ignoring anaerobic metabolism in approaching this question.

[INSERT FIGURE 7 HERE]

Investigations which report a lower metabolic rate during exercise in the heat also report increased plasma or muscle lactate levels (157,245,255) or an increased respiratory exchange ratio (24), also suggesting an increased anaerobic metabolism. However, any inference about metabolic events within the skeletal muscle based on changes in plasma lactate is open to debate (144). Plasma lactate concentration reflects the balance between efflux into the blood and removal from the blood. Rowell et al. (170) have shown that during exercise in the heat, the splanchnic vasoconstriction reduced hepatic removal of plasma lactate. Therefore, the greater

blood lactate accumulation during submaximal exercise in the heat can be attributed, at least in part, to a redistribution of blood flow away from the splanchnic tissues.

Skeletal Muscle Metabolism

Several investigations examined the effects of environmental heat stress on skeletal muscle metabolism during exercise. Fink et al. (58) had six subjects perform 45 minutes of cycle exercise (70 to 85% of $\dot{V}O_2max$) in a cold (9°C) and a hot (41°C) environment. They found greater plasma lactate concentrations and increased muscle glycogen utilization during exercise in the heat. Also, muscle triglyceride utilization was reduced during exercise in the heat as compared to the cold. In addition, serum glucose concentration increased, and serum triglyceride concentration decreased during exercise in the heat, compared to the opposite responses during exercise in the cold. During exercise in the heat, the increased muscle glycogen utilization was attributed to an increased anaerobic glycolysis resulting from local muscle hypoxia, caused by a reduced muscle blood flow. Since these investigators (58) did not perform control experiments in a temperate environment, the differences reported could be due partially to the effects of the cold exposure.

Young et al., (255) had 13 subjects perform 30 minutes of cycle exercise (70% of $\dot{V}O_2max$) in a temperate (20°C) and a hot (49°C) environment. They found skeletal muscle and plasma lactate concentrations were greater during exercise in the heat, but there was no difference in muscle glycogen utilization between the two experimental conditions. They speculated therefore that during exercise in the heat, an alternative substrate for glycolysis, such as blood glucose, might have been utilized. Rowell et al., (170) demonstrated a dramatic increase in hepatic glucose release into the blood during exercise in a hot compared to a temperate environment. Such an increased release of hepatic glucose could account for the elevated serum glucose concentration reported in the hot environment by Fink et al., (58).

Two studies (140,184) have measured arterial and venous lactate concentration across the active musculature during exercise in the heat. Savard and colleagues (184) had subjects wear a water-perfused suit under a water-impermeable coverall and perform cycle exercise (~25% $\dot{V}O_2max$) for a 25-min control period with no water

circulating, and then perfused the suit with ≈45°C water as exercise continued for another 25 min. They found heat stress did not alter oxygen uptake, lactate concentrations or muscle blood flow. Nielsen et al. (140) had subjects perform treadmill exercise (55-77% $\dot{V}O_2max$). During the initial 30 min they exercised in a temperate (≈20°C) environment, and then exercised in a hot (40°C) environment for an additional 60 min or until exhaustion. They found that heat stress increased oxygen uptake, but did not alter lactate concentrations, glycogen utilization rate, or muscle blood flow. Generalizations from these two studies are limited, since neither compared two identical bouts of exercise with and without heat strain.

Heat Acclimation

The oxygen uptake response to submaximal exercise appears to be affected by heat acclimation (196). Most reports indicate oxygen uptake and aerobic metabolic rate during submaximal exercise are reduced by heat acclimation (54,74,100,165,196,203,213,224), although a significant effect is not always observed (33,159,173,238,252). Large effects (14-17% reductions) have been reported for stair-stepping (203,213,224), but some of the reduction in oxygen uptake required for stair stepping can be attributed to increased skill and improved efficiency acquired during the acclimation program. By contrast, other studies (54,74,100,165,196) report acclimation-induced reductions in the oxygen uptake required for treadmill and cycle-ergometer exercise that, although statistically significant, were much smaller than the large reductions reported for stair stepping.

Lactate accumulation in blood and muscle during submaximal exercise is generally found to be reduced following heat acclimation. King et al., (104) and Kirwan et al., (106) both observed that heat acclimation reduced muscle glycogen utilization during exercise in the heat by 40-50% compared to before acclimation. Young et al., (255) also observed a statistically significant glycogen-sparing effect due to heat acclimation, but the reduction in glycogen utilization was small, and apparent only during exercise in cool conditions. Glycogen utilization during exercise in the heat was negligibly affected. The mechanism(s) for reduction in lactate accumulation during exercise after heat acclimation remains unidentified.

HEAT LOSS MECHANISMS

Evaporation

When ambient temperature increases, there is a greater dependence upon evaporative heat loss to defend core temperature during exercise (Figure 4). The sweat glands secrete sweat onto the skin surface which cools the body when it evaporates. When a gram of sweat is vaporized at 30°C, 2.43 kilojoules (kJ) of heat (the latent heat of evaporation) is absorbed in the process (239). The rate of evaporative heat loss is proportional to the product of the water vapor pressure gradient between the skin and environment, multiplied by the evaporative heat transfer coefficient and the wetted area of skin. The wider the gradient, the greater the rate of evaporation for a given evaporative heat transfer coefficient. The evaporative heat transfer coefficient incorporates the effects of such factors as air movement and the geometry of the surface. In addition, the water-vapor permeability of any clothing that is worn has a major effect on the rate of evaporation.

During cycle ergometer exercise a fit man may easily sustain a metabolic rate of 800 W ($\dot{V}O_2$ ≈2.3 L·min^{-1}). If the exercise performed is 20% efficient then the remaining 80% of the metabolic rate is converted to heat in the body, so that 640 W (i.e., 0.64 kJ·s^{-1} or 38.4 kJ·min^{-1}) needs to be dissipated to avoid body heat storage. The specific heat of body tissue is ≈3.5 kJ·kg^{-1}·°C^{-1} so that a 70-kg man has a heat capacity of 245 kJ·°C^{-1}. If this man performed exercise in an environment that enabled only dry-heat exchange, and he did not sweat, body temperature would increase by ≈1.0°C every 6.4 min (245 kJ·°C^{-1}÷38.4 kJ·min^{-1}). Since the latent heat of evaporation for sweat is 2.43 kJ·g^{-1}, this man would need to evaporate ≈16 g of sweat per min (38.4 kJ·min^{-1}÷2.43 kJ·g^{-1}) to achieve a steady-state body temperature.

Thermoregulatory sweating can begin within a few seconds to minutes after starting muscular exercise (190,233). The increase in thermoregulatory sweating closely parallels the increase in body temperature (79,133,176). As sweating rate increases, there is initially a recruitment of sweat glands and then an increased sweat secretion per gland (179). Therefore, the sweat secretion for a given region of skin is dependent upon both the density of sweat glands as well as the sweat secretion per gland. Different skin regions have different sweating responses for a given core

temperature (91,133). The back and chest have the greatest sweating rates, while the limbs have relatively low sweating rates.

The sudomotor signal descends through the brain stem and spinal tracts to exit into the paravertebral ganglionic chain. The post-ganglionic sympathetic fibers which innervate the sweat gland are nonmyelinated class C fibers that are primarily cholinergic. The sweat glands respond to thermal stress primarily through sympathetic cholinergic stimulation, but circulating catecholamines, in particular epinephrine, may facilitate thermoregulatory sweating as there are α and β adrenergic receptors associated with eccrine sweat glands (180,181,182). The relative effects on sweat secretion are 4:1:2 for cholinergic, α adrenergic and β adrenergic receptor stimulation, respectively (178). In addition, sweat gland metabolism is primarily oxidative phosphorylation with plasma glucose being the principal substrate (178).

For a given sudomotor signal to the sweat gland, local skin temperature and skin wettedness influence the amount of sweat secreted. Bullard et al., (26) were among the first to systematically evaluate the relationship between local skin temperature and thermoregulatory sweating. They found that in actively sweating skin, raising local skin temperature induced a greater sweating rate. These results have been verified and extended by many other investigators (56,130,143). Nadel et al., (130) measured local sweating rate from patches of skin whose temperature they varied, between 30 and 38°C, independently of core temperature and whole-body mean skin temperature. They concluded that local skin temperature affects the sensitivity of the sweating response to the reflex sudomotor signal, determined by core and whole-body mean skin temperatures.

The physiological mechanism by which elevated local skin temperatures enhances the sweating response is not entirely clear, but several factors may contribute. Local skin heating may increase the amount of neurotransmitter released by each sudomotor nerve impulse arriving at the sweat gland (26,56,130,143). The greater neurotransmitter release would, in turn, stimulate greater sweat production and release. In addition, Ogawa and Asayama, (143) provided evidence that local heating increases the sweat glands' responsiveness to a given amount of neurotransmitter

substance. It is unknown if this increased glandular responsiveness is receptor-mediated or reflects increased cellular metabolism within the secretory coil.

As mentioned previously, the evaporation rate is dependent upon the water vapor pressure gradient between skin and the environment, and on the evaporative heat transfer coefficient. The drier the air, or the warmer and wetter the skin, the wider the gradient and thus the greater the rate of evaporation for a given evaporative heat transfer coefficient. The evaporative heat transfer coefficient, in turn, depends on air movement, and thus sweat tends to collect on the skin in still or moist air. Wetting the skin gradually reduces sweat secretion (79,135), an effect called hidromeiosis. Several mechanisms are proposed to account for hidromeiosis (31,135,156). Peiss et al., (156) suggested that wetting the skin might cause the stratum corneum to swell and mechanically obstruct the sweat duct. A systematic test of this idea by Brown and Sargent (25) supported the concept that hydration of the stratum corneum is responsible for hidromeiosis.

Heat Loss and Skin Blood Flow

Blood flow transfers heat by convection from the deep body tissues to the skin. When core and skin temperatures are low enough that sweating does not occur, raising skin blood flow brings skin temperature nearer to blood temperature, and lowering skin blood flow brings skin temperature nearer to ambient temperature. Thus, the body can control dry heat loss by varying skin blood flow, and thereby skin temperature. In conditions in which sweating occurs, the tendency of skin blood flow to warm the skin is approximately balanced by the tendency of sweating to cool the skin. Therefore after sweating has begun, skin blood flow serves primarily to deliver to the skin the heat that is being removed by sweat evaporation. Skin blood flow and sweating thus work in tandem to dissipate heat under such conditions.

Skin circulation is affected by temperature in two ways: local skin temperature affects the vascular smooth muscle directly, and temperatures of the core and of the skin elsewhere on the body affect skin blood flow by reflexes operating through the sympathetic nervous system. Blood flow in much of the human skin is under a dual vasomotor control (102,168,198). In the hands, feet, lips, ears, and nose, adrenergic

vasoconstrictor fibers are probably the predominant vasomotor innervation, and the vasodilation that occurs in these regions during heat exposure is largely the result of withdrawing vasoconstrictor activity. Over most of the skin area, however, there is relatively little vasoconstrictor activity under conditions of thermal comfort, and vasodilation during heat exposure depends on intact sympathetic innervation. Since it depends on the action of neural signals, such vasodilation is sometimes referred to as active vasodilation. Both active vasoconstriction and active vasodilation play a major part in controlling skin blood flow of the upper arm, forearm, thigh, and calf (18). However, active vasodilation is believed to be primarily responsible in controlling skin blood flow on the trunk and on most of the head (19,66).

The vasoactive agonist responsible for active cutaneous vasodilation in man has not been identified. Roddie et al. (166) proposed that acetylcholine has a major role, although they allowed that it may not be the only vasodilator responsible for active cutaneous vasodilation. Such a role for acetylcholine has not been definitively tested, but has received relatively little attention in recent years. Other investigators, because of the long-known association between the occurrences of sweating and active cutaneous vasodilation, have proposed that the vasodilation may depend in some way on activation of the sweat glands (69,120). Fox and Hilton (69) proposed that when sweat glands are stimulated to secrete sweat, they also release bradykinin, a powerful vasodilator, into the skin, thus causing the cutaneous arterioles to dilate. Their hypothesis now seems unlikely, and Rowell (167) has summarized the arguments against it. However, Brengelmann and colleagues (23) have provided further evidence for some such link between sweating and cutaneous vasodilation by demonstrating that patients with congenital absence of active sweat glands do not undergo active vasodilation during heat exposure.

Recently, vasoactive intestinal polypeptide (VIP) has been proposed as a candidate for the link between sweating and vasodilation. Lundberg and associates (121) have shown that VIP and acetylcholine are concomitantly released from the same post-ganglionic fibers innervating exocrine glands of the cat. Although both transmitters caused exocrine secretions, the VIP primarily acts by dilation of local vasculature to facilitate sweat gland function. Therefore, it has been proposed that co-release of acetylcholine and VIP at the human sweat gland might help explain the

relationship between sweating and active vasodilation (108,121,232). Recently, Savage and colleagues (183) reported that men with cystic fibrosis show essentially normal active vasodilation in response to increases in core temperature. They also reported that immunohistochemical analyses of skin biopsies from these subjects showed that VIP innervation was sparse, while calcitonin gene-related peptide (CGRP) and substance P were present in normal amounts. They therefore expressed caution about a dominant role for VIP in active cutaneous vasodilation, and proposed that release of one or both of the other peptides, CGRP and substance P, may be the mechanism responsible for active cutaneous vasodilation.

In skin regions where skin blood flow is controlled primarily through vasoconstrictor fibers, the effect of local temperature on skin blood flow depends largely on local temperature's modifying the vasoconstrictor response to nerve signals or directly applied constrictor agonist (for discussion see Reference # 244). This effect appears to be mediated through changes in α-adrenergic receptor affinity (97). However, in human skin regions where active vasodilation has a major role in controlling blood flow, local temperature has a large effect on skin blood flow that seems to be largely independent of nervous signals, since it is unaffected by complete nerve block (244).

In the limbs especially, the relation between core-to-skin heat transfer and skin blood flow can be modified by altering the caliber of the superficial veins. This is possible because of the dual venous drainage of the limbs, which consists of deep veins, which ordinarily drain blood mainly from the muscles; and superficial veins, which lie in the skin and subcutaneous tissue and ordinarily drain blood mainly from the skin. However, since many penetrating veins connect the deep veins with the superficial veins, blood from anywhere in the limbs can potentially return to the heart through either deep or superficial veins. The sympathetic innervation of the deep veins is rather sparse, but the superficial veins have a rich sympathetic innervation (237), and dilate when the body core or skin is warmed, and constrict when the core or skin is cooled (236,241). Thus in a cool subject, most blood from the limbs returns to the core via the deep veins, and does not readily exchange heat with the environment, so that heat tends to be conserved. In a warm subject, by contrast, the superficial veins dilate, allowing large amounts of venous blood to return to the core

along paths close to the skin surface, so that heat exchange with the skin and the environment is facilitated. In addition, dilation of the smaller superficial veins may further improve heat transfer by increasing the time that the blood remains in the skin, as suggested by evidence that the degree of venous filling affects heat transfer to the skin (78). Though advantageous for thermoregulation, peripheral venodilation in the heat adds to the burden on cardiovascular homeostasis, as discussed in the next section.

Cardiovascular Support for Thermoregulation and Exercise

As noted earlier, skin temperature is higher in warmer environments, while core temperature is relatively unaffected by environmental temperature over a wide range. Thus as ambient temperature increases, the core-to-skin thermal gradient becomes narrower, and skin blood flow increases in response to the high skin temperature, so as to achieve core-to-skin heat transfer sufficient for thermal balance. During exercise, metabolic rate and heat production may be ten times their levels at rest, and delivery of heat to the skin must increase proportionately, in order to re-establish thermal balance. Core temperature does rise during exercise, thus widening the core-to-skin temperature gradient. However, the increase in the core-to-skin gradient is not proportionate to the increase in heat production, so that only by an accompanying increase in skin blood flow does core-to-skin heat transfer increase enough to eventually match the increase in metabolic heat production, and allow heat balance to be reestablished. Since the core-to-skin temperature gradient is relatively narrow in hot environments, skin blood flow must be rather high to achieve sufficient heat transfer to maintain thermal balance during exercise (168,198). Actual levels of skin blood flow attained during exercise-heat stress, though not precisely known, are probably as high as several liters per minute (168).

During exercise in the heat, the primary cardiovascular challenge is to provide simultaneously enough blood flow to exercising skeletal muscle to support its metabolism, and enough blood flow to the skin to dissipate heat. High skin blood flow often is associated with reduced cardiac filling and stroke volume, which require a higher heart rate to maintain cardiac output (129,168,198). This reduction in cardiac filling occurs because the venous bed of the skin is large and compliant, and dilates

reflexively during heat stress. For these reasons, the venous bed of the skin—especially below heart level—tends to become engorged with blood, at the expense of central blood volume, as skin blood flow increases. In addition, sweat secretion can result in a net loss of body water, and thereby reduce blood volume (188,200). Therefore, heat stress can reduce cardiac filling both through pooling of blood in the skin and through reduced blood volume.

Several reflex adjustments compensate for peripheral pooling of blood and for decreases in blood volume due to changes in fluid balance, and help to maintain cardiac output and arterial pressure during exercise-heat stress. Splanchnic and renal blood flows are reduced during exercise in proportion to relative exercise intensity (168). These blood flows also undergo a graded and progressive reduction in subjects heated while resting; and in the splanchnic bed, at least, the vasoconstrictor effects of temperature and of exercise appear to be additive, so that at any exercise intensity the reduction in splanchnic blood flow is greater at a higher skin temperature (168). Reduction of renal and splanchnic blood flow allows a corresponding diversion of cardiac output to skin and exercising muscle. Moreover, secondarily to the reduction in splanchnic blood flow, a substantial volume of blood can be mobilized from the compliant splanchnic beds to help maintain cardiac filling during exercise and heat stress. In addition, there may be an increased cardiac contractility, to help defend stroke volume in the face of impaired cardiac filling. If these compensatory responses are insufficient, skin and muscle blood flow will be compromised, leading to reduced exercise performance and possibly dangerous hyperthermia.

HEAT ACCLIMATION

Induction and Decay

Repeated heat exposure will reduce physiological strain and improve exercise performance during subsequent heat exposures[4]. Exercise in the heat is the most

[4]According to a frequently-observed convention, such changes are called acclimatization if they are observed following a change in a naturally-occurring environment, and acclimation if they are produced in a controlled experimental setting.

effective method for developing heat acclimation; however, even resting in the heat results in some acclimation, though to a lesser degree (15,53,240). The full development of exercise-heat acclimation need not involve daily 24-h exposure. A continuous, daily 100-min period of exposure appears to produce an optimal heat acclimation response in dry heat (118)[5].

The effect of heat acclimation on performance can be quite dramatic, so that acclimatized subjects can easily complete exercise in the heat which earlier was difficult or impossible. Figure 8 depicts the improvement in exercise-heat tolerance for 24 men who attempted 100 min of treadmill exercise at 49°C, 20% relative humidity, for seven days. This figure shows that no subjects completed the 100-min walk on day 1; however, 40% were successful by day 3, 80% by day 5, and all but one of these men were successful by the seventh acclimation day (155).

[INSERT FIGURE 8 HERE]

During acclimation through daily exercise in a hot environment, most of the improvement in heart rate, skin and core temperatures, and sweat rate is achieved during the first week of exposure although there is no sharp end to the improvement (1). Heart rate shows the most rapid reduction (148,165,250), most of which occurs in four to five days (148). After seven days, the reduction in heart rate is virtually complete while most of the improvement in skin and core temperatures has also occurred (95,148,165). The thermoregulatory acclimation response is about three quarters developed by the end of the first week of exposure, and is generally thought to be complete after 10-14 days of exposure (155). The improved sweating response and ease of walking reported during heat acclimation may take longer to achieve (95,240).

Heat acclimation is transient and gradually disappears if not maintained by repeated heat exposure. It is believed that the heart rate improvement, which develops more rapidly during acclimation, is also lost more rapidly than are the other

[5]Although this seems to be true for dry heat, longer daily heat exposures may be needed to produce optimal acclimation in humid heat, as discussed later.

improvements in thermoregulatory responses (118,148,246). However, there is considerable variability in the literature concerning the rate of decay for heat acclimation (240). Lind (117) believed that heat acclimation might be retained for two weeks after the last heat exposure, but then be rapidly lost during the next two weeks. However, Williams and colleagues (246) report some loss of acclimation in sedentary individuals after one week, with the percentage loss being greater with increasing time; and, by three weeks losses of nearly 100% for heart rate and 50% for core temperature. Other authors (178), however, report greater retention of the acclimation benefits in physically trained and aerobically fit persons. Warm weather may also favor retention of acclimation (53), but there is some conflicting evidence on this issue (246).

Underlying Mechanisms

The three classical signs of heat acclimation are lower heart rate and core temperature, and higher sweat rate during exercise-heat stress. Skin temperature is lower after heat acclimation than before, and thus dry heat loss is less (or, if the environment is warmer than the skin, dry heat gain is greater). **Figure 9** illustrates the effects on three healthy young men, acclimated by daily treadmill walks in the heat for 10 days (54). To compensate for the changes in dry heat exchange, there must be an increase in evaporative heat loss, in order to achieve heat balance. As noted earlier, changes in sweating develop more slowly than those in heart rate and rectal temperature, so that changes in sweating probably cannot account completely for the other changes.

(INSERT FIGURE 9 HERE)

After acclimation, sweating starts earlier and at a lower core temperature, i.e., the core temperature threshold for sweating is decreased. The sweating response to local application of a standard dose of methacholine, a synthetic cholinergic agonist, increases (37,111) and a given elevation in core temperature elicits a higher sweat rate. The sweat glands also become resistant to hidromeiosis and "fatigue" (36,67) so that higher sweat rates can be sustained. These changes reduce the levels of core and skin temperatures reached during a given exercise-heat stress, increase the

sweat rate, and prolong tolerance time. In addition, the threshold for cutaneous vasodilation is reduced so that heat transfer from core to skin is maintained. The concomitant reduction in thresholds for both sweating and cutaneous vasodilation is evidence that heat acclimation is an example of a set-point change.

On the first day of exercise in the heat, heart rate reaches much higher levels than in temperate conditions, and stroke volume is lower. Thereafter, heart rate begins to decrease and, usually, stroke volume starts to increase as early as the second day of heat acclimation (e.g., Reference # 250). These changes are rapid at first, but continue more slowly for about a week. The time course of the decrease in heart rate is roughly similar to that of the increase in plasma volume, and the two changes are significantly correlated (204), though the maximum decrease in heart rate appears to precede the maximum increase in plasma volume (**Figure 10**).

(INSERT FIGURE 10 HERE)

Perhaps it is an oversimplification to expect a single explanation to account for the heart rate reduction during exercise in the heat after heat acclimation. There may be several mechanisms that participate, and their relative contributions may vary, both over the course of the heat acclimation program and also among subjects. Three explanations have been proposed for the decrease in heart rate during heat acclimation.

One explanation is the decrease in heart rate results from a plasma volume expansion. The plasma volume expansion at rest during the first week of acclimation probably contributes somewhat in reducing heart rate and circulatory strain; however, plasma volume at rest returns toward control levels after one to two weeks of acclimation (14,206). A second explanation is the decrease in heart rate results from an increased venous tone, since venoconstriction can mobilize up to 25% of the blood volume (14). Wood and Bass (247), however, found a significant reduction in forearm venous volume only on the third and fourth days of heat acclimation, well after much of the reduction in heart rate had already occurred. Thus, although peripheral venomotor adjustments may play a transitional role in heat acclimation, there is no evidence of their making a persistent contribution early in the acclimation program. It

is possible, however, that other (non-cutaneous) venous beds participate in the circulatory changes that occur with acclimation, but this matter has not been investigated. A third explanation is the decrease in heart rate is secondary to the fall in core and skin temperatures with heat acclimation (173). Since heart rate at a given core temperature is the same or higher after acclimation than before (67,68), lower body temperatures are a necessary condition for lower heart rate with a given exercise-heat stress after acclimation. However, besides lowering core and skin temperatures during exercise-heat stress, acclimation lowers the core temperature thresholds for vasodilation and venodilation, so that lower core and skin temperatures by themselves are not a sufficient explanation for lower heart rate after acclimation. A final reservation with respect to this explanation is that exercise heart rate usually decreases more rapidly than exercise core temperature over the course of heat acclimation (249).

The effects of heat acclimation on stroke volume responses to exercise-heat stress are not clear-cut. For example, two studies (173,250) report increased stroke volume with little change in cardiac output as heart rate fell; another study (248) reports a decrease in cardiac output, associated with a decrease in "surface blood flow" (estimated calorimetrically) as heart rate fell, and little change in stroke volume; still another study (252) reports a mixed pattern, with two subjects showing a steady increase in stroke volume, one a transient increase, reversing after the sixth day, and one showing no increase. The reason for these differences is not clear. Rowell et al. (173) describe dry heat acclimation, and Wyndham (248) and Wyndham and colleagues (250,252) all describe humid heat acclimation.

Heat acclimatization increases total body water, but the division of the total increase between intracellular fluid and extracellular fluid is quite variable (see Reference # 240, for a discussion). Much of the increase is accounted for by an expansion of resting plasma volume (14,204,206); however, Young and colleagues (256), recently have demonstrated that an expanded plasma volume is not always present after repeated exercise-heat exposure. The mechanism(s) responsible for this hypervolemia are unclear, but may include an increase in extracellular fluid mediated by retention of crystalloids (primarily sodium chloride) and perhaps an increase in plasma volume selectively mediated by the oncotic effect of intravascular protein (84).

Heat acclimated persons also exhibit a more stable plasma volume and more consistent intravascular fluid response to exercise-heat stress than do persons who are not heat acclimated (185). The increase in total body water can be explained in part by increased aldosterone secretion and/or renal sensitivity to a given plasma concentration. Francesconi and colleagues (70) have shown that exercise-heat exposure markedly increased plasma aldosterone concentration which was subsequently abated by heat acclimation. Heat-acclimation effects on other hormones that affect fluid conservation, such as vasopressin and atrial natriuretic factor, have not been reported.

An unacclimatized person may secrete sweat with a sodium concentration of 60 meq·L^{-1} or higher[6] and therefore, if sweating profusely, can lose large amounts of sodium. With acclimatization, the sweat glands become able to conserve sodium by secreting sweat with a sodium concentration as low as 5 meq·L^{-1} (164). **Figure 11** shows the effects of heat acclimation on sweat sodium concentration over a range of sweating rates (1). This salt-conserving effect of acclimation depends on the adrenal cortex, and aldosterone, which is secreted in response to exercise and heat exposure as well as to sodium depletion, appears to be necessary for its occurrence (see Reference # 240 for a discussion). However, it is not clear that aldosterone (together with other endogenous steroids with a mineralocorticoid action) is solely responsible for causing the sweat glands to conserve salt; an alternative explanation is that other factors associated with acclimation have a contributory role, but are effective only in the presence of aldosterone. Based on comparisons of sweat sodium content and plasma aldosterone concentration over the course of acclimation, Kirby and Convertino (105) proposed that acclimation increases sweat gland responsiveness to aldosterone. The interpretation of such changes is complicated, however, by the rather slow response of the sweat glands to aldosterone (34,35) with the result that the effect of aldosterone on the sweat glands is cumulative, and not due solely to its present concentration. The conservation of salt also helps to maintain the number of

[6]This is near the upper limit of sodium concentrations usually observed in sweat from normally aerated skin, although substantially higher concentrations have been reported in sweat collected from skin areas enclosed in impermeable barriers (164)

osmoles in the extracellular fluid, and thus to maintain extracellular fluid volume at the expense of intracellular fluid, as a subject becomes dehydrated (185).

(INSERT FIGURE 11 HERE)

Dry vs. Humid Heat

Although heat acclimation in a dry environment confers a substantial advantage in humid heat (15,53), the physiological and biophysical differences between dry and humid heat lead one to expect that humid heat acclimation would produce somewhat different physiological adaptations from dry heat acclimation. Though the pertinent literature is rather meager, there is evidence to support this expectation.

Fox et al., (64) compared the effects of acclimation to dry and to humid heat on the inhibition of sweating. They acclimated resting subjects with controlled hyperthermia, maintaining core temperature near 38.2°C for 2 hours a day for 12 days, using dry heat for one group and moist heat for the other group. To collect sweat, both groups had their left arms in plastic bags, which created a warm, humid microclimate. After acclimation, both groups showed similar decreases in heart rate and core and skin temperatures, with similar increases in sweating during an exercise-heat test. In a two-hour controlled hyperthermia test while they rested in very humid heat, both groups had about the same whole-body sweat rates. The arms that were exposed to humid heat during acclimation had—compared to pre-acclimation responses—similar and large increases in their sweat production during this test, and sweat rates of these arms declined more slowly during the test. During the same test the right arms of the "dry" group, which had not experienced humid heat during acclimation, also had a higher initial sweat rate than before acclimation, but thereafter their sweat rate declined as fast as before acclimation, so that their total sweat secretion during the test was substantially less than that either of the contralateral arms or of the arms of the "humid" group. Thus most of the improvement in the ability to maintain high sweat rates in high humidity after acclimation was apparently owed to a diminution of hidromeiosis.

Strydom and Williams (223) tested subjects' responses to 4 h of exercise in a humid environment both before and after a program of physical training, and compared their responses to those of another group of subjects who were well acclimated to humid heat. During the first hour of exercise, subjects in the training group showed better heat tolerance after training than before, and their responses after training approached those of the well-acclimated group. During the second hour of exercise, however, their heart rates and rectal temperatures increased more than those responses for the well-acclimated subjects, and by the end of the second hour their responses after training had come to appear more like their responses before training, and less like the responses of the well acclimated subjects. Except during the first hour of the exercise-heat exposure, the physically trained subjects sweated considerably less than the well acclimated subjects (223). Therefore, the probable reason for the greater physiological strain that the physically trained subjects experienced in the second hour and beyond was their inability to secrete and evaporate sweat at a rate sufficient to achieve thermal balance.

To achieve a high evaporative cooling rate in a humid environment, it is necessary to overcome the high ambient water vapor pressure by maintaining either a higher vapor pressure at the skin (which requires a higher skin temperature) or a larger wetted skin area, as compared to what would be necessary in a dry environment. Unless core temperature is allowed to rise along with skin temperatures, the higher skin temperature must be achieved by increasing core-to-skin thermal conductance, which requires a higher skin blood flow. Therefore, one expected difference between acclimation to humid heat and acclimation to dry heat is for the former to involve greater circulatory adaptations, to support higher skin blood flow with minimal circulatory strain. Unfortunately, the differences in circulatory adaptations to humid versus dry heat acclimation have not been investigated.

Another difference that might be expected between acclimation to humid heat and dry heat is for the former to enable more efficient use of the skin as an evaporating surface. In humid heat, a greater portion of the sweat production is on the limbs after acclimation than before (65,94,112,211). We are not aware of any reports of changes in the regional distribution of sweating after acclimation to dry heat. Before acclimation, mean sweating intensity (i.e., sweat rate per unit area) is much

lower on limbs than on the trunk, so acclimation tends to make the sweating intensity more uniform over the skin surface. This is an advantage in humid heat, because it increases the wet body surface area, and therefore sweat evaporation rate, and probably reduces the extent to which sweating in some regions is in excess of the rate it can be evaporated.

BIOMEDICAL FACTORS MODIFYING EXERCISE-HEAT PERFORMANCE

Aerobic Fitness

An improvement in aerobic fitness achieved through endurance training in a temperate environment reduces the physiological strain and increases tolerance to exercise in the heat (74,75,76,134,159,160), and endurance-trained individuals exercising in the heat exhibit many of the characteristics of heat-acclimated individuals (see Figure 9) (76,148,159,160,213). In addition, high aerobic fitness facilitates acquisition of heat acclimation (148).

Several authors suggest that high maximal aerobic power is related to improved exercise-heat tolerance (213) or a rapid rate of heat acclimation (148). These two groups of investigators utilized different hot climates (wet or dry) and independently reported that an individual's $\dot{V}O_2max$ accounts for approximately 44% of the variability in core temperature after 3 h of exercise in the heat, or the number of days of heat acclimation required to achieve a steady state in final core temperature as illustrated in Figure 12. However, endurance training alone does not totally replace the benefits of heat acclimation produced by a program of exercise in the heat (4,145,224). Some investigators (9,89) have proposed that in order for endurance training to improve thermoregulatory responses during exercise in the heat, the training sessions must be associated with substantial elevations of core temperature and sweating rate. Henane and colleagues (89) compared thermoregulatory responses of six skiers ($\dot{V}O_2max$=66.5 $ml \cdot kg^{-1} \cdot min^{-1}$) with those of four swimmers ($\dot{V}O_2max$ = 65.8 $ml \cdot kg^{-1} \cdot min^{-1}$), found that skiers were more heat tolerant and better acclimatized than swimmers, and attributed the difference to a smaller increase in the swimmers' core temperature produced by training in cold water. In support of this interpretation, Avellini et al. (9) found that four weeks of training by cycle exercise in 20°C water increased $\dot{V}O_2max$ by about 15%,

but did not improve thermoregulation during exercise-heat stress. Thus high aerobic fitness is not always associated with improved heat tolerance.

(INSERT FIGURE 12 HERE)

To achieve optimal thermoregulatory results from endurance training in temperate environments, either strenuous interval training or continuous training at an intensity greater than 50% $\dot{V}O_2max$ should be employed (74,89,134,145,161). Lesser training intensities produce questionable effects on performance during exercise-heat stress (212). The endurance training must last at least one week (134,161,214), and some authors (74,89) show that the best improvements require 8-12 weeks of training.

Dehydration

Generally, loss of body water impairs aerobic exercise performance in the heat (195); the warmer the environment, the greater the adverse effect of dehydration. Furthermore, the thermoregulatory advantages conferred by high aerobic fitness and heat acclimation are negated by dehydration (27,30,197). In comparison to euhydration, a water deficit of as little as 1% of body weight increases core temperature during exercise in both comfortable and hot environments (55); the greater the water deficit, the greater is the elevation of core temperature during exercise (128,200). Dehydration impairs both dry and evaporative heat loss (or, if the air is warmer than the skin, dehydration aggravates dry heat gain) (186,195). In addition, dehydration causes exhaustion from heat strain to occur at lower core temperatures during exercise-heat stress (201).

Dehydration may be associated with either reduced or unchanged sweating rates at a given metabolic rate in the heat. However, even when dehydration is associated with no change in sweating rate, core temperature is usually elevated, so that sweating rate for a given core temperature during dehydration still is lower (195). The physiological mechanisms mediating the reduced sweating rate during hypohydration are not clearly defined, but both the separate and combined effects of plasma hyperosmolality (63,202) and hypovolemia (62,191) have been suggested to play a part (see Reference # 186 for discussion).

Dehydration also affects cardiovascular responses to exercise-heat stress. During submaximal exercise with moderate to severe thermal strain (128.131,193), dehydration by 3%-4% of body weight increases heart rate and decreases stroke volume and cardiac output relative to euhydration. Dehydration also reduces cutaneous blood flow for a given core temperature (61,63,131,230), and therefore the potential for dry heat exchange. Likewise, hyperosmolality, in the absence of hypovolemia, can also reduce the cutaneous blood flow response during exercise-heat stress (61).

Circadian Patterns and Sleep Loss

Time of day affects thermoregulatory set point. Core temperature at rest varies with time of day in a sinusoidal fashion, with the minimum at night, and the maximum, which is 0.5 to 1°C higher, occurring in the late afternoon or evening. This pattern coincides with patterns of activity and eating, but does not depend on them. This pattern is an example of a circadian rhythm, i.e., a rhythmic pattern in a physiological function with a period of about one day. Since the circadian rhythm in core temperature results from a similar circadian rhythm in the thermoregulatory set point, it is accompanied by corresponding changes in the thresholds for all the thermoregulatory responses that have been studied (220) as **Figure 13** shows for sweating and neurogenic vasodilation. At the time when core temperature at rest is at its minimum, the sensitivity of the forearm vasodilator response to changes in core temperature (i.e., the slope of the forearm blood flow/core temperature relationship during leg exercise) is also at its minimum (216,218). These changes are consistent with the observation that exercise at a given intensity produces a greater core-temperature change when core temperature at rest is at its minimum (218).

(INSERT FIGURE 13 HERE)

Sleep deprivation disrupts the circadian rhythm, and delays the time of minimum core temperature at rest. The slope of the forearm blood flow/core temperature relation is reduced, and this reduction in slope seems not to be affected by phase of the circadian cycle. Recently, two studies demonstrated that sleep loss decreases the thermosensitivity of both sweating and peripheral blood flow, with no

evidence for a change in core temperature threshold during exercise (109,190). In addition, sleep deprivation is reported to negate the effects of heat acclimation (13,122).

Skin Disorders

Certain skin disorders will impair the ability to dissipate body heat and cause increased thermal strain (146). Both heat rash and sunburn may have profound thermoregulatory effects, and their adverse effects on exercise-heat tolerance deserve the same attention as lack of heat acclimation, low aerobic fitness, dehydration, and heavy clothing.

Artificially induced miliaria rubra (heat rash) over as little as 20% of the body surface causes an observable reduction in exercise-heat tolerance (153); involvement of 40% or more of the body surface markedly reduces tolerance time during exercise in the heat (49°C, 20% rh), and causes greater heat storage compared to responses in a non-rash state (152). These effects were observed both 7 and 14 days after a 72-h miliaria-induction period (152), and may persist for up to three weeks after the heat rash has resolved clinically (153). The relation of the degree of heat intolerance to the total area of skin affected by heat rash appears to depend on the specific region of the body and that region's normal sweating responses; smaller rashed areas of the trunk, because of the greater sweating capacity of normal trunk skin, may affect responses to dry-heat stress as much as larger rashed areas of the limbs. These investigators suggest that the absence of observable sweating in the rashed areas results from a physical occlusion of the affected sweat glands by keratotic plugs. Had the humidity of the test environment been higher, it is possible that the resultant impairment in thermoregulation and heat tolerance after heat rash would have been even more dramatic (152).

Mild artificial sunburn, induced with twice the minimal erythemal dose (MED) of ultraviolet light, impairs sweat gland activity during exercise in the heat (49°C, 20% rh) (150). Both the local sweating sensitivity and steady-state sweating rate from the burned areas are reduced 24 h post-sunburn compared to pre-sunburn values, but return to normal within one week. Mild sunburn thus appears to have a locally-

mediated effect on both the responsiveness of the sweat gland and its capacity to deliver sweat to the skin surface. These investigators suggest that this effect results from damage to the cuboidal epithelial cells composing the sweat duct, which blocks the egress of sweat from the affected duct. More severe levels of artificially-induced sunburn such as three or four MEDs may have more profound thermoregulatory consequences during exercise-heat exposure. In addition, artificially-induced sunburn also results in impaired ability to vasoconstrict during cold-water immersion (T_w=22°C), resulting in greater heat loss (151). These adverse thermoregulatory effects in the cold were still present one week after sunburn when the associated erythema had disappeared.

Medications

Many drugs inhibit sweating (most obviously those used for their anticholinergic effects), such as atropine and scopolamine. Since the military had fielded atropine citrate to protect personnel from nerve gas poisoning, considerable research has been performed on its thermoregulatory effects during exercise-heat stress (108). In addition, several drugs used for other purposes, such as glutethimide (a sleep medicine), tricyclic antidepressants, phenothiazines (tranquilizers and antipsychotic drugs), and antihistamines, also have some anticholinergic action, and all of these, plus a number of others (32), have been associated with heat stroke. It is likely that many such drugs, whether or not they possess an anticholinergic action, also alter thermoregulation through their effects on neurotransmission. A specific example that has particular importance to the defense community is the anticholinesterase drug pyridostigmine, used as a pre-treatment against nerve gas poisoning, which, in addition to increasing sweating, inhibits cutaneous vasodilation during exercise-heat stress (217). The mechanism by which pyridostigmine inhibits cutaneous vasodilation is not known, although a direct action on the central nervous system is unlikely, since pyridostigmine contains a quaternary ammonium group and thus does not readily cross the "blood-brain barrier". However, it is hard to reconcile this effect on skin blood flow with the notion that active cutaneous vasodilation is mediated by cholinergic sympathetic fibers (166).

SPECIFIC POPULATIONS

Women

Core temperature changes in a cyclical fashion according to the phase of the menstrual cycle (216,219). Core temperature is at its lowest point just before ovulation, and over the next few days rises 0.4 to 0.6°C to a plateau which persists throughout the luteal phase. The variation in core temperature during the course of the menstrual cycle results from a similar variation in the thermoregulatory set point, and thus is accompanied by corresponding changes in the thresholds for all the thermoregulatory responses (92). During the follicular phase, thresholds for women's thermoregulatory responses are similar to those of men in the same condition of physical training and heat acclimation, and, if suitable adjustments are made for these factors and anthropometric factors, so are the sensitivities of these responses to changes in core temperature (216,219). There is some evidence that sensitivities of the thermoregulatory responses to changes in core temperature differ between the follicular and luteal phases, but other studies show no difference between phases (216,219). In addition, estrogen replacement therapy can reduce thermoregulatory strain in middle-aged women during exercise in the heat (228).

It was once believed that women were less tolerant to heat strain than men; however, it now seems that studies on which that belief was based compared relatively unfit women to more fit men (216,219). Thermoregulatory responses to heat exposure at rest appear to be similar for both genders (42). Recent research demonstrated that if male and female populations are matched for aerobic fitness (or relative exercise intensity), they have similar tolerance and body-temperature responses to exercise in dry and humid heat (8,10,72,73,207), heat acclimation (8,9) and dehydration (197).

Blacks

In a study of American soldiers, black soldiers had slightly lower heart rates, rectal temperatures, and sweating rates than white soldiers during marches in hot humid weather (11). These differences between black and white soldiers seemed unrelated to where the soldiers had lived before, since southern white soldiers were no more heat tolerant than northerners. The differences disappeared in hot dry weather,

if the soldiers marched in the shade or were fully clothed, so as to eliminate differences in absorption of solar radiation by the skin.

In a study of Mississippi sharecroppers in summer, white sharecroppers had slightly higher final rectal and skin temperatures, and heart rates than black sharecroppers after a two-hour treadmill walk in humid heat (163). The differences were partly due to a higher metabolic rate for the whites, both absolutely and in relation to body weight. The whites also sweated more, both absolutely (by 14%) and in relation to their heat dissipation. Since the blacks had mean skin temperatures 0.54°C lower than the whites, differences in dry heat exchange are in the wrong direction to explain differences in sweating. Thus the blacks apparently used their sweat more efficiently, evaporating more of their sweat to obtain cooling and wasting less by dripping.

Some of the above differences between black and white Americans find parallels in data reported from Africa. At high wet bulb temperatures, acclimated African laborers sweated much less, and had slightly lower heart rates than reported for acclimated Europeans at similar heat stress levels (251). In another study, unacclimated whites were less heat-tolerant (as indicated by symptoms of heat strain) than unacclimated Bantu during four-hour exercise in humid heat, and had higher rectal temperatures even though they sweated significantly more (253). The whites also had a greater metabolic rate during a given exercise task. After acclimation, differences in heat tolerance and rectal temperature disappeared, and differences in metabolic rate were less, but persisted. However, after the first hour the whites still sweated more, and the difference in sweat rate increased with time.

Fully acclimated Europeans and Nigerians have similar rectal temperatures and sweat rates early during exercise under severe conditions, but the Nigerians can continue longer and are less likely to collapse (113). The sweat of Nigerians fully acclimated to humid heat is more dilute than that of similarly acclimated Europeans. During exercise in humid heat, sweat rate of acclimated Nigerians (114) or Bantu (253) decreases more quickly than that of Europeans (114). Furthermore, Europeans living in Nigeria but not fully acclimated, thermoregulate as well as similarly acclimated Nigerians during moderate heat stress. However, in more severe heat stress, they do

not thermoregulate as well, even though they sweat more than their Nigerian counterparts.

In summary, studies conducted both in Africa and in the United States indicate that in hot humid conditions blacks use their sweat more efficiently for evaporative cooling, and thus waste less sweat, than whites; experience less circulatory strain, and have slightly lower metabolic rates than whites during similar exercise tasks. In interpreting these studies, however, it should be remembered that the subject populations were not matched for factors such as aerobic fitness.

Children and Older Adults

Children and Adolescents. Children and adolescents generally have a greater surface area-to-mass ratio, which should result in faster absorption of heat in hot environments where the ambient temperature exceeds skin temperature or there is a high radiant heat load (12). Children also display a higher metabolic rate for a given work rate, and a slightly lower cardiac output for a given metabolic rate when compared to adolescents or adults (12). In addition, prepubertal children have a lower sweating rate during exercise-heat exposure than post-pubertal children and young adults. These physical and physiological characteristics of children and adolescents could have implications for their ability to thermoregulate.

The anthropometric and physiological differences between children and young adults suggest that the transition in thermoregulatory effectiveness may occur during puberty. Falk and colleagues (57), however, did not show these expected thermoregulatory differences between pre-, mid-, and late-pubertal boys during exercise in dry heat. Their findings further indicate the change from childlike to adultlike thermoregulation during exercise in the heat may happen at a later developmental stage, but not during puberty. Several investigators report similar thermoregulation and heat tolerance for children and adults (44,47,52), while other investigators report impaired thermoregulation and heat tolerance in children (52,84,234). Bar-Or (12) concludes that many of these discrepancies may be ascribed to different degrees of acute heat stress.

In temperate environments, children appear to thermoregulate as effectively as young adults although children rely more on dry heat loss rather than evaporative heat loss mechanisms (44,52). In warm environments where ambient temperature is not greater than 5-7°C above skin temperature and the relative humidity is not high, the thermoregulatory responses of children and young adults are also similar during acute-heat stress (47,52,84). However, in very hot environments where ambient temperature is more than 10°C higher than skin temperature (i.e., greater than ~45°C), children compared to young adults display a lower heat tolerance, with elevated body temperatures and a lower sweating rate during acute-heat stress (52,84,234). Bar-Or (12) suggests that the higher surface area-to-mass ratios, lower sweating rates, and less effective cardiovascular adjustments in children during exercise in the heat contribute to their impaired thermoregulation in very hot environments.

Prepubertal and postpubertal boys display the ability to acclimate during repeated exercise-heat exposure through a decreased heart rate and rectal temperature, increased sweating rate, and improved exercise-heat tolerance (234). However, the rate and degree of heat acclimation achieved is not as great for the pre-pubertal boys when likened to the postpubertal boys and young men. Even younger boys (8-10 yr) show the ability to acclimate to exercise in the heat, but again they take longer than young men to achieve complete heat acclimation (96).

Adults. The exercise-heat tolerance for middle-aged men and women was reported to be less than for younger adults (50,234). It is unclear from these reports whether this intolerance was the result of age per se or associated with other factors such as poor health, decreased physical activity and/or low aerobic fitness. In contrast, the exercise-heat tolerance of habitually-active middle-aged men (162) or aerobically-trained middle-aged men (149) was equivalent or better than that for comparable groups of younger men. These two studies emphasized the importance of a habitually-active lifestyle or high aerobic fitness and pertinent anthropometric factors such as body fat, body weight and surface area (A_D) in maintaining exercise - heat tolerance with aging.

For the general population of middle-aged men and women, physiological heat strain during exercise was somewhat greater than that observed for younger adults,

particularly in the heat (50,86,87,88,119,210,234). The greater physiological heat strain for these middle-aged compared to younger persons was associated with higher core and mean skin temperatures, heart rate and skin blood flow, as well as lower total body sweat losses and/or altered local sweating responses. While resting or performing intermittent exercise-rest cycles during an acute heat exposure (49,51), the physiological heat strain seemed not to differ as greatly between middle-aged and younger men or women as during continuous exercise in the heat (50,234). The heat exposure length appeared to influence the differences in heat strain between adult age groups, as prolonged exposures resulted in accentuated heat strain for these middle-aged individuals (86,88). More dramatic differences in heat strain between age groups were observed at higher levels of environmental heat stress such as those exceeding the prescriptive zone (50,119). When training state or aerobic fitness and selected pertinent anthropometric factors were not matched between age groups, the observed heat strain differences between groups were accentuated.

When middle-aged and younger men and women were matched for aerobic fitness and anthropometric factors (such as A_D, A_D-to-weight ratio and/or percent body fat), the heat strain between age groups was generally the same or less for middle-aged than younger persons during acute exercise-heat stress (2,102,149). Recently, Tankersley and colleagues (229) reported no significant differences in thermoregulation between younger and middle-aged or more elderly men who were matched for aerobic fitness, body weight, percent body fat and A_D-to-weight ratio while performing exercise in a warm environment. These observations seemed to be valid for both genders through the fifth, sixth and seventh decades of life, and to apply to both desert and warm-humid conditions.

Clearly, middle-aged men or women can acclimate to exercise in the heat; however, questions remain regarding the rate and/or degree of acclimation achieved by middle-aged compared to younger persons (147). Two studies reported that middle-aged men acclimated to heat, in terms of thermoregulatory responses, at the same rate and to the same degree as younger aged men (149,162). Both of these studies evaluated either habitually-active or aerobically-trained middle-aged men. Two other studies reported that middle-aged men or women also acclimated to exercise-heat stress (2,234); but the degree of heat acclimation was not as great for the

middle-aged as younger persons. Experimental design limitations in the study by Wagner and colleagues (234) created some difficulties in comparing acclimation responses between age groups.

While these reported discrepancies can not be resolved presently, it seems plausible that middle-aged and younger women respond more adversely to a dry-heat challenge than middle-aged and younger men (207). During dry-heat exposures (49°C, 20% rh; 54°C, 10% rh) or desert walks (40-44°C), thermoregulatory advantages are observed for men compared to women (149,257). It is likely that these gender-related differences could help explain some of the discrepancies among these reports, and possibly that middle-aged women are at an even greater thermoregulatory disadvantage while performing physical work in dry heat.

Spinal Cord Injured

Spinal cord injury (SCI) impairs a person's ability to thermoregulate (194). The magnitude of impairment is related to the level and completeness of the lesion; the higher and more complete the SCI, the greater the thermoregulatory impairment. Normell (142) has defined the areas with loss of cutaneous vasomotor and sweating function for a given lesion level. The consequence of the loss of sympathetic control of heat loss (via vasomotor and sudomotor adjustments) over large areas of skin is a higher core temperature during rest (6) and exercise (59,94) in the heat.

Investigators have performed detailed analyses of SCI subjects' thermoregulatory sweating and cutaneous blood flow responses to passive heat exposure (71,227). During heat exposure, some sweating can occur over the insensate skin but is sparse and not synchronous with sensate skin sweating. SCI individuals have reduced local sweating responses for a given core temperature during passive heat exposure (227). Similar to the sweating response, cutaneous (forearm) blood flow is lower for SCI individuals (compared to able-bodied) for a given core temperature during passive heat exposure (71). Freund and colleagues (71) found that when insensate skin was heated, there was no increase in forearm blood flow (FBF); but the heating of sensate skin increased FBF by a relatively small amount. These investigators suggested that isolation of thermoreceptors in areas past the

lesion for SCI might result in reduced afferent input to the hypothalamic thermoregulatory centers, resulting in reduced effector drive for sweating and cutaneous vasodilation.

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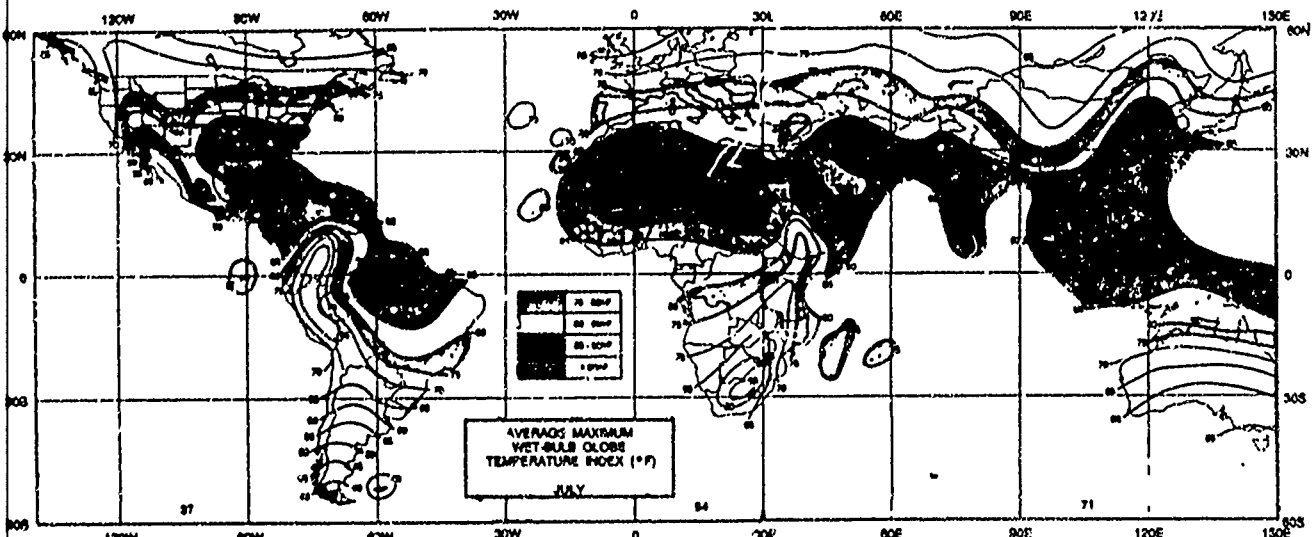
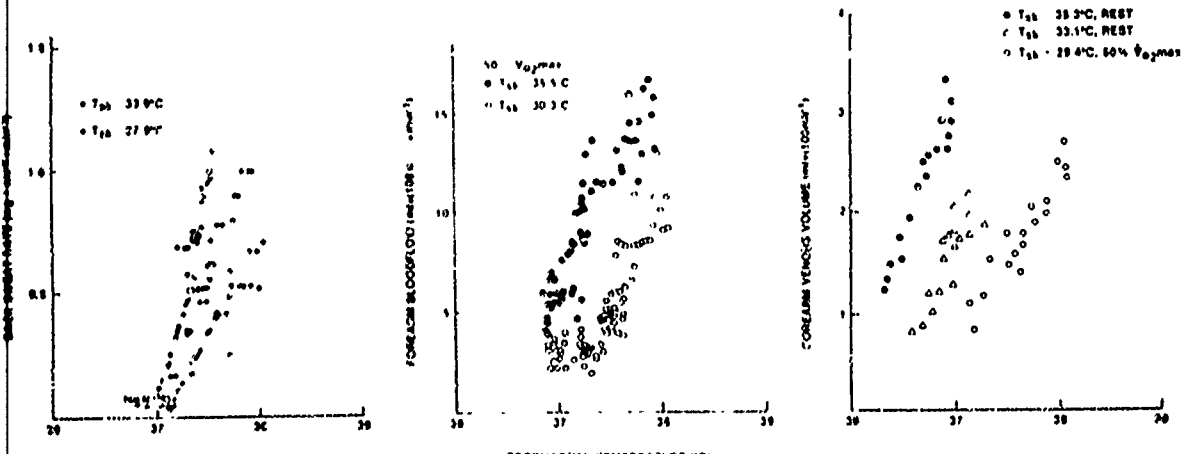
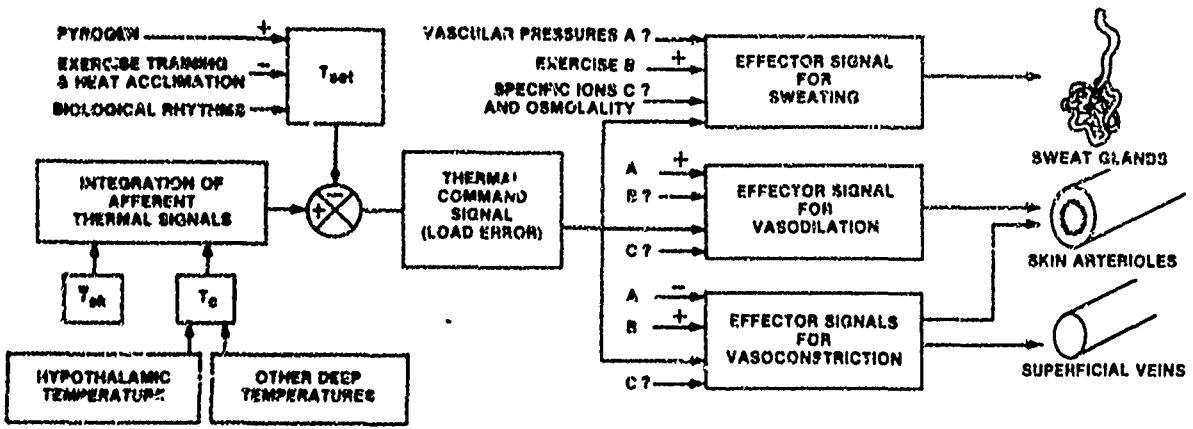
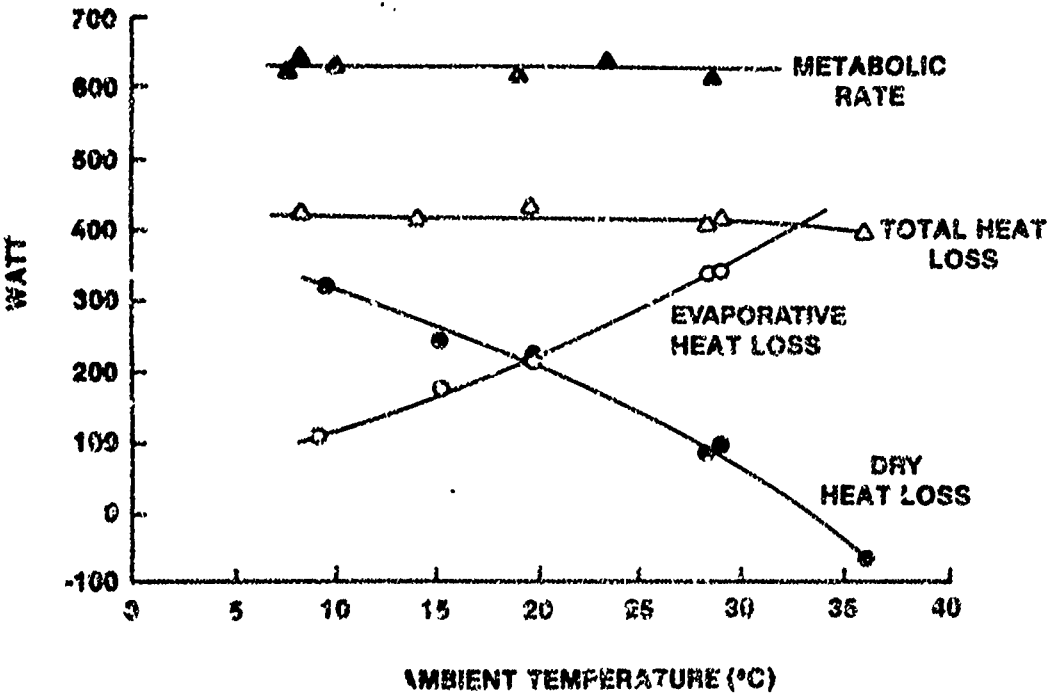
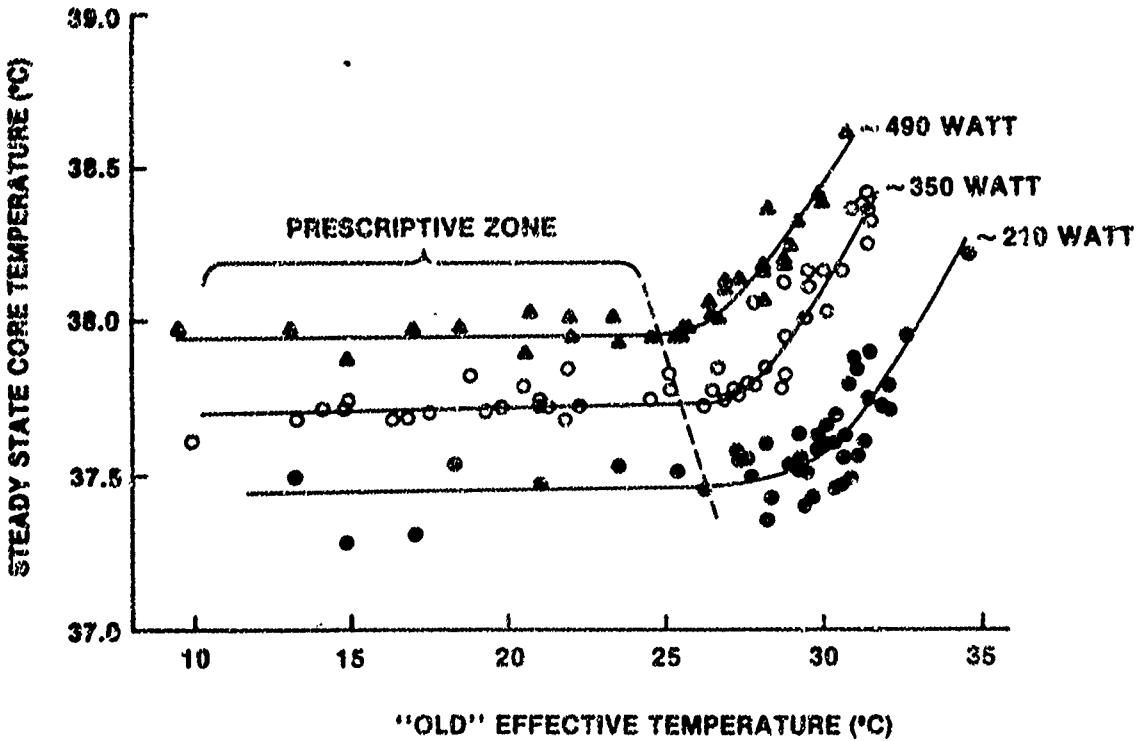
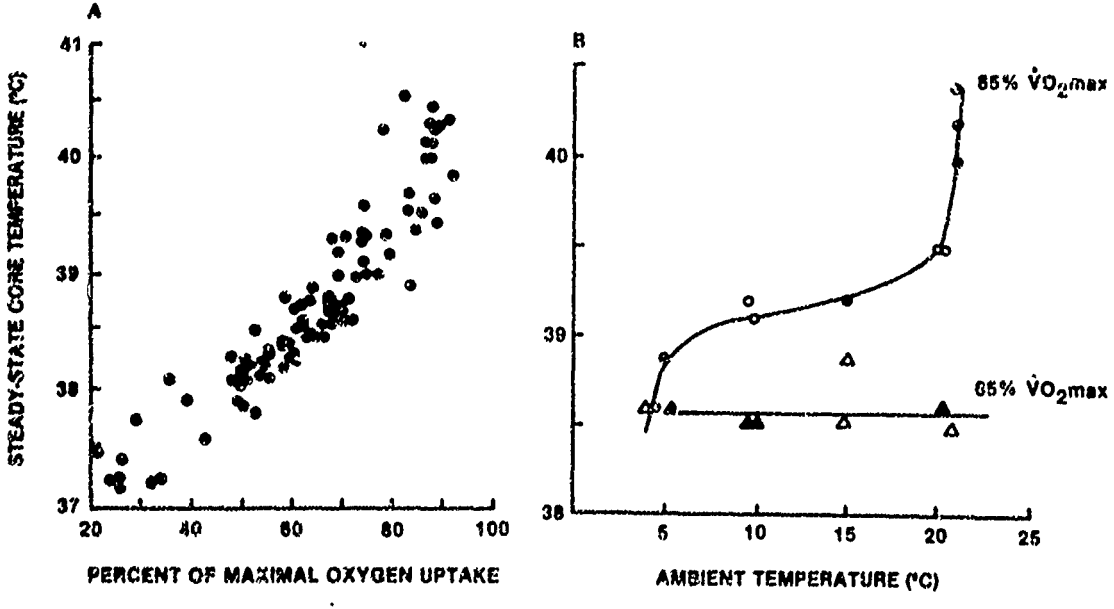

120W
90W
60W
30W
0
30E
60E
90E
120E
150E
60N
30N
0
30S
60S
AVERAGE MAXIMUM
WET-BULB GLOBE
TEMPERATURE INDEX (°F)
JULY
97
94
71

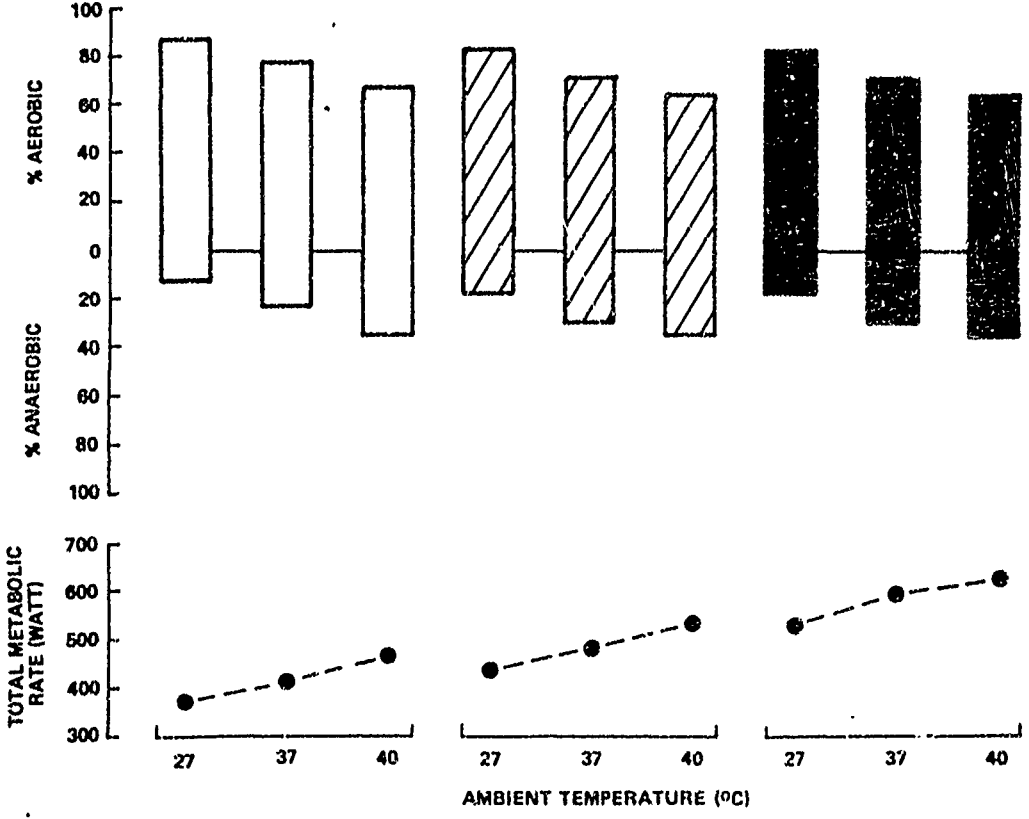

• T_{sk} 33.9°C
• T_{sk} 27.9°C
10 V_{O_2max}
• T_{sk} 35.1°C
○ T_{sk} 30.3°C
• T_{sk} 35.3°C, REST
○ T_{sk} 33.1°C, REST
○ T_{sk} = 29.4°C, 60% $\dot{V}_{O_2max}$
FOREARM BLOODFLOW
FOREARM VENOUS VOLUME
ESOPHAGEAL TEMPERATURE (°C)


PYROGEN
+
EXERCISE TRAINING
& HEAT ACCLIMATION
−
BIOLOGICAL RHYTHMS
T_{set}
INTEGRATION OF
AFFERENT
THERMAL SIGNALS
T_{sk}
T_c
HYPOTHALAMIC
TEMPERATURE
OTHER DEEP
TEMPERATURES
+
−
THERMAL
COMMAND
SIGNAL
(LOAD ERROR)
VASCULAR PRESSURES A ?
EXERCISE B
+
SPECIFIC IONS C ?
AND OSMOLALITY
EFFECTOR SIGNAL
FOR
SWEATING
A
+
B ?
C ?
EFFECTOR SIGNAL
FOR
VASODILATION
A
−
B
+
C ?
EFFECTOR SIGNALS
FOR
VASOCONSTRICTION
SWEAT GLANDS
SKIN ARTERIOLES
SUPERFICIAL VEINS

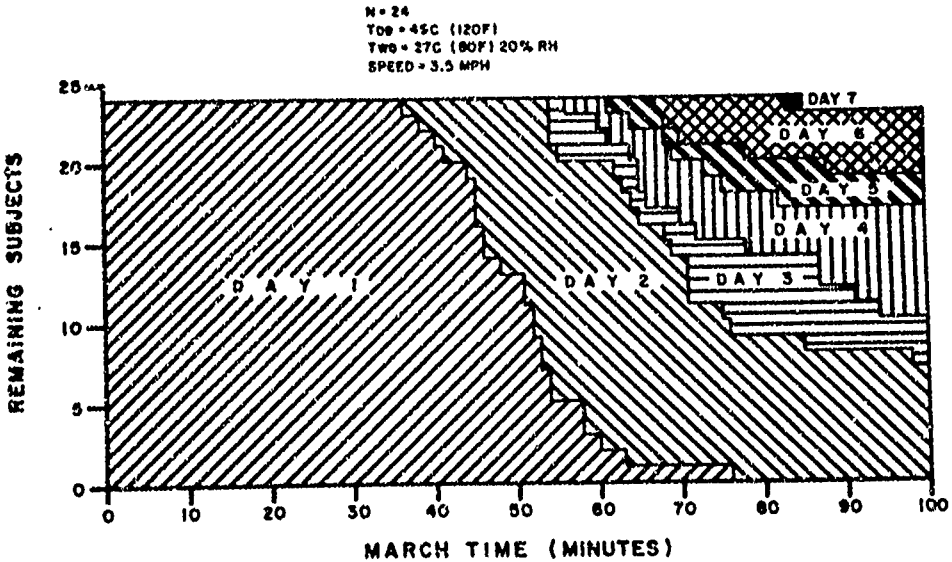

700
600
500
400
300
200
100
0
-100
WATT
METABOLIC RATE
TOTAL HEAT LOSS
EVAPORATIVE HEAT LOSS
DRY HEAT LOSS
0
5
10
15
20
25
30
35
40
AMBIENT TEMPERATURE (°C)

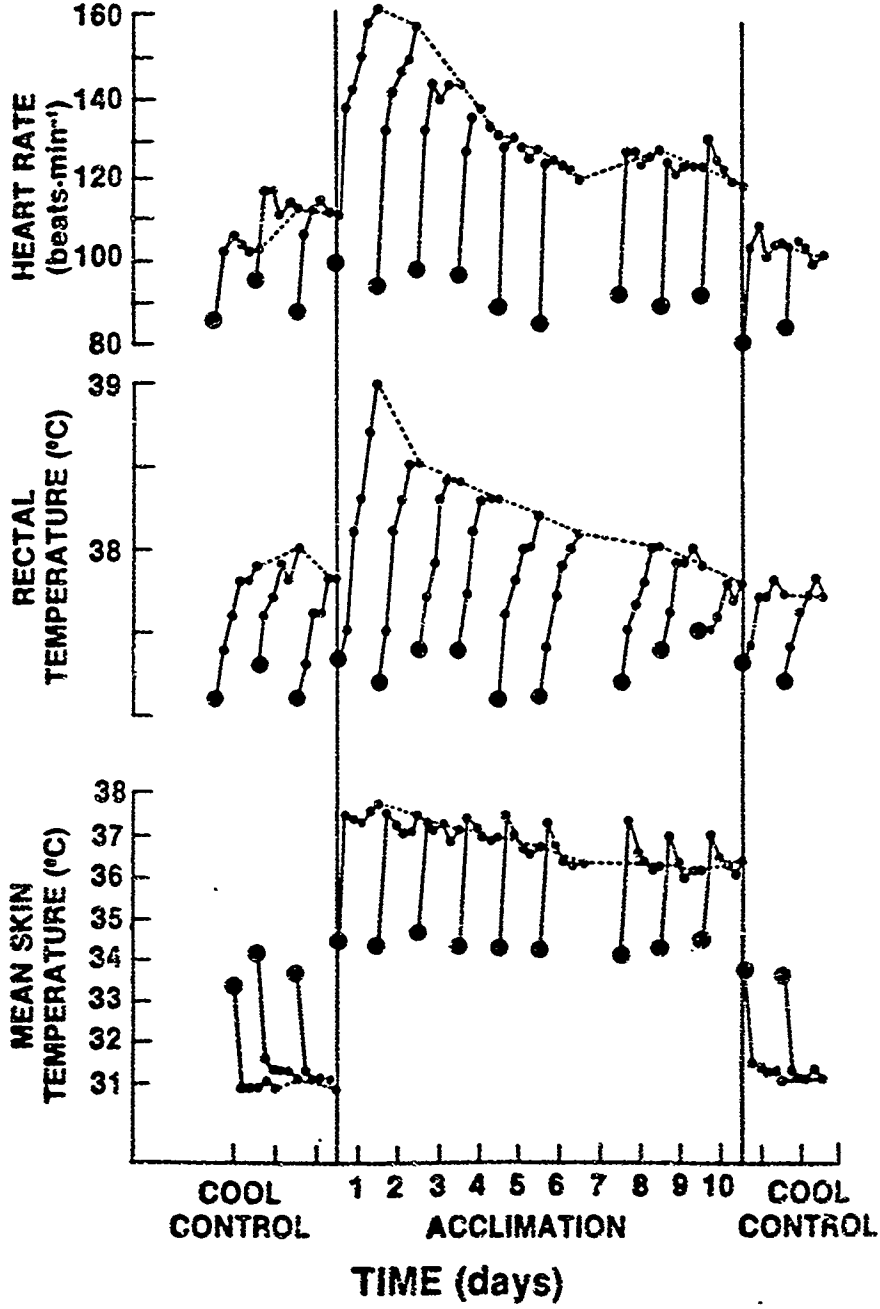

39.0
38.5
38.0
37.5
37.0
STEADY STATE CORE TEMPERATURE (°C)
PRESCRIPTIVE ZONE
~490 WATT
~350 WATT
~210 WATT
10
15
20
25
30
35
"OLD" EFFECTIVE TEMPERATURE (°C)

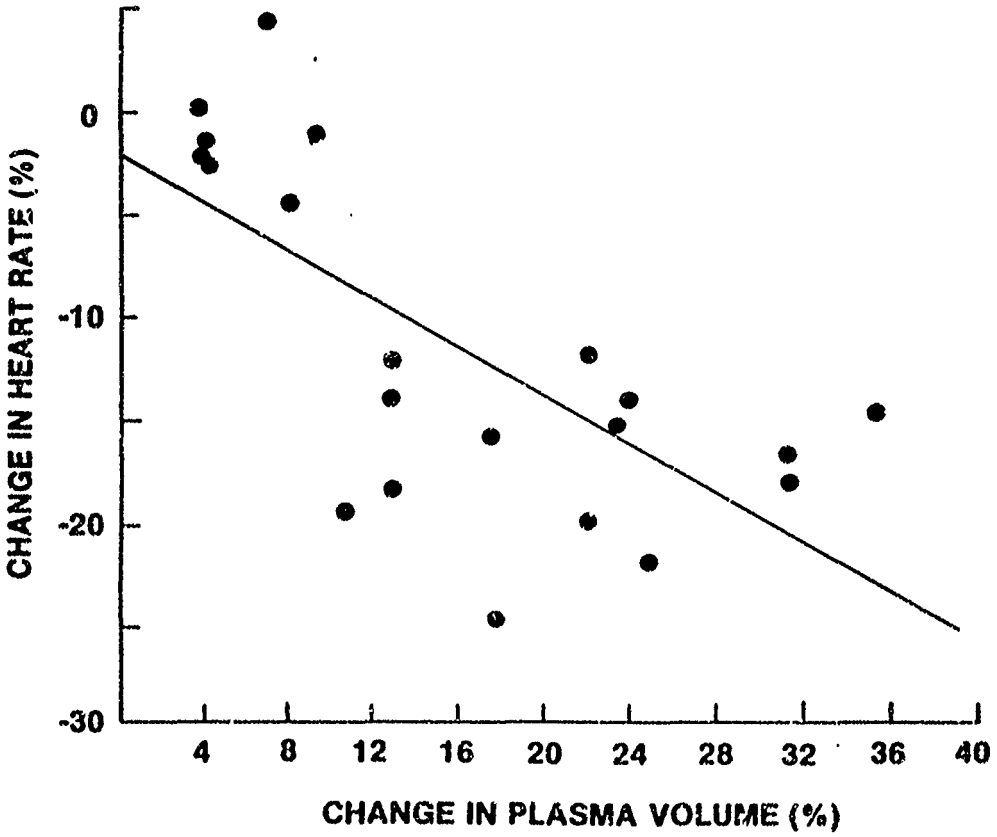

A
41
40
39
38
37
20
40
60
80
100
STEADY-STATE CORE TEMPERATURE (°C)
PERCENT OF MAXIMAL OXYGEN UPTAKE
B
40
39
38
5
10
15
20
25
85% $\dot{V}O_2$max
65% $\dot{V}O_2$max
AMBIENT TEMPERATURE (°C)

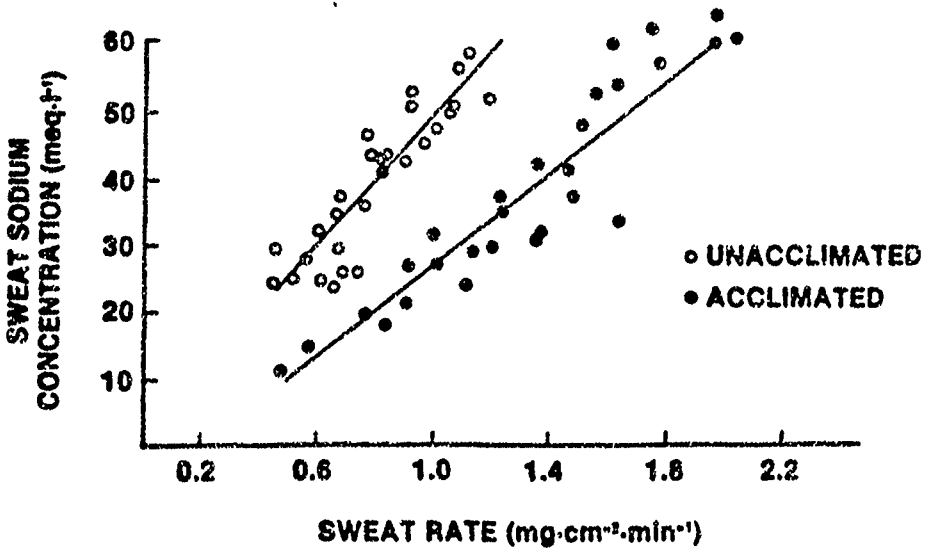

100
80
60
40
20
0
20
40
60
80
100
% AEROBIC
% ANAEROBIC
700
600
500
400
300
TOTAL METABOLIC RATE (WATT)
27
37
40
27
37
40
27
37
40
AMBIENT TEMPERATURE (°C)

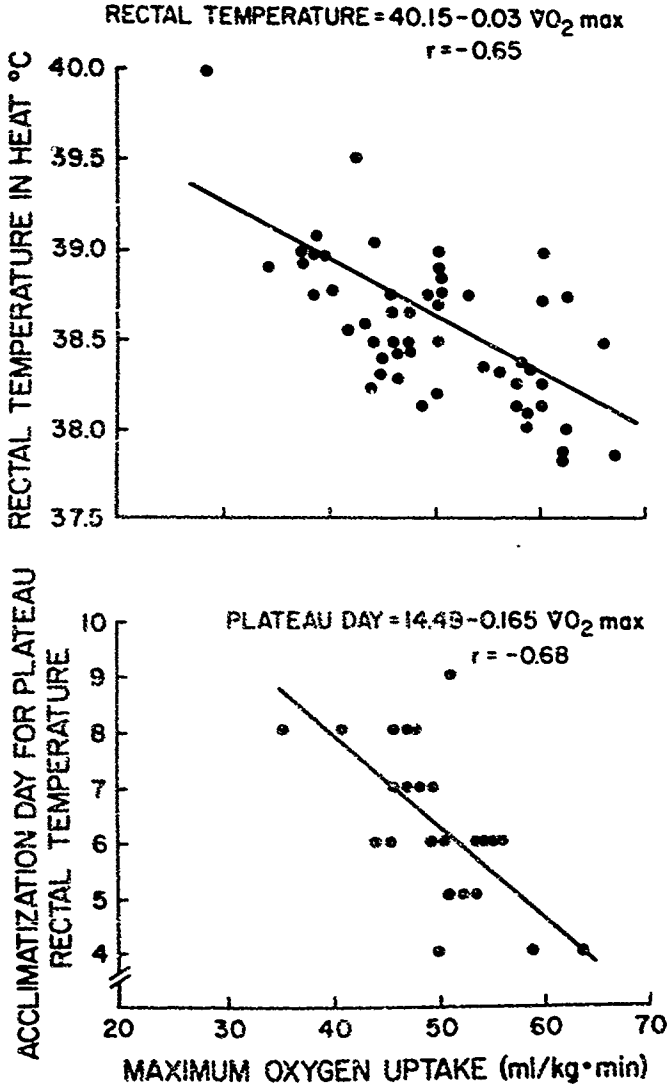
WORK TOLERANCE IN HEAT DURING 7 DAYS ACCLIMATION

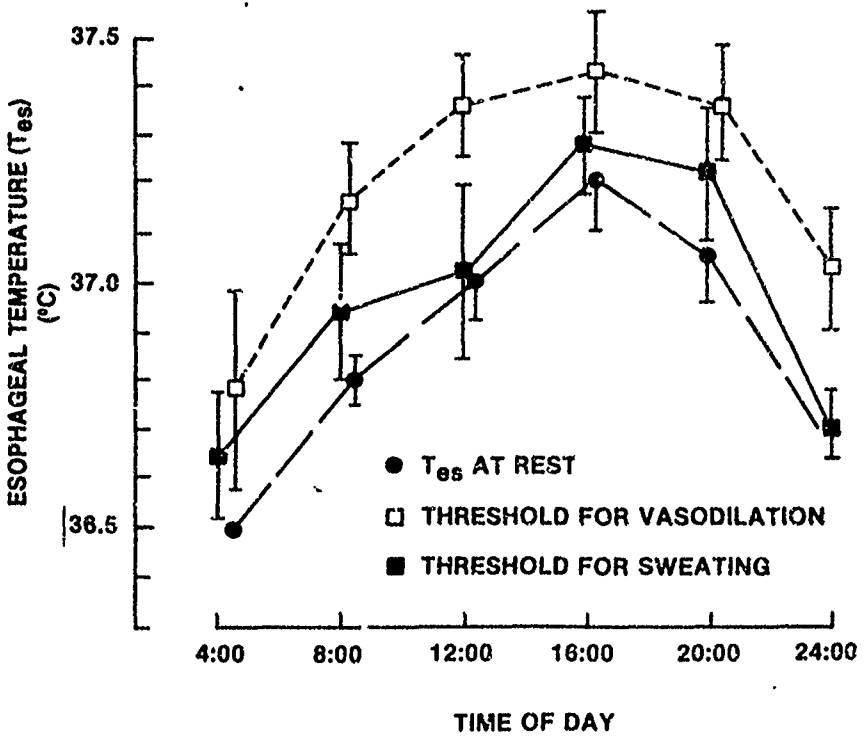



HEART RATE (beats·min⁻¹)
160
140
120
100
80
RECTAL TEMPERATURE (°C)
39
38
MEAN SKIN TEMPERATURE (°C)
38
37
36
35
34
33
32
31
COOL CONTROL
1 2 3 4 5 6 7 8 9 10
ACCLIMATION
COOL CONTROL
TIME (days)


CHANGE IN HEART RATE (%)
0
-10
-20
-30
4
8
12
16
20
24
28
32
36
40
CHANGE IN PLASMA VOLUME (%)


SWEAT SODIUM CONCENTRATION (meq·l⁻¹)
60
50
40
30
20
10
0.2
0.6
1.0
1.4
1.8
2.2
SWEAT RATE (mg·cm⁻²·min⁻¹)
o UNACCLIMATED
• ACCLIMATED


RECTAL TEMPERATURE = 40.15 - 0.03 $\dot{V}O_2$ max
r = -0.65
RECTAL TEMPERATURE IN HEAT °C
40.0
39.5
39.0
38.5
38.0
37.5
PLATEAU DAY = 14.49 - 0.165 $\dot{V}O_2$ max
r = -0.68
ACCLIMATIZATION DAY FOR PLATEAU RECTAL TEMPERATURE
10
9
8
7
6
5
4
20
30
40
50
60
70
MAXIMUM OXYGEN UPTAKE (ml/kg•min)


37.5
37.0
36.5
ESOPHAGEAL TEMPERATURE (T_{es}) (°C)
T_{es} AT REST
THRESHOLD FOR VASODILATION
THRESHOLD FOR SWEATING
4:00
8:00
12:00
16:00
20:00
24:00
TIME OF DAY

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MEMORANDUM FOR ADMINISTRATOR, DEFENSE TECHNICAL INFORMATION CENTER, ATTN: DTIC-OCC/DELORES CAMPBELL, CAMERON STATION, ALEXANDRIA, VA 22304-6145

SUBJECT: Erratum to Technical Report, "Human Responses to Exercise-Heat Stress"

1. A technical error has been discovered on pages 3 and 4 in the subject printed technical report.

2. Request the enclosed erratum sheets be substituted in DTIC ADA272581 to correct the original technical previously forwarded to the Defense Technical Information Center.

3. Point of contact for this action is Mrs. Judy Pawlus, DSN 343-7322.

Encl

CAREY O. LEVERTT
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ERRATA AD-A272 581

INTRODUCTION

Environmental heat stress increases the requirements for sweating and circulatory responses to dissipate body heat, when the environment is warmer than the skin it also causes the body to gain heat from the environment, and thus increases the amount of heat that the body must dissipate. In addition, muscular exercise increases metabolic rate above resting levels, and thus also increases the rate at which heat must be dissipated to the environment to keep core temperature from rising to dangerous levels. Environmental heat stress and muscular exercise therefore interact synergistically, and may push physiological systems to their limits This technical report provides an overview of the normal physiological responses to environmental and exercise-related heat stress, with emphasis placed on acute heat exposure and acclimation produced by repeated exposure to heat stress. More complete reviews of some material covered in this technical report can be found in other recent reviews (4,129,168,195,197,198,219,240,254).

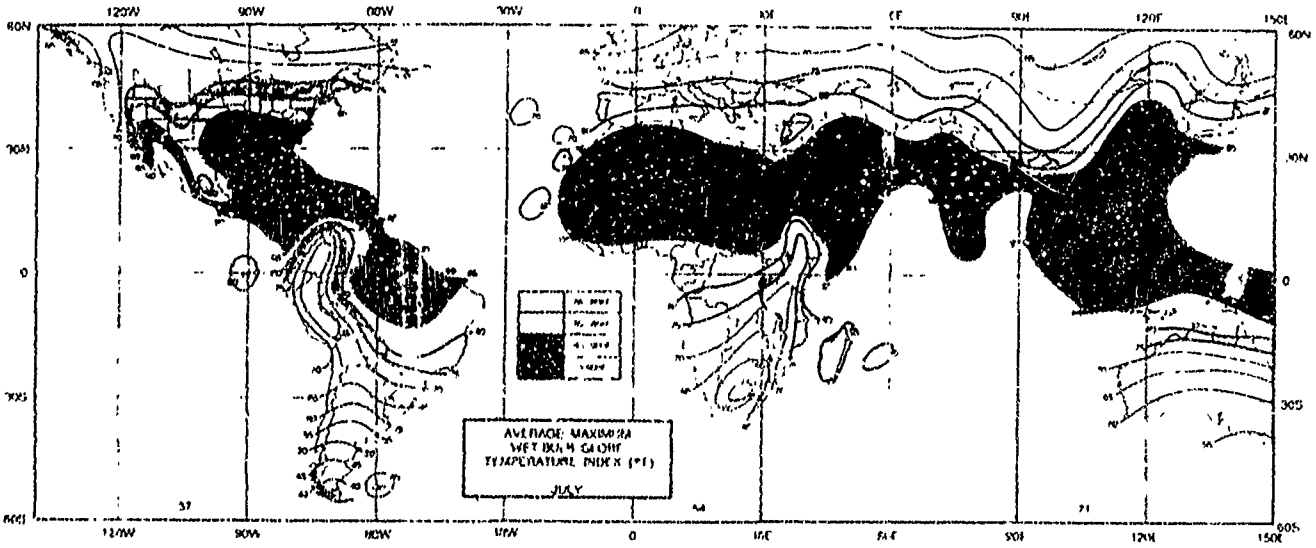
HEAT STRESS

Humans are tropical animals, and it is primarily through cultural and behavioral means that they have adapted to life in temperate and cold environments (65,123,137). This generalization is based on the following evidence. (a) humans rely primarily on physiological thermoregulation in the heat, but primarily on behavioral thermoregulation in the cold (b) the thermoneutral ambient temperature for nude humans and the temperature necessary for undisturbed sleep are relatively high (~27 C), and (c) humans demonstrate substantial heat acclimatization, but only modest cold acclimatization.

Hot climates are present over large areas of the earth and are tolerated well by humans Figure 1 is a global map of Wet Bulb Globe Temperature (WBGT)[1] during July, the hottest month in the northern hemisphere (97). During July much of North America, South America, Europe and Asia have WBGT values above 29°C (85°F).

---

[1] WBGT is a computed index of environmental heat stress. Outdoors WBGT = 0.7 wet bulb + 0.2 black globe + 0.1 dry bulb, indoors WBGT = 0.7 wet bulb + 0.3 black globe



19

FIGURE 1. Wet Bulb Glob Temperature (WBGT) during July in the northern hemisphere (4).