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be transported, 2) less muscle mass and strength, and 3) a disproportionately smaller circulating blood volume. Women have lower blood hemoglobin concentrations, which necessitates higher cardiac output from their smaller hearts for equivalent muscular work, resulting in higher heart rates. In the heat, they generally show 1) relatively more peripheral blood pooling when vasodilated, 2) a greater increase in heart rate, 3) a greater tendency for circulatory embarrassment, 4) lower maximal sweat rates, 5) higher skin temperatures with greater body heat storage, 6) lesser ability to maintain circulating blood volume and 7) greater effects from dehydration. A smaller proportion of women than men can be acclimated to heat, but they successfully achieve thermoregulation at similar core temperatures with less body water loss (particularly if physically trained), but still requiring higher heart rates and body heat storage. In the cold, women generally have 1) lower extremity and average skin temperatures (particularly the foot), 2) greater surface heat loss, especially from the markedly thinner cylinders of the extremities, 3) increased rates of extremity (but not core) cooling, 4) a more extensively vasoconstricted shell, 5) less capability for maximal heat production by either shivering or exercise, and 6) relatively greater risk of cold injury. Because of these differences, it is even more essential for women than men that they be given adequate time for adaptation to thermal stress and, where possible, be provided appropriate environmental protection. In particular, heat stress requires maximizing evaporation of the limited sweat production, engineering of activity levels to minimize muscular heat production and providing sufficient cool breaks to permit development of acclimation. Cold stress requires providing adequate clothing insulation, maintaining appropriate muscular activity to maintain circulatory heat input to the periphery and providing adequate facilities and time for checking and rewarming over-cooled extremities. Despite their relative disadvantages, women have shown adaptation to severe environments provided that their lesser muscular capabilities and smaller heart volumes are not over-taxed prior to adaptation.

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**A Comparison of the Physiological Responses in Young Men and Women  
to Heat and Cold Stress**

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**August 1978**

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Abstract

*S*  
The current literature on male-female differences in response to thermal stress has been reviewed. Morphological differences of women (20% smaller body mass, 14% more body fat, 33% less lean body mass, but only 14-22% less surface area) impinge greatly on their relative ability to balance body heat production and losses. Women have greater body insulation against thermal transients when fully vasoconstricted (except on hands and feet) and a greater peripheral body "shell" for a heat sink, but at the cost of 1) a greater burden of body fat to be transported, 2) less muscle mass and strength, and 3) a disproportionately smaller circulating blood volume. Women have lower blood hemoglobin concentrations, which necessitates higher cardiac output from their smaller hearts for equivalent muscular work, resulting in higher heart rates. In the heat, they generally show (1) relatively more peripheral blood pooling when vasodilated, 2) a greater increase in heart rate, (3) a greater tendency for circulatory embarrassment, (4) lower maximal sweat rates, (5) higher skin temperatures with greater body heat storage, (6) lesser ability to maintain circulating blood volume and (7) greater effects from dehydration. *2 over* A smaller proportion of women than men can be acclimated to heat, but they successfully achieve thermoregulation at similar core temperatures with less body water loss (particularly if physically trained), but still requiring higher heart rates and body heat storage. In the cold, women generally have 1) lower extremity and average skin temperatures (particularly the foot), 2) greater surface heat loss, especially from the markedly thinner cylinders of the extremities, 3) increased rates of extremity (but not core) cooling, 4) a more extensively vasoconstricted shell, 5) less capability for maximal heat production by either shivering or exercise, and 6) relatively greater risk of cold injury.

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Key words: human male-female differences, environmental stress, sex factors, thermoregulatory mechanisms.

## I. Introduction

The relatively recent entry of significant numbers of women into physically demanding occupations and athletic activities traditionally performed by men has resulted in the need for separating real from imaginary differences between the sexes. Within the real differences there is a further need for separating those based on biology from those based on social or cultural conditioning.

The most apparent differences between the sexes are morphologically and physiologically associated with the differences in reproductive function. These fundamental differences have induced the suspicion that behavioral and biological responses to stress agents will may differ in kind, as well as degree, between the sexes. It is the purpose of this paper to succinctly review the current literature on differences between men and women in their physiological and subjective responses to environmental heat and cold stress. Where possible, methods will be suggested whereby real differences may be minimized in order to assist the planner, designer, architect and engineer in providing environments which are suited to as much of the user population as is realistically feasible. Age has been shown to have marked effects on some biological functions; because of the scarcity of research results on older people, the differences in function herein discussed will be based on those reported for mature, young adults in the age range extending roughly from the late teens through the early forties.

## II. Body Heat Balance

Maintenance of a constant internal temperature in homeotherms requires that body heat gains and losses be balanced. The body gains heat principally by means of radiation and convection (R and C, respectively) from an environment

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warmer than skin temperature and from the heat of metabolism (M) not used to perform physical work (W) upon the environment. The body loses heat principally by means of R and C to an environment cooler than skin temperature and by evaporation of water (E), either from the respiratory mucosa or from sweat diffusing through or actively secreted on the skin surface. The equation for thermal equilibrium is therefore:

$$M - W + R + C - E = 0 \quad (1)$$

If the result is not equal to zero, body heat storage or debt results.

Heat is circulated throughout the body via the blood, from warmer to cooler regions, but can be exchanged with the environment only at the body surface. Therefore, in addition to providing nutrients to metabolically active tissue, blood flow is required for exchange of heat between the body's surface and core regions. Blood flow is modulated by active constriction or dilatation of the blood vessels from their state under conditions of heat balance when the body is at rest, and is responsive both to metabolic demands and to thermal stimuli.

#### A. Mechanisms of heat production and conservation.

In a cold environment the physiological basis of heat balance rests on the body's ability to produce and conserve heat. The various body tissues do not have equal metabolic rates; heat production principally derives from the "metabolically active" tissues: the internal organs, which produce heat at slightly variable rates, and the skeletal muscles, which have a 10-fold variability in capability for heat production. Bone, connective and nerve tissue and fat are all metabolically active, but at relatively constant rates which are much lower than those of the muscles and internal organs. Heat production of the muscles can result from either the voluntary contractions of physical exercise or by the involuntary contractions



of shivering in response to neural mediation of cold thermal stimuli. The overall metabolic rate of humans has been observed to increase in cold environments prior to the onset of overt shivering; whether such "non-shivering" thermogenesis is due to a generalized increase of muscle tone or to endocrine stimulation of cellular metabolism, or a combination of both, is not yet clear.

Heat conservation is achieved physiologically by reducing the amount of heat transferred to the skin from the body core by constriction of the cutaneous blood vessels, thereby increasing the cutaneous tissue insulation, and by reducing blood flow to all the tissues of the limbs and allowing them to cool, thereby reducing conductive heat losses to the limb surfaces. In the cold, the extent of the warm body core is reduced while the surrounding cooler shell is enlarged. In addition, there are behavioral responses: attempting to reduce the exposed surface area; seeking sheltered locations; huddling in groups; and, in man, altering the microenvironment by adding external insulation or developing an external heat source.

#### B. Mechanisms of heat dissipation

In a hot environment, heat balance depends upon the ability to dissipate the heat from the environment and that resulting from metabolism. Dissipation of metabolic heat requires blood flow from the core to a cooler skin. Dissipation of environmental heat and maintenance of a skin which is cooler than core requires both the production and evaporation of adequate sweat, which are limited by the body's maximum rate of sweat secretion and the environment's maximum capacity for evaporation, a function of wind speed and water content. Physical activity in hot environments requires partitioning of the available blood flow from

the heart (cardiac output) to support not only tissue nutrition of the basal metabolism and the added muscular work but also the requirements for heat transfer to the skin. The total cardiac output available may not be adequate to maintain blood pressure in the face of the sum of these separate demands, inducing either cardiovascular instability or inadequate oxygenation of the central nervous system and eventual collapse. In addition, the vascular volume is the source of the water in sweat; inadequate replacement of body water losses diminishes the available blood volume despite partial compensation by shifts of interstitial and intracellular fluid.

Under conditions of environmental heat stress with no restriction to evaporation ("dry heat"), thermal balance can be achieved when the body temperature rises just far enough to stimulate an adequate sweat rate, as long as the available cardiac output can support requirements for both tissue nutrition and heat dissipation.

When a hot environment also imposes a restriction to the evaporation of sweat secreted (humid or "wet" heat), the stress is much greater. Thermal balance requires that the body temperature rise far enough to induce a sweat rate in excess of that which would achieve thermal balance under full evaporation; much sweat drips off and is wasted. This insures that the skin surface will be completely wetted, irrespective of regional differences in sweat rate, and that whatever evaporation is possible will have a maximum effect. However, high rates of sweating cannot be long maintained. Also, the skin is not cooled as much as it is in dry heat; removal of metabolic heat from the core to the skin requires more cutaneous blood flow to compensate for the diminished thermal gradient, exacerbating the impact of inadequate water intake.

Acclimation to work in dry heat results in an improvement in the ability to maintain or enlarge the vascular volume during exposure and thereby augment venous return. This enhances stroke volume and diminishes heart rate for the required cardiac output. There is also some increase in sweat rate (which usually is not the limiting factor, anyway), which can be achieved at reduced core (and often skin) temperatures. Acclimation to work in wet heat results not only in an improved ability to maintain or enlarge the vascular volume during exposure, with the concomitant diminution in heart rate, but also a markedly enhanced capacity for sweat secretion at lower core and skin temperatures.

C. Change in body heat content

Failure to achieve heat balance results in a change in body heat content, either storage or debt. In a warm environment, heat can be transferred by augmented blood flow from the body core region to peripheral regions in the extremities with little change in core or skin temperatures. This enlarges the body core at the expense of the shell. Only the relative mass ratio of body core-to-shell changes. In this way the body shell serves as a peripheral heat sink. The converse is also possible, with one difference. In a cool environment arterial inflow to the surface and the interior of the extremities can be reduced, while core temperature remains constant; this enlarges the body shell as the core region contracts. However, maintaining body core temperature at the expense of the shell is usually detectable (unlike the heat sink situation). As the interior of the limbs cool the skin temperature also falls, revealing the change in body heat content. Active thermoregulation by means of the vasomotor changes just described makes calculation of mean body temperatures (MBT) very difficult, as it is difficult to assign precise proportional weights to the body core and shell regions, and the average

temperature of the shell must be inferred from the skin ( $T_{sk}$ ) and core ( $T_{re}$ ) temperatures. Several different weighting schemes have been proposed for different stress situations:

$$\text{In heat, full vasodilatation: } MBT = 0.9 T_{re} + 0.1 T_{sk} \quad (2)$$

$$\text{Thermoneutral: } MBT = 0.67 T_{re} + 0.33 T_{sk} \quad (3)$$

$$\text{In cold, full vasoconstriction: } MBT = 0.5 T_{re} + 0.5 T_{sk} \quad (4)$$

Unfortunately, these do not adequately describe situations of vasomotor flux. However, they have proven useful in describing the limiting cases.

When heat storage or debt occurs beyond the capability of the periphery to compensate, body skin and core temperatures change (usually in that order), reflecting the change in body heat content. There are upper and lower limits to  $T_{re}$  which remain compatible with safety: 40-41°C and 33-35°C. There are also limits to skin temperature: At about 42°C pain results, while cell water freezes at just below 0°C (although supercooling to -5°C can occur). In heat storage situations, core temperature elevation is usually the limiting factor, although intense environmental heat may cause burns or pooling of blood in the cutaneous vasculature may impair cardiac output. In heat debt situations, depression of local skin temperature of the extremities to freezing levels (especially in wet and/or windy conditions) is usually the limiting factor, although prolonged cooling at above-freezing temperatures can injure the vasculature (circulatory stasis) or induce core temperature depression below the limits of normal thermoregulatory function (clinical hypothermia).

### III. Morphological differences between women and men affecting thermoregulation

In addition to the physiological differences between women and men which impact on thermoregulation, there are two morphological differences which exert

a great effect on their relative abilities to produce heat internally and to receive heat from, or transfer heat to, the environment. Women have smaller body sizes than men from the same population base; they also have a greater percentage of their body masses as body fat, unless they are highly trained athletes. The body masses of women (mostly aged 17-30) in the U.S. military services average from 77-81% those of men in the same services; 80% is an acceptable average (Hertzberg, 1972; Churchill, Churchill, McConville and White, 1977; McConville, Churchill, Churchill and White, 1977). A large sample of 17-29 year old women ranged from 13-35% body fat content, averaging 29%; males of the same age ranged from 10-26%, averaging 15% (Durnin and Womersley, 1974). Since fatty tissue is not very active metabolically, the woman is left at a double disadvantage: her total mass is only 80% that of the male and almost twice as much of that lesser mass is the metabolically less active fat. On average, her fat-free "active" body mass is only two-thirds as large as that of her male counterpart. Unfortunately, her body surface area is not decreased proportionately; it is calculated to average 78-85% that of men (DuBois and DuBois, 1916). Not surprisingly, her maximum oxygen consumption during severe muscular work, and therefore her maximum heat production, also averages only two-thirds that of men (Åstrand, 1960). However, there is a fairly extensive range of overlap between the sexes in maximum oxygen consumption, which becomes progressively less as lifestyles become more active. Among non-athletic students, 76% of the women equal or exceed 47% of the men; among active students, 32% of the women equal or exceed 32% of the men; however, among athletic students, 22% of the women are able to equal or exceed only 7% of the men (Drinkwater, 1973).

These differences in morphology affect thermoregulation in several ways. The smaller body mass of women results in less total heat capacity and thermal

inertia, such that the same number of calories gained or lost will result in a greater change in body heat content. Because of geometric considerations, smaller masses of similar shape have larger surface area-to-mass ratios than do larger masses. Since heat transfer between the body and the environment is a surface phenomenon, women therefore exhibit greater environmental heat transfer per unit body mass.

A. Cold stress

Since maximum heat production is a function of total body mass, in particular the mass of metabolically active tissue, women are at a severe disadvantage in extreme cold. Their heat loss-to-production ratios are greater than men and their greater percentage of body fat further reduces the relative size of their active body mass. The insulation added by the subcutaneous fat layer provides little relative advantage in preventing frostbitten extremities, as it does not cover the body regions most at risk - the hands and feet. Neither does it retard core cooling more during prolonged exposure in air, as the blood vessels in skin, fat and non-working muscle apparently constrict similarly in both sexes. Thus, for the same degree of peripheral vasoconstriction, women have a much smaller central heat content per unit surface area as a result of their larger surface area-to-mass ratio. However, women appear to have a definite advantage in cold water (Sloan and Keatinge, 1973), apparently due to a different distribution of body fat. Women have relatively more leg than trunk fat, while men have more trunk than leg fat. Because women have 1.75 times as much fat thickness as men, they have 25% more fat on the legs than men, while men have only 15% more fat on the torso (Edwards, 1951).

Fortunately, there are physiological compensations for these morphological differences. The basal metabolism of women is greater than that of men, when

expressed either per unit surface area, per unit of lean body mass or per unit of cell mass (Bernstein, Johnston, Ryan, Inouye and Hick, 1956). However, this higher basal heat production leaves even less reserve for maximal heat production from the woman's smaller muscle mass, either involuntarily by shivering or voluntarily by exercise. Fortunately, the layer of subcutaneous fat which offers little protection to the extremities during prolonged exposure is of benefit in insulating the body core, particularly during short term exposure.

**B. Heat stress**

The larger surface area-to-mass ratio which is so disadvantageous to women in the cold results in three advantages in the heat: the ratio of evaporatively cooled surface to metabolically active tissue is greater; the relatively greater shell volume is covered with relatively more subcutaneous fat, providing more insulation and a proportionally larger peripheral heat sink during thermal transients (Fox, Lofstedt, Woodward, Ericksson and Werkstrom, 1969); and the thermally-induced increase in metabolism from elevated core temperature ( $Q_{10}$  effect) is relatively less, as it is proportional to the smaller "active" body mass. During vasodilatation, this insulating layer apparently offers no additional hindrance to heat dissipation from the body core.

But there are also morphological disadvantages under heat stress. Because their much smaller muscle mass is burdened with a proportionally greater non-working mass, heat production of women is relatively greater when transporting the same external load. Blood volume is proportional to lean body mass, rather than to total body mass, but the volume of peripheral tissue to which blood is shunted for the dissipation of internal heat is proportional to surface area. The peripheral blood volume during vasodilatation thus represents a relatively greater fraction of the total volume; the central blood volume is reduced proportionately

more, requiring a greater heart rate to maintain cardiac output. A unit of water lost in sweating represents a greater fraction of the woman's body plasma water, so that the relative effects of dehydration are greater if the lost water is not replaced.

The above morphological differences put women at a relative disadvantage under both heat and cold stress. In the cold, the laws of physics require that they lose heat from a smaller production volume at a greater rate through a larger relative surface area. They must maintain the temperature of a smaller core volume while risking a proportionately greater mass of extremity tissue, which is inadequately covered by a relatively larger mass of insulating tissue, improperly distributed over the regions at risk and adding to their total body burden when moving. In the heat, their smaller blood volume is partitioned to a relatively larger pool in the periphery, from which plasma water is secreted over a relatively larger surface area. This induces relatively greater dehydration, which further diminishes the blood volume available for distribution to an often inadequate and overburdened working muscle mass. Fortunately, this bleak picture painted by the laws of physics, which is common to both men and women of similar morphology, is brightened considerably by some rather subtle differences between men and women in physiological compensation.

#### IV. Differences in Physiological Compensation to Heat Stress

There are five major differences shown by women working under the same conditions of heat exposure as men:

1. Thermoregulatory strain is greater in women, as evidenced by greater rates of heat storage and higher average body temperatures.



2. Except for very fit athletes, cardiovascular strain is greater in women, as evidenced by higher heart rates and more frequent syncopal episodes.

3. The maximal sweat rate per unit body surface area is less than men, despite greater surface area-to-mass ratio.

4. The tolerance time of women under heat exposure is generally less and is often accompanied by greater levels of subjective distress.

5. Apparently a lesser proportion of women than men can be successfully acclimated to severe levels of heat stress, but similar levels of acclimation can be reached by those women who are able to endure the acclimating exposures.

A. Body temperature responses:

The core temperatures achieved by women appear to be the same or slightly higher than those of men in moderate dry or wet heat conditions (Morimoto, Slabochova, Naman and Sargent, 1967; Bittel and Henane, 1975), and even in brief exposures to severe dry heat (Shoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978). Only in severe wet heat are core temperatures markedly higher (Wyndham, Morrison and Williams, 1965), but the differences disappear with acclimation (Hertig and Sargent, 1963; Wyndham, Morrison and Williams, 1965). In the case of women apparently more fit than men (Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967), their core temperatures were lower than men initially and continued so throughout acclimation to wet heat.

On the other hand, average skin temperatures of women are generally 0.6 - 1.20°C higher than men (Hertig and Sargent, 1963; Bittel and Henane, 1975; Shoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978), although there are reports from one laboratory of similar skin temperatures in both wet and dry heat (Morimoto, Slabochova, Naman and Sargent, 1967; Weinman, Slabochova, Bernauer,

Morimoto and Sargent, 1967).

The generally higher skin temperatures of women accompanying core temperatures similar to those of men reduces the core-to-skin temperature gradient available for heat transfer. This results in lower heat conductance, which averages only 65-88% that of men (Bittel and Henane, 1975; Shoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978). Obviously, since the mean body temperature is a weighted average of core and skin temperature (a representative temperature of the body shell, or even its extent, being impossible to ascertain), the calculated body heat content of women tends also to be higher than men when equilibrium is achieved under any given set of conditions.

B. Cardiovascular responses:

There is little dispute that women exhibit higher heart rates than men under any similar set of environmental conditions. The more severe the conditions, the greater the difference (Hertig and Sargent, 1963; Wyndham, Morrison and Williams, 1965; Morimoto, Slabochova, Naman and Sargent, 1967; Fox, Lofstedt, Woodward, Eriksson and Werkstrom, 1969), unless the women are fitter than the men (Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967) or the exposure time is very short (Shoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978). However, with acclimation to heat, these heart rate differences are reduced (Hertig and Sargent, 1963; Wyndham, Morrison and Williams, 1965).

C. Tolerance limits:

There appear to be no sex differences in the levels of heart rate or body temperature which represent the upper limits of tolerance to heat exposure. However, if equilibrium to a set of environmental conditions cannot be reached, women generally reach their thermoregulatory limits quicker and under less stressful conditions than men (Hertig and Sargent, 1963; Wyndham, Morrison and Williams,

1965). Women show the same pattern of response as men during acclimation to heat stress, but at a slower rate and with relatively fewer being able to tolerate the acclimating exposures unless their physical fitness is greater (Hertig and Sargent, 1963; Wyndham, Morrison and Williams, 1965; Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967). Physical fitness increases the ease with which thermoregulation is accomplished and the levels of heat acclimation which can be achieved (Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967; Drinkwater, Denton, Kupprat, Talag and Horvath, 1976; Drinkwater, Kupprat, Denton and Horvath, 1977).

D. Sweating:

Under comparable stress levels, women generally have been observed to exhibit rates of sweating only 30 to 86% those of men. The differences generally increase as the rates of sweating increase, such that the greatest differences occur with maximal sweating in humid heat (Hertig and Sargent, 1963; Haslag and Hertzman, 1965; Wyndham, Morrison and Williams, 1965; Morimoto, Slabochova, Naman and Sargent, 1967 (wet heat only); Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967; Fox, Lofstedt, Woodward, Eriksson and Werkstrom, 1969; Dill, Yousef and Nelson, 1973; Yousef and Dill, 1974; Shoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978). However, equal sweat rates have been reported for men and women under some dry conditions (Morimoto, Slabochova, Norman and Sargent, 1967; Senay, 1973). Women do not exhibit the phenomenon of sweat suppression or hidromeiosis (progressive decline in sweating rates during a single exposure to constant conditions, Hertig, Riedesel and Belding, 1961; Brown and Sargent, 1965) to as great an extent as men; in fact, their sweating rates may progressively increase over the first 2-3 hours of exposure (Wyndham, Morrison

and Williams, 1965; Fox, Lofstedt, Woodward, Eriksson and Werkstrom, 1969). However, physically trained women may show relatively more suppression than men (Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967). Morimoto, Slabochova, Naman and Sargent (1967) found that in dry heat, men exhibited greater sweat rates than women with activation of fewer sweat glands, and progressively increased their sweat rates and numbers of activated glands as the heat stress was increased. However, in wet heat, although men and women had similar numbers of activated glands, the sweat rates of men still exceeded those of women. Both groups showed similar levels of sweat suppression in wet heat. Apparently, men's sweat glands can maintain high flow rates under dry conditions, so that all sweat glands need not be activated until the stress becomes great; women have a lower flow per gland and must therefore activate more glands at lower stress levels. Under conditions of high flow, or in high humidity, the gland flow of both sexes is diminished. This means that the sensitivity of the sweat mechanism in women is less and they either secrete less at the same body temperatures, or must have higher body temperatures in order to maintain the same rate of sweating. Even after full acclimation to humid heat, women are able to maintain only 64-69% the sweat rate of men for 3 hours and 41% during the fourth hour (Wyndham, Morrison and Williams, 1965).

Women also require higher body core and skin temperatures in order to initiate sweating (Hardy, Milhorat and DuBois, 1941; Hertig and Sargent, 1963; Fox and Lofstedt, 1968). This means that higher mean body temperatures and therefore greater heat storage are accomplished before evaporative cooling begins (Bittel and Henane, 1975). The rate at which sweating increases to the steady-state level in women is also less than that of men (Bittel and Henane, 1975). This

places women at an even greater disadvantage as their initial heat storage is greater, becomes progressively more so as sweating begins and is never compensated for during steady-state sweating. Nonetheless, thermal equilibrium is often reached; body core temperatures even may be the same as in men after acclimation (Wyndham, Morrison and Williams, 1965), albeit at the cost of greater cardiovascular strain to maintain adequate core-to-skin heat transfer despite the lower thermal gradient.

E. Core to skin heat transfer:

The evaporation of sweat removes heat from the skin surface; heat must be brought from the working muscles to the skin surface as fast as it is produced or body temperature will continue to rise. The rate of heat transfer from core to skin is termed "conductance" and is an indication of the degree of vasodilatation of the body's peripheral shell (principally the cutaneous vascular bed). It must be kept in mind that skin temperature is the resultant of two processes: The rate at which the skin is cooled by evaporation of sweat (limited by both the maximal ability of the skin to secrete sweat and the maximum capacity of the environment to accept water vapor) and the rate at which the skin is heated by circulation of blood from the warmer body core. The skin temperature of women is generally higher than that of men under the same stress. This is beneficial in reducing the heat transfer from an environment hotter than skin, but narrows the core-to-skin gradient for heat transfer, requiring that a greater volume of blood be brought to the skin in order to dissipate the same amount of heat. It must be recalled that women are at a disadvantage in this respect, due to their smaller heart stroke volumes, smaller total blood volumes and relatively greater peripheral blood pooling when vasodilated. Therefore, it is not surprising that

women have heat conductances less than those of their male counterparts. Lack of adequate sweating is not the sole reason for the higher skin temperatures; in extremely hot ( $70^{\circ}\text{C}$ ) temperatures, when sweat production was great enough to completely wet and drip off both men and women, women still showed skin temperatures averaging  $39.6^{\circ}\text{C}$ ,  $1.0^{\circ}\text{C}$  higher than men exposed simultaneously (Schoenfeld, Udassin, Shapiro, Ohri and Sohar, 1978). Onset of sweating at higher body temperatures, lower sensitivity of the sweating mechanism and a lower maximal sweat rate may all be adaptations in women which serve to conserve body water during heat exposure. Because thermal balance is achieved with less sweat loss, women have been considered by some investigators to be more "efficient" thermoregulators than men (Wyndham, Morrison and Williams, 1965; Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967). Such increased efficiency may well be required by women in order to minimize reductions in circulating blood volume which would impair both tissue nutrition and heat transfer within the body.

F. Vasodilatation and body fluid shifts:

The cutaneous vascular bed dilates during work in the heat; this expansion of the peripheral shell of the body is proportional to skin surface area. However, the total blood volume is proportional to lean body mass, not surface area. Thus, women have a greater fraction of their total blood volume pooled in the periphery during work in the heat than do men because of their greater surface area to lean body mass ratios (Senay, 1973).

Untrained men working at moderate levels in the heat show a loss of fluid from the vascular compartment with a concomitant loss of circulating protein, presumably into the interstitial spaces of working muscle (Senay, 1972; Senay and Kok, 1977). Work-training contributes to the stabilization of the plasma

volume and a net influx of protein into the vascular volume, possibly as a result of increased rates of movement of water, salts and protein through muscle interstitium; acclimation to heat results in an addition of fluid to the vascular volume (hemodilution) with circulating protein levels either maintained or increased, suggesting a further improvement in the movement of protein, water and salts (Senay and Kok, 1977).

Untrained women working in the heat show a loss of plasma volume equal to or greater than men (Wells and Horvath, 1974; Senay and Fortney, 1975; Drinkwater, Denton, Kupprat, Talag and Horvath, 1976), with the exact changes in circulating protein apparently dependent on how "untrained" they are. Training results in less marked losses in plasma volume (Drinkwater, Denton, Kupprat, Talag and Horvath, 1976; Drinkwater, Kupprat, Denton and Horvath, 1977; Fortney and Senay, 1977), with better maintenance of, or increases in, circulating protein. Acclimation results in an enlarged resting blood volume, less marked hemoconcentration and better retention of circulating protein, though not to a level equal to that achieved by men (Fortney and Senay, 1978) unless trained and acclimatized to the level of marathon runners (Drinkwater, Kupprat, Denton and Horvath, 1977). The basic disadvantage of women appears to be their greater movement of plasma water into the muscle interstitium during exercise and a failure to retain in the vascular volume that protein initially flushed from the cutaneous interstitium when vasodilatation occurs (Senay, 1972; Senay and Fortney, 1975). Better retention of fluid and protein in the vascular compartment can account for many of the cardiovascular improvements seen after acclimatization, but it is clear that the resulting lower skin temperatures may reduce peripheral blood pooling in the cutaneous veins without interfering with arterial inflow. This reduces the amount

of peripheral blood pooling (Drinkwater, Denton, Kupprat, Talag and Horvath, 1976; Drinkwater, Kupprat, Denton and Horvath, 1977).

The initial disadvantages that women show in maintaining plasma volume are further compounded by the effects of dehydration on their relatively smaller body water stores. Dehydration decreases plasma volume and increases plasma osmolarity, which reduces sweating rate. Successful thermoregulation in women thus requires a compromise between the amount of water lost through sweating, an increase in the body temperatures required to induce adequate sweating for evaporative cooling, an increase in the heart rate to maintain cardiac output adequate for both thermoregulation and tissue nutrition and a reduction in the work intensity or its endurance time (Wyndham, Morrison and Williams, 1965; Weinman, Slabochova, Bernauer, Morimoto and Sargent, 1967; Fox, Lofstedt, Woodward, Eriksson and Werkstrom, 1969; Senay, 1973).

G. Effects of the menstrual cycle:

The effects of the menstrual cycle on responses to heat have been studied intensively, initially in resting women (because of the post-ovulatory rise in basal body temperature) and more recently when physical exercise is being performed (Kawahata, 1960; Hertig and Sargent, 1963; Haslag and Hertzman, 1965; Kenshalo, 1966; Sargent and Weinman, 1966; Cunningham and Cabanac, 1971; Drinkwater, 1973; Wells and Horvath, 1973 and 1974; Senay, 1973; Bittel and Henane, 1975 and Bonjour, Welti and Jequier, 1976). Menstrual effects have been found generally to be minimal. Basal body core temperature is increased 0.1 - 0.5°C after ovulation. This may result from diminished estrogen or increased progesterone modifying the body "set point" temperature, or enhancing post-ovulatory basal metabolic rate (Haslag and Hertzman, 1965; Bittel and Henane, 1975), mediated by increased levels of circulating norepinephrine stimulating carbohydrate or fat metabolism



(Zuspan and Rao, 1974). The increased post-ovulatory basal body temperature may or may not be reflected during rest in the heat (Haslag and Hertzman, 1965; Senay, 1973; Wells and Horvath, 1973; Bittel and Henane, 1975), but does not appear to affect body surface temperatures at rest, nor any body temperatures achieved during work (Ibid.; Wells and Horvath, 1974). Steady state sweating rates at rest or during work are probably not different after ovulation than before (Senay, 1973; Wells and Horvath, 1973, 1974; Bittel and Henane, 1975; Bonjour, Welte and Jequier, 1976), although resting sweating rates may be reduced during the menses (Kawahata, 1960). It is clear that the onset of sweating in response to heat exposure during the post-ovulatory portion of the cycle is delayed until mean body temperatures (and therefore heat content) are 0.3 - 0.6°C higher (Senay, 1973; Wells and Horvath, 1973; Bittel and Henane, 1975; Bonjour, Welte and Jequier, 1976). These authors agree that the change in mean body temperature required to initiate sweating is about the same during the post-ovulatory period as in the pre-ovulatory period; apparently only the baseline is shifted. However, there is also a post-ovulatory reduction in the rate of rise of sweating once it has begun (Wells and Horvath, 1973; Bittel and Henane, 1975), which can delay the arrival of the steady-state until higher body temperatures are reached. Because of the variability between subjects, body temperatures at the end of similar exposures may show no differences before and after ovulation, however (Senay, 1973). Therefore the differences in onset of sweating after ovulation, although informative of the nature of the thermoregulatory control processes, appear to be of minimal practical importance.

Body heat conductances and skin blood flow during resting heat exposure in the pre-ovulatory period are less than those of men, but become very similar after ovulation (Haslag and Hertzman, 1965; Kenshalo, 1966; Bittel and Henane,

1975). These differences have not been shown to be of much practical importance either, but may be the cause of the post-ovulatory increase of 1-2°C in the threshold for thermal comfort (Kenshalo, 1966; Cunningham and Cabanac, 1971).

Although the menstrual cycle has little apparent effect on responses to heat stress, it does impact on physical performance. Drinkwater (1973) reports that 17% of women athletes feel their performance is adversely affected during the menses. Further, heat stress and physical activity have been reported to disturb both the timing and the subjective effects of menstrual flow (Hertig and Sargent, 1963; Drinkwater, 1973).

#### V. Physiological Differences under Cold Stress

Unfortunately, relatively little has been reported about the differences between women and men in their responses to cold stress, especially when working. Based on a most comprehensive review of available studies, Buskirk (1966) adopts the position that cold exposure in air has similar effects in men and women on their resting metabolic heat production. The effects appear to vary with the degree of stress; women increase their metabolic heat production by shivering at 0.3 - 1.2°C cooler temperatures than men (DuBois, Ebaugh and Hardy, 1952; Hong, 1963). During non-shivering exposures, young women increase their resting metabolic rates up to 15% while men and older or obese women do not (DuBois, Ebaugh and Hardy, 1952; Bernstein, Johnston, Ryan, Inouye and Hick, 1956; Buskirk, 1963). During exposures at 5°C, women's shivering heat production was the same as men, with similar core temperatures but 2°C less  $\bar{T}_{sk}$  than men (Wyndham, Morrison, Williams, Bredel, Peter, Von Rahden, Holdsworth, Van Graan, Van Rensburg and Monroe, 1964). There are definite indications of racial differences in these responses, though: these same authors present the caveat that

European men have higher core temperatures than African men and Tanaka (1972) has reported that during 10°C exposure, while clothed, the resting metabolic rates and oral temperatures of Japanese men exceed those of Japanese women.

During laboratory studies of cold exposure down to 5°C, the core temperatures of women are generally the same as men, but average skin temperatures are from 1-2°C lower (Hardy and DuBois, 1940; DuBois, Ebaugh and Hardy, 1952; Buskirk, Thompson and Whedon, 1963; Wyndham, Morrison, Williams, Bredel, Peter, Von Rahden, Holdsworth, Van Graan, Van Rensburg and Munro, 1964). However, Hanna (1970) has shown that average skin temperatures may not be different for Quechua men and women in Peru during laboratory cold exposure, and that clothed average skin temperatures during cold exposure outside the laboratory are very nearly equal, suggesting possible genetic differences. He has verified the 1-3°C colder lower extremity temperatures in women that are common subjective experience. The generally reported lower mean skin temperatures of cold-exposed women apparently do not result in greater levels of adrenocortical stress, as their 17-ketosteroids do not increase during 8 hours post-exposure, but those of men did (Tanaka, 1972).

The cooler skin temperatures of women result in greater body heat debt (Buskirk, 1963). However, it is a superficial phenomenon if their core temperatures remain the same as men. Clearly, the calculated body heat conductance of women is lower than that of men, with a greater penetration of the environmental gradient towards the body core with time (Hardy, Milhorat and DuBois, 1941). These authors calculate the difference between men and women in insulation to approximate a layer of 4 mm more of completely vasoconstricted body fat. Wyndham's group (1964) reports an average difference of 3.4 mm in the average measured thickness of body fat, and Hong also reports (1963) that non-cold exposed women have a

tissue insulation significantly greater than men, but by an unspecified amount. This is not due merely to more body fat, but to greater vasoconstriction also: in the cold, obese women have average skin temperatures  $0.8 - 3.1^{\circ}\text{C}$  higher than non-obese women with equivalent core temperatures (Bernstein, Johnston, Ryan, Inouye and Hick, 1956). Their greater tolerance to cold, despite a greater surface heat loss, appears to result from the greater thermal inertia of their body mass. Burkirk (1963) makes the same point: Body fat buffers the effects of cold very effectively, but only after vasoconstriction. Hardy and DuBois (1940) calculate the heat loss per unit surface area in women to be 10% less than that of men at  $22-27^{\circ}\text{C}$ , although their respiratory and insensible sweat losses are the same; Wyndham's group (1964) estimates the difference in conductance at  $5^{\circ}\text{C}$  to be 15%. More body fat is correlated with less heat loss (Sloan and Keatinge, 1973). In  $20^{\circ}\text{C}$  water the cooling rates of young male and female swimmers were inversely correlated to the amount of body fat on the trunk and limbs. The association was even greater when differences in body surface area-to-mass ratios were removed. Males have less limb fat for the same trunk fat thickness or surface-area-to-lean body mass ratio than women (Edwards, 1951; Sloan and Keatinge, 1973); young men cooled faster when swimming in water than young women of the same percent lean body mass and their core temperatures finished at lower levels (Sloan and Keatinge, 1973). However, women may not have a similar advantage when remaining still in cold water; Hayward, Eckerson and Collis (1975) report no differences in the body cooling rates of quiescent men and women in  $18.2$ ,  $10.5$  and  $4.6^{\circ}\text{C}$  water. Metabolic rates were increased 2.5 fold when swimming was begun at  $10.5^{\circ}\text{C}$ ; despite this, the average net cooling rate increased 1.35 times. Presumably there were no differences between men and women in this regard, although this was not mentioned specifically.

The reduced conductance of women may well result from a diminution of arterial inflow to the limbs as well as from generalized vasoconstriction of the cutaneous circulation. Arterial inflow to the fingers of women is only one-half that of men, and women reduce their arterial finger inflow much more than men in response to indirect (foot) cooling as well as direct (finger) cooling (Bollinger and Schlumpf, 1976). Clearly, lower surface temperatures of the limbs makes women much more susceptible to cold injury than men, although their resistance to core cooling seems to be as good as that of men, or better.

#### VI. Comfort

Although not properly within the purview of this survey, the topic of comfort is worth a brief mention. Despite their differences in morphology and response to heat and cold stress, men and women appear to react subjectively in similar fashion. Despite measured differences in foot temperature of  $2.1^{\circ}\text{C}$ , the preferred air temperatures for men and women were  $25.6$  and  $25.4^{\circ}\text{C}$ , respectively (Olesen and Fanger, 1973). These resulted in average body skin temperatures of  $33.6$  and  $33.4^{\circ}\text{C}$ , despite the insensible water losses of women being only 83% those of men. Age has long been thought of as increasing the temperature needed for comfort, but older women preferred the same levels and combinations of air and radiant temperatures as younger men (Griffiths and McIntyre, 1973).

The range of "comfortable" temperatures tolerated by women has been reported to be wider than that of men,  $6^{\circ}\text{C}$  compared to  $2-3^{\circ}\text{C}$  (Hardy and DuBois, 1941). This was attributed to their better adaptation of skin temperature, which reduces heat flow between the skin and the environment. When wearing standard clothing, however, women and men show similar ranges of comfortable temperatures, more nearly so after the first hour of exposure (Robles, 1971). Obesity

apparently does not affect the range of comfortable temperatures (Bernstein, Johnston, Ryan, Inouye and Hick, 1956) or the subjective perception of uncomfortably cool ones (Buskirk, 1963). Most likely, the reports of women preferring much warmer temperatures arose in the days when typical office wear for women weighed less than half as much as that for men and provided less insulation. The current trend of similar levels of clothing insulation for women and men seems to have reduced much of the reported disparity, although over the short term (1 hour or less) women still prefer slightly warmer temperatures (Robles, 1971).

#### VII. Injury Prevention

The prevention of heat and cold injury in stressful environments obviously is a greater problem for women than it is for men. Adequate environmental protection, properly engineered jobs, and sufficient recovery periods in less stressful environments are even more necessary for women than they are for men, particularly during the period of adaptation to the stress.

The principal difficulties for women working in heat stress are their higher heart rates and lower maximal sweat rates. Tasks must be engineered to minimize lifting the proportionately fatter body weights, with due recognition given their lesser muscular strength, particularly in the upper body (Churchill, Churchill, McConville and White, 1976). Rest periods in the cool must be provided to prevent heart rates increasing progressively throughout the work day. Any factor which will retard evaporative cooling of their limited sweat production will place women at an even greater disadvantage. For this reason, air movement must be maximized and dehydration prevented. Relatively impermeable protective clothing (rainwear, reflective suits or chemical protective clothing) can be expected to have a greater impact on the tolerance time of women than men. Since core temperatures cannot

readily be determined on the job, heart rates and subjective reports must be substituted as indicators of excessive stress levels. Women must be introduced to severe heat stress for brief periods at first (1 hour or less), with progressive lengthening in exposure time as tolerance increases. Otherwise, heat injury may well supervene and prevent acclimatization. In heat exposure, as in exercise, individuals must be able to withstand the conditioning procedures before they can benefit from them.

Cold injury, not hypothermia, is the greater risk for women working in the cold. The geometry of women's thinner extremities results in a greater heat outflow for the same circulatory heat input per unit tissue mass. Their enhanced peripheral vasoconstriction further inhibits their ability to maintain safe skin temperatures. Therefore a greater amount of clothing insulation is required on the extremities to both retard the onset of vasoconstriction and to reduce the rate of cooling once vasoconstriction has occurred. Very little heat can enter the extremities after vasoconstriction; extremities will continue to cool as Newtonian masses in severe environments. Despite added clothing insulation, cold injury will occur unless the extremities are rewarmed in time, by either increasing the metabolic rate or providing external heat. For tasks in severe cold that are not physically demanding, external heat is mandatory and it is best provided early enough to retard vasoconstriction; about one fourth less heat is required to inhibit vasoconstriction than to overcome it during rewarming.

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