PHYSIOLOGICAL RESPONSE TO HEAT EXPOSURE

HEAT BALANCE

Fundamentals of heat transfer

Humans are homeothermic, which means they must maintain body temperature within a narrow range in varying environmental conditions. The normal deep body temperature (core body temperature) at rest is between 36-37.5 ^{o}C, although extremes in excess of 40 ^{o}C have been recorded in athletes and workers exposed to very severe environmental conditions. These temperatures are at the upper limit of human physiological tolerance, however they illustrate that people do get exposed to such conditions during their work practice. The variation of resting core body temperatures also demonstrates the individual diversity that may exist in a working population. This variation means that people may have different tolerances to working in the heat. Some people cannot tolerate mild increases in core body temperature whereas others, as illustrated, can continue to work at much higher temperatures. The factors that may account for this variation among workers are still, however, poorly understood.

Thermal homeostasis is maintained by achieving a balance between the various avenues of heat gain and heat loss from the body. There are two recognised sources of heat load;

a) Environmental, which may be positive or negative, that is, there may be a heat gain or a heat loss from the body.
b) Metabolic, which is generated by muscular activity.

ENVIRONMENTAL FACTORS AFFECTING THERMOREGULATION

The principal methods of heat exchange between the body and the external environment are: convection, conduction, radiation and evaporation.

Convection

The rate of convective exchange between the skin of a person and the ambient air in close proximity to the skin, is dictated by the difference in temperature between the air and the skin temperature together with the rate of air movement over the skin. When the air temperature is greater than the skin temperature, there will be a gain in body heat from the surrounding air, conversely when the skin is warmer than the air temperature there will be a loss of heat from the body. Because warm air rises (less dense than cool air) the warm air will rise from the body and cool air will come in to take its place. This process is then repeated. The process is called convection.

Radiation

The surface of the human body constantly emits heat in the form of electromagnetic waves. Simultaneously, all other dense objects are radiating heat. The rate of emission is determined by the absolute temperature of the radiating surface. Thus if the surface of the body is warmer than the average of the various surfaces in the environment, net heat is lost, the rate being directly dependent on the temperature difference. This form of heat transfer does not require molecular contact with the

warmer object. The sun is a powerful radiator, and exposure to it greatly decreases heat loss by radiation. When the temperature of the objects in the environment exceeds skin temperature, radiant heat energy is absorbed from the environment. Under these conditions the only avenue for heat loss is by evaporative cooling.

Conduction

The difference between heat loss by conduction and radiation is that with conduction the body must be in contact with the object. In such circumstances the heat moves down its thermal gradient from the warmer to the cooler object, the heat energy being transferred from molecule to molecule. The warmer molecule slows down after it has lost some of its heat and the cooler molecules move faster having gained heat. The temperature transfer continues until eventually the temperature of the two objects equalises. The rate of the heat transfer through conduction depends on the difference in temperature between the two objects and the thermal conductivity of the two objects.

Evaporation

When water evaporates from the surface of the skin, the heat required to transform it from a liquid to a gas is dissipated from the skin, this acts to cool the body. Evaporative heat loss occurs from the respiratory tract lining as well as from the skin. There is a constant gradual loss of water from the skin that is not related to sweat glands. The skin is not fully waterproof and so some water is lost out through pores in skin, and lost by evaporation. This loss is not subject to physiological control and is termed insensible perspiration. Sweating is an active process requiring energy and controlled by the sympathetic nervous system. The rate at which this process proceeds can be controlled and therefore the amount of heat loss can be controlled. Radiation and convection are insufficient to prevent warming up of the body during heavy manual work or at high surrounding temperatures. Under these circumstances heat loss is aided by evaporation of water. At environmental temperatures above about 36 ^{o}C, heat is lost exclusively by evaporation. At higher temperatures heat is taken up by the body from the environment by radiation, conduction and convection. Sweating then becomes profuse in order to maintain the balance between heat uptake and heat loss by evaporation. In order to be effective, sweat must be evaporated from the skin. If sweat merely drips from the surface of the skin or is wiped away, no heat will be lost.

Thermal equilibrium of the body is maintained by balancing the relationship

$M \pm C \pm K \pm R - E \pm S = O$

Where M = metabolic heat (always positive).

C = convective heat exchange.

K = conductance heat exchange through surfaces in direct contact with the skin.

R = radiative heat exchange between skin or clothing surroundings.

E = evaporation of water from the skin surface and respiratory tract .

S = heat storage (heat balance exists when S is zero).

METABOLIC FACTORS AFFECTING THERMOREGULATION

In addition to heat exchange between the body and the environment, internal heat is produced by metabolic processes. Although digestion and other body processes

contribute slightly, by far the greatest influence is the heat generated during external work. Body heat is gained directly from the reactions of energy metabolism. When muscles become active, their heat contribution can be tremendous. For example, at rest, the rate of body heat production is relatively low; the resting oxygen consumption is approximately 250 mL/min corresponding to a rate of heat production of 70W. During work, the rate of oxygen consumption can increase eightfold, and the rate of heat production is correspondingly increased. There are four work components which can affect metabolic heat load: work rate, work nature, work pattern and posture.

Work rate

Muscular work is mechanically a very inefficient process. The major muscles such as the upper or lower limb muscles can only achieve 20-25% mechanical work efficiency. A substantial proportion of the rest of the energy is generated as heat. There is therefore a direct relationship between work rate and metabolic heat production.

Work nature

Much of work is a mixture of dynamic and static components. As the proportion of static work (meaning no movement) increases, the muscles become even less efficient with the result that more energy is produced as heat. The nature of the work can therefore influence the metabolic heat load.

Work pattern

The role that scheduling plays in modern work can also influence the heat load. Set timing of breaks at work may mean that the worker cannot stop and cool down. This may effect thermoregulatory efficiency and therefore work tolerance. On the other hand, self paced work may allow the worker to operate more safely in conditions of thermal stress.

Posture

The effect of poor posture when working may place an additional burden or loading on muscles. This will result in additional heat generation by the muscles as a consequence of mechanical disadvantage.

THE INFLUENCE OF CLOTHING ON THERMAL LOAD

Heat exchange is relatively easy to analyse in the basic model, that is the exchange between human skin and the micro-climate created by a layer of ambient air. However, when layers or even a layer of clothing is used heat exchange becomes far more complex. The space between the skin and the outermost garment becomes a very complicated micro-climate consisting of air and fabric layers changing depth with each body movement. The insulative characteristics of this environment are given by the behaviour of the air trapped between the skin and clothing. This means that any factor that alters the thickness of the air layers and so the insulative properties will lead to a decrease or increase in heat loss from the body. The difficulty of understanding the effect of heat balance is enhanced when the behaviour of the air and air exchange between the clothing layers and the environment is considered. The size, shape, and number of pores in the garment influence air exchange and movement

and therefore the insulative properties of the clothing. These factors in turn influence the ability of the person wearing the clothing to lose heat generated by muscular activity. A change of clothing insulation is the easiest and the quickest method of thermal adaptation, although there are practical or cultural limits to this form of physical thermoregulation. There are cases, however, where humans may adjust their metabolism rather than clothing insulation, as has been shown by in a North American elderly population. If the heat generated by these factors is not lost, then a rise in core body temperature and heat illness will result.

The subject of thermal comfort is complex and beyond the confines of this work, however, it is relevant to persons working in hot environments. The importance of protecting the skin from harmful ultra-violet radiation and against non thermal hazards in the workplace is becoming increasingly important. Because workers should wear clothing to protect themselves from these hazards, the thermal properties required of clothing to maximise cooling is an important consideration. There have been a number of reviews on this subject.

HUMAN THERMOREGULATION

It was previously stated that as ambient temperature increases, the effectiveness of heat loss by radiation, conduction and convection decreases. When ambient temperature exceeds body temperature, heat is actually gained by these mechanisms of thermal transfer. In such environments, or when conduction, convection and radiation are inadequate to dissipate substantial metabolic heat loads, the only means for heat dissipation is by sweat evaporation. The rate of sweating increases directly with the ambient temperature.

The total amount of sweat evaporated from the skin depends on three factors:

1) The surface exposed to the environment.
2) The temperature and humidity of the ambient air.
3) The convective air currents around the body.

Relative humidity is by far the most important factor determining the effectiveness of evaporative heat loss. When humidity is high the ambient vapour pressure approaches that of moist skin and evaporation is greatly reduced. Thus this avenue for heat loss is essentially closed, even though large quantities of sweat are produced. This form of sweating represents a useless water loss that can lead to dehydration and overheating. As long as the humidity is low, relatively high environmental temperatures can be tolerated. For this reason, hot, dry desert climates are more comfortable than cooler but more humid tropical climates.

Most studies on human thermoregulation have been performed in climate chambers as opposed to the outdoor natural environment where most physical activity takes place. The thermal environment under outdoor conditions may include significant radiant heat gain, especially on sunny days. For humans, the colour of skin and clothing is of importance for the reflection and absorption of solar radiation, with darker colours having greater absorptive heat gain. It has also been reported by that the radiant heat gain from sun, direct and indirect, is between 160-230W per hour. It has also being shown that the heat load gained from direct sunshine is significant, for this reason predictions of heart rate and sweating rate based on climate chamber experiments will

give too low values for outdoor exercise in the sun. Although solar radiation is only moderate in heat balance, the addition of this extra heat stress, even in temperate climates, may be critical for near maximal exercise performance, or for unacclimatised, physically untrained workers where thermoregulation and cardiovascular stability become important for physical performance and endurance.

Cardiovascular and neural response to heat exposure

The mechanisms for heat loss are the same whether the heat load is produced internally (metabolic heat) or externally (environmental heat). The circulatory system serves as the 'workhorse' in maintaining thermal homeostasis. In the heat and even while at rest, the heart rate and cardiac output increase while superficial venous and arterial blood vessels dilate to divert warm blood to the body surface. With extreme heat stress, 15 - 25% of the cardiac output passes through the skin. This greatly increases the thermal conductance of peripheral tissues and can favour radiative heat loss to the environment. Thus an effective heat defence can be established when the evaporation of sweat is combined with a large cutaneous blood flow.
Simply stated, when working in the heat, the body is faced with two equally important and competing demands;

1) The muscles require oxygen to sustain energy demands.
2) Metabolic heat must be transported by the blood from the deep tissues to the periphery.

As a consequence of the competitive demands for blood, a decreased delivery of oxygen to working muscles may result. In addition, adequate cutaneous and muscle blood flow are attempted at the expense of tissues that can temporarily compromise their blood supply such as the gut. It is also reported that aside from redirecting blood to areas in greater need, vasoconstriction in the viscera serves to increase total peripheral resistance. In this way, arterial blood pressure would be maintained during exercise in the heat. In addition to vascular adjustments, the dissipation of metabolic heat during exercise in a hot environment is almost totally dependent upon the cooling effect of sweat evaporation. This however places extra demands on the body's fluid reserves and a relative state of dehydration may occur.

As the core body temperature rises, either from exercise or the environment, it soon reaches a level sufficient to stimulate thermosensitive neurons in the central nervous system. Sensory feedback from these sites, primarily to the preoptic anterior hypothalamus, evokes autonomic neural outflow to appropriate heat loss effectors. Thus skin blood flow and sweating both exhibit a characteristic internal temperature 'threshold', beyond which they rise with increasing core body temperature. Heat loss early in an exercise session is much less than the heat produced by muscular contraction, due to initial vasoconstriction of blood vessels in the skin, which retards heat dissipation. Consequently, most of the heat produced within the first several minutes of exercise is stored and core body temperature rises. When heat storage becomes sufficient to raise core body temperature above the threshold(s) for vasodilation and sweating, heat transfer to the surface and subsequent elimination to the environment, rapidly increases to match metabolic heat production. Upright and dynamic exercise cause the greatest increase in skin blood flow and therefore a rise in core body temperature, due to the greater need for vasoconstriction to maintain central pressure for cardiac filling. report that reflexes associated with exercise cause an

elevated vasodilator threshold, by delaying activation of the vasodilation system during exercise, rather than through the adrenergic vasoconstrictor system. The study also shows a divergence in active vasodilation and sudomotor activities during exercise, and suggests separate neural controls for these systems.

If core body temperature, mean skin temperature and local skin temperature were the only inputs for local sweating control, sweat rate would be equal between an exercising and non-exercising limb. It is postulated that muscle temperature may be an additional factor in local sweat rate control, after it was demonstrated an increased sweat rate in the exercising over the non exercising leg. Thermoreceptors, either within the muscle or the efferent veins, may possibly influence local sweat rate through a different central processing area, independent of the processing of afferent input from skin receptors. Local muscle thermoreceptors are probably overridden by central core body temperature effectors.

Hypervolemia

Convertino in a review article, discussed the physiological adaptation of increased blood volume (hypervolemia) with endurance training. This net increase in body water with physical exercise is associated with increased water intake and decreased urine output. The mechanism of reduced urine volume output appears to be increased renal tubular absorption of sodium through a more sensitive aldosterone action. The well-documented exercise stimulus for hypervolemia consists of both thermal and non-thermal components that serve to increase total circulating levels of electrolytes and proteins. The hypothesis that a thermal challenge is the primary stimulus for exercise induced hypervolemia is reasonable, since characteristics of heat acclimatisation can be partially induced by exercise training. A study by Convertino used eight subjects, four who cycled at 65% VO_2 max for 2 hours per day for 8 consecutive days, producing an average rectal temperature of 38.2 ^{o}C, and four who sat in a heat chamber (42 ^{o}C; 93% RH) for 2 hours per day for 8 consecutive days, averaging the same rectal temperature. The exercise group had a 12% increase in plasma volume compared to 5% for the resting group. Another study combined exercise and heat exposure using the same frequency and duration as Convertino. These subjects had a 17% increase in plasma volume at the end of the study, equal to the sum of that reported by exercise alone (12%) and heat alone (5%) by Convertino. This is further supported in a review article by Fellmann (1992). Thus, there appear to be both thermal and exercise factors that stimulate hypervolemia, and these factors have an additive effect on the volume of plasma expansion. This hypervolemia may provide advantages of greater body fluid for heat dissipation and thermoregulatory stability, as well as larger vascular volume and filling pressure for greater cardiac stroke volume and lower heart rate during exercise.

Heat loss from the head

A study by Rasch et al to verify whether the head can function as a 'heat sink' for selective cooling, demonstrated that the heat loss from the head was larger than the heat brought to the brain by arterial blood during hyperthermia (estimated to be 45W for each 1 ^{o}C above normal temperature; heat produced by brain estimated at 20W). It is argued that although many species of animals exhibit selective brain cooling through the virtue of the presence of rete mirabile on the internal carotid artery,

human species lack a rete mirabile and thus do not have the capacity for selective brain cooling. Rasch et al. however, demonstrated tympanic temperature being lower than oesophageal temperature during hyperthermia only, and postulated this to be indicative of selective brain cooling, provided that tympanic temperature is a good index of intracranial temperature. Another study reported tympanic temperature to be indicative of core body temperature and intracranial temperature. It was concluded that the human head, from a strictly physical point of view, can function as a heat sink with a heat loss capacity greater than the heat produced by the brain and received from arterial blood during hyperthermia. The practical application of these findings lie mainly in the treatment of hyperthermic patients, where the intense heat loss capacity of the head should be considered when attempting to return a patient rapidly to normothermia. During exercise or work in the heat, allowing the head free evaporative capacity may protect the brain, the most heat susceptible nervous tissue, from damage.

ENDOCRINE CONTROL OF THERMOREGULATION

The role of the plasma hormones renin, aldosterone and cortisol in the reduced sweat rate and skin blood flow, and increased physiological strain reported with varying levels of dehydration was investigated by Francesconi et al.. The study was conducted on six heat acclimatised male subjects (mean age 23.2 years; mean VO_2 max 53.8 mL/kg.min), who were dehydrated to 3%, 5% and 7% of body weight by exercising in the heat. It was reported that plasma volume decrements and increments in stress and fluid regulating hormones were well correlated. Graded levels of hypohydration up to 5% body weight loss, elicited incremental levels of plasma renin activity and aldosterone. However, between 5% and 7% dehydration no further elevations were reported, while plasma cortisol was consistently elevated with increasing severity of hypohydration through 7% of body weight. Heat acclimatisation reportedly moderated and modified the hormonal responses to exercise in the heat, and resulted in the altered response of fluid regulation by hormones measured in the study.

Researchers have measured the effects of heat acclimatisation on the following circulating plasma hormones: antidiuretic hormone (ADH), aldosterone, cortisol, atrial natriuretic peptide and B-endorphin (Armstrong & Maresh, 1991; Sato et al.,1990). The results of these studies have shown ADH functions to reduce urinary water loss and to conserve body fluids. Although ADH synthesis and secretion is strongly stimulated by an increase in osmolality, several other factors may mediate its release during exercise in the heat, such as alterations in plasma volume, blood pressure and renal and hepatic blood flow. It was also shown that the increase in plasma volume associated with heat acclimatisation decreases the ADH response. Greenleaf (1979) reports that the levels of ADH increase with hypovolemia during exercise.

The renin-angiotensin-aldosterone system reportedly represents an important biochemical pathway that controls Na^+ and Cl^- concentrations in urine and sweat (Armstrong & Maresh, 1991). Conservation of Na^+ and Cl^- in these fluids allows the body to optimally maintain blood and extracellular fluid volumes. The levels of these hormones in the blood decrease following heat acclimatisation (Armstrong & Maresh, 1991). In support of this the increased cardiovascular stability and plasma volume associated with heat acclimatisation may collectively serve to lessen the stimulation and the physiological need for large elevations of fluid and electrolyte regulating hormones (Francesconi et al.,1985).

Brandenberger et al. (1989), however, report that continuous heat exposure modifies renin-aldosterone nocturnal patterns by increasing their mean levels and oscillation amplitude, indicating increased secretion which, via aldosterone actions on the kidneys, counteracts the water and salt losses due to sweating. Hargreaves et al. (1989) report that plasma renin activity and aldosterone levels were higher after a low sodium intake (50 mmol/day) and increased during exercise-heat exposure. It was suggested that the increased hormonal responses to a low sodium intake were important in maintaining plasma sodium levels and exercise performance.

Barr et al. (1991) reported elevated aldosterone levels during exercise, which decreased urinary sodium loss but had no effect on sweat sodium concentrations. Greenleaf (1979) reported increased renin-angiotensin levels in response to hyponatremia during exercise. Meyer et al. (1992) showed that plasma aldosterone levels were similar at rest and during exercise, in pre-pubescent individuals and young adults, although the sweat sodium concentrations were higher among the young adults, and concluded that sweat sodium concentrations may not be related to plasma aldosterone levels. The receptors of the adult sweat gland may be more sensitive to aldosterone than those of younger individuals but this has not been investigated. Convertino (1991) found no change in 24 hour resting plasma aldosterone levels and speculates that exercise-heat induced hypervolemia is maintained by increased renal tubular absorption of sodium, through increased sensitivity of receptors to aldosterone. Exercise training also increases thirst, which aids in the oral replacement of fluids, post-exercise.

Plasma cortisol levels are used as a general indicator of the degree of strain experienced by the body. Well hydrated, heat acclimatised individuals show no significant increase in cortisol when exercise in a hot environment is mild. Under the same conditions, however, in unacclimatised and dehydrated individuals, this can result in large cortisol increases, indicating an increase in physiological strain (Armstrong & Maresh, 1991).

THE EFFECT OF AGE AND BODY COMPOSITION ON THERMOREGULATION

From the literature to this point it is obvious that numerous factors determine or influence the physiological strain imposed by heat. These factors may include individual variations in body size and obesity, sex, age and fitness, as well as the external factors such as convective air currents, radiant heat gain, intensity of exercise, amount, type and colour of clothing, and the relative humidity.

There are few reports on the amount of fluid lost by people working in the heat as opposed to exercising. Most of the research on fluid loss has concentrated on athletes, rather than workers exposed to often extreme heat stress for the duration of a shift (Fortney et al.,1981; Sawka et al.,1985; Candas et al.,1986; Nose et al.,1988; Davis et al.,1988; Hargreaves et al.,1989; Gisolfi & Duchman, 1992; Montain & Coyle, 1992). As a consequence of the imbalance in the reported literature the cited findings in this review are sport rather than occupationally directed. The subjects used in most of the reviewed literature tend to have high fitness levels, low body fat and are generally young. The findings of these studies therefore may have limited application to the workplace setting.

The effect of body morphology

Obesity is a liability when working in the heat because the specific heat of fat is much greater than that of muscle tissue, excess fat increases the insulatory properties of the body surface and retards conduction of heat to the periphery (Nunnely, 1978). However, the extent that percentage body fat may impede thermal regulation is not fully known. In addition to possibly interfering with heat exchange, excess fat directly adds to the metabolic cost of activities in which the body weight must be moved, resulting in increased metabolic heat production (Kolka, 1992).

The effect of gender

Sweat rate differences between males and females have been reported (Meyer et al., 1992; Kolka, 1992). Men repeatedly have been shown to have higher sweat rates than women in hot-dry and hot-wet climates, with their sweat rates often increasing more than those of women during heat acclimatisation. Women however, seem to be at least as efficient in temperature regulation as men because they maintain the same core body temperature (Armstrong & Maresh, 1991). Meyer et al. (1992) however, report that men and women have similar sweating rates when matched for VO_2 max (45 mL/kg.min). These authors also report men having higher sweat sodium concentrations than women (60 mmol/L compared to 40 mmol/L) when exercising for 40 mins at 50% VO_2 max in 42 ^{o}C and 18% RH. These losses were attained by the collection of sweat via a plastic bag attached to the lower back. The same author also reported that the concentration of sweat sodium in healthy subjects increases with age. The sweating response in this study was however pharmacologically rather than thermally induced. The method used to induce sweat may be important since the response by the sweat gland cannot be assumed to be the same for both modalities.

The effect of age

Maughan (1991) reports that the rate of sweating depends on many factors and is increased in proportion to the work rate and the environmental temperature and humidity. Sweat rates are highly variable among individuals, and can often reach 2 L/hr for marathon runners exercising for prolonged periods. Middle aged men 45 years or older were shown to have higher heart rates, higher rectal temperatures and lower sweat rates than their younger counterparts during exercise in the heat, both pre- and post acclimatisation. Other reports of thermoregulatory decrements occurring with age have also been reported (Araki et al.,1979; Davies, 1979; Cena & Spotila, 1986; Kirk & Westwood, 1989)

However, one valid criticism by Armstrong and Maresh (1991), regarding studies on the effect age has on thermoregulation, is that they did not match the older and younger individuals for pertinent physical and/or morphological characteristics, or answered whether age per se or a decrease in training volume or intensity, or a lower VO_2 max attributed to the reduced heat tolerance.

Other studies have shown that a higher VO_2 max does not offer older men physiological advantages when exposed to conditions of heat stress, but rather the duration of time spent on their regular weekly physical training. Pandolf et al. (1988) acclimatised younger (mean 21yrs) and older (mean 46yrs) men by exercising them on a treadmill (5% grade) twice/day for 50 mins, for 10 consecutive days in a hot/dry (49 ^{o}C; 20% RH) environment. The subjects were matched for body weight, surface area, body fatness and VO_2 max (mL/kg.min). There was no significant difference in thermoregulatory ability between the younger and older men following heat acclimatisation. The older men had a thermoregulatory advantage during the initial days of heat exposure which was reportedly related to their significantly greater regular weekly aerobic activity compared to the young men (24.1±19.7 miles/week compared to 4.8±0.8 miles/week).

A study by Anderson and Kenney (1987) matched older and younger women (mean age 56 and 25 years respectively), exercising at 40% VO_2 max (mean VO_2 max 35.2 mL/kg.min) for two hours in a climate chamber (48 ^{o}C; 25% RH), and reported that the older women responded with a higher rectal temperature and lower sweating rate than the younger women. The lower sweating rates were not a function of fewer heat-activated sweat glands recruited, but a diminished sweat flow per heat-activated sweat gland. A more recent study by Kenney (1988), however using well matched (approximately 40 mL/kg.min VO_2 max), unacclimatised older (age 55-68 years) and younger (age 19-30 years) men and women, performing 75 mins of cycle exercise at 40% VO_2 max in 37 ^{o}C; 60% RH, showed no significant inter-group difference in sweat rate (older 332 mL/m^{-2}.hr, younger 435 mL/m^{-2}.hr). However, an altered control of cutaneous vasodilation during exercise in the heat in the older subjects was found. The lower arm blood flows shown in the older group had little impact on heat storage since skin temperature was very close to the environmental temperature, but appeared to reflect a lower cardiac output response to a given intensity of exercise. The attenuated arm blood flow response appeared to be unrelated to VO_2 max, and was assumed to reflect an age-related change in thermoregulatory cardiovascular function.

In another paper, Kenney and Johnson (1992) report that older individuals typically respond to exercise with less skin vasodilation than that exhibited by their younger counterparts. The limited vasodilatory response can be seen as early as age 50, and is not a function of age-related differences in hydration status or VO_2 max. The lower response of skin blood flow is not due to an increased adrenergic vasoconstrictor tone, but probably relates to structural changes in the vascular bed of aged skin. However, older subjects who maintain a high VO_2 max may partially offset this decrement.

THE EFFECTS OF PLASMA TONICITY ON THERMOREGULATION

Montain and Coyle (1992) report a relationship between elevated serum osmolality or serum sodium and a rise in core body temperature. Hyperosmolality of plasma has been shown to result in reduced thermoregulatory effector response: the threshold for sweating is elevated and the cutaneous vasodilator response is reduced (Sawka et al.,1985; Maughan, 1991; Kenney & Johnson, 1992). Sawka, (1992) reports that both core body temperature and sweating rate responses are more strongly correlated with plasma tonicity than with blood volume. In addition, Sawka reviewed articles which demonstrated the elevation of core body temperature and reduced sweating rate responses when solutions of 2-3% saline were ingested during exercise in the heat, in euhydrated subjects. The combined results of nine studies indicate that plasma hypertonicity exerts a powerful influence on thermoregulatory sweating and body temperature responses during exercise in conditions of heat stress. It is postulated by Sawka (1992), that plasma tonicity changes may relate to tonicity changes in the extracellular fluid bathing hypothalamic neurons. This may result in the alteration in the initiation of sweating (threshold) which appears to be central nervous system controlled. Homeostatically this may be explained by the fact that if there is a shortage of body water, it is conserved to maintain cardiovascular stability instead of cooling.

Tonicity may also exert a peripheral effect: a high osmotic interstitial pressure would tend to inhibit the fluid availability for eccrine sweat gland function. It has also been shown that an iso-osmotic hypovolemia alone can mediate an increased core body temperature and reduced heat loss during exercise exposure (Sawka, 1992). The reduction in heat loss is due primarily to reduced skin blood flow for a given core body temperature in hypovolemic subjects, resulting in a decrease in the potential for evaporative heat exchange. The purpose of this may be explained homeostatically; the activation of low pressure baroreceptors would result in a more central distribution of blood volume to provide adequate perfusion of active skeletal muscles, and the maintenance of central venous pressure. It would appear that when there is insufficient blood volume to meet both metabolic and thermoregulatory demands, the metabolic demands take precedence.

Other studies have also reported reductions in sweat rate with iso-osmotic hypovolemia (Fortney et al.,1981; Sawka et al.,1985). The human strategy for hypohydration appears to be that sweat output is reduced to conserve blood volume, and heat transfer to the skin is reduced to maintain blood flow to active muscles and the brain. The reduced sweating and skin blood flow are regulated (assuming that the subjects will make the decision to employ behavioural thermoregulation), by discontinuing exercise and removing themselves from the heat. This however does not always occur and can result in severe hyperthermia, peripheral circulatory collapse, and life threatening thermal injury (Sawka, 1992).

THE EFFECTS OF HYPOHYDRATION ON THERMOREGULATION

During exercise in the heat, sweat output often exceeds water intake, the result of which is a body water deficit or hypohydration. The water deficit occurs from both the intracellular and extracellular fluid compartments (Nose et al.,1988), and can lead to hyperosmotic-hypovolemia of the blood (sweat is hypotonic to plasma therefore the blood becomes hypertonic). Aerobic exercise tasks are adversely affected by hypohydration, and the warmer the environment the greater the potential for performance decrements (Cena & Bates, 1992). Hypohydration results in greater heat storage and reduces physiological tolerance to heat strain. Water is the largest component of the human body and represents 45-70% of body weight (Candas et al.,1986). The average male (75 kg) contains about 45 litres of water, which corresponds to approximately 60% body weight. An individual's total body water is dependant on their body composition, since adipose tissue is 10% water and muscle tissue 75% water (Havenith et al.,1990). In addition, muscle water will vary with glycogen content, because of the osmotic pressure exerted by the glycogen granules within the muscle sarcoplasm (Sawka, 1992). As a result, athletes tend to have a greater body water than their sedentary counterparts, by virtue of their larger muscle mass and higher skeletal muscle glycogen stores. Sawka (1992) reports that at low levels of hypohydration, the water deficit comes primarily from the extracellular fluid compartment; as the body water loss increases, a proportionately greater percentage of the water deficit comes from the intracellular compartment. The redistribution of water between the intracellular and extracellular fluid compartments is dependent upon the osmotic gradient between these spaces. As cell membranes are freely permeable to water but selectively permeable to solutes, transient alterations in the solute concentration causes water redistribution across cell membranes until the two fluid compartments are in equilibrium, with respect to osmotic pressure. It is well documented that exercise-induced or heat-induced hypohydration increases the osmotic pressure of the plasma, and therefore the plasma becomes hypertonic when the dehydration is induced by sweat output (Nose et al.,1988a; Nose et al.,1988b; Noakes, 1992). Sodium, potassium, and their principle anion, chloride, are the primary electrolytes responsible for the elevated blood tonicity during hypohydration. The plasma hypertonicity that results in mobilisation of fluid from the intracellular to the extracellular fluid compartments, enables the defence of the plasma volume in hypohydrated subjects (Nose et al.,1988a; Pichan et al.,1988; Sawka, 1992).

Generally, body water deficits adversely affect exercise and work performance (Sutton et al.,1972). The critical water deficit and magnitude of performance decrements are dependant on the exercise task and the environmental temperature (Pichan et al.,1988; Rehrer, 1991b; Sawka, 1992). Warmer, more humid environments have greater potential for exercise and work performance decrements, due to the need for higher sweat rates to maintain core body temperature. Short term anaerobic tasks are less likely to be adversely effected by hypohydration, unlike prolonged aerobic tasks. In addition, the thermoregulatory advantages conferred by high aerobic fitness and heat acclimatisation are negated by hypohydration during work in the heat (Francesconi et al.,1985; Sawka, 1992).

Cognitive performance is also adversely effected by hypohydration (Pichan et al.,1988; Sawka, 1992). This has been confirmed in a study by Gopinathan et al.

(1988). They assessed the mental performance of 11 heat acclimatised individuals under different levels of heat stress induced dehydration (1-4% loss of body weight). They reported a significant reduction in mental performance at 2% dehydration, with the mental impairment being proportional to the degree of dehydration. All measured mental functions, that is short term memory, arithmetic efficiency, and visuomotor tracking involving motor speed and attention, were significantly impaired at 2% dehydration. They postulated that the decrements in mental performance were due to heat-induced dehydration, rather than the direct effect of elevated core body temperature on brain function.

Pichan et al. (1988) state that the level of mental attention determines performance status, and that complex tasks may not be adequately or safely accomplished when attention is diverted by thermal stress. In many complex tasks whether they be athletic or industrial, both the mental decision and physiological function are closely related. As a result, Sawka (1992) felt that hypohydration probably has a more profound effect on real life tasks than on solely physiological performance measures. The same author also reported that water deficits of 1-2% of body weight in a neutral environment resulted in no appreciable reduction in maximal aerobic power, but a 6-7% reduction in physical work capacity. Water deficits of 3-4% in the same environment resulted in a 4-8% reduction in maximal aerobic power, and a 22W reduction in physical work capacity. In hot environments, a water deficit of just 2% resulted in a significant reduction in maximal aerobic power (10%) and physical work capacity (22%). Body water losses of 4% in a hot environment resulted in a physical work capacity reduction of approximately 50%, with a 25% reduction in maximal aerobic power. Thus, hypohydration and environmental stress appear to act independently to limit cardiac output, and therefore cellular oxygen delivery, resulting in the reduced capacity to perform work.

It is generally accepted that subjects suffering hypohydration will have an increased core body temperature during exercise in temperate and hot environments, compared to euhydrated subjects. As the magnitude of hypohydration increases, there is a concomitant elevation in core body temperature during exercise (Pichan et al.,1988; Sawka, 1992). Sawka (1992) reports that hypohydration linearly increases core body temperature by 0.15 ^{o}C for each 1% deficit in body water. When hypohydrated, the increased heat storage is due to reduced heat dissipation, largely from the reduction in sweat rate and skin blood flow. Evaporative and dry heat exchange are therefore adversely effected by hypohydration.

To confuse the issue, a study by Pichan et al. (1988) showed that dehydration of 1-2% of body weight resulted in an increased sweat rate, whereas dehydration of 3% resulted in a significant reduction in sweat rate. This study was conducted on 25 heat acclimatised males (aged 20-25 years; mean VO_2 max 39 mL/kg.min), who were hypohydrated to varying levels of body weight (1.3-3.3%) prior to exercising for 40 minutes at 40W in a climate chamber (45 ^{o}C; 30% RH; 34 ^{o}C WBGT). In addition to sweat rate reductions for the 3% dehydrated subjects, it was also stated that sodium and potassium concentrations in arm sweat increased with increasing hypohydration levels. This occurred despite the sweat rates differing significantly between the hypohydration levels. However, increases in potassium concentration were noted only at the 3% dehydration level. The sweat sodium concentration increased from 55

mmol/L euhydrated to 68.3 mmol/L, 75.2 mmol/L and 83 mmol/L for the 1%, 2% and 3% dehydrated subjects respectively. This would be expected if the sweat rate steadily decreased (dilution effect) however, it increased for the 1% and 2% dehydrated subjects and decreased only for the 3% subjects. Pichan et al. (1988) postulate that the increase in sweat electrolytes is therefore not related to sweat rate which is in contradiction to the findings of Sato (1971), Sawka (1985) and Greenleaf and Castle (1971).

Sawka et al. (1985) studied eight heat acclimatised males (mean age 23 years; mean VO_2 max 54.2 mL/kg.min), at euhydration and at 3%, 5% and 7% hypohydration, to ascertain blood and thermoregulatory responses to a hot and dry environment at graded hypohydration levels.
Several conclusions were made concerning hypohydration during exercise in conditions of heat stress.

1) A low to moderate level of hypohydration (up to 3%), primarily reduced plasma volume with little effect on plasma osmolality. It was postulated that the reduced blood pressure activated ADH release, which decreased renal water clearance and defended osmolality.
2) More severe levels of hypohydration (5-7%) resulted in no further volume reduction but a large increment in plasma osmolality. The authors explained the increased plasma osmolality as a result of reduced glomerular filtration rate which activated aldosterone release and sodium absorption, despite elevated plasma sodium levels.
3) Core body temperature and heart rate were increased with severity of hypohydration.
4) Sweating rate responses for a given rectal temperature, were systematically decreased with severity of hypohydration.
5) The reduction in sweating rate was more strongly correlated with hyperosmolality than with hypovolemia.

Nose et al. (1988a) conducted a study on ten male subjects (mean age 27.8 years; mean VO_2 max 50.2 mL/kg.min) who had been dehydrated by 2.3% of body weight following submaximal exercise (40% VO_2 max) in 36 ^{o}C and 30 % RH, to clarify the effect of sweat sodium concentration on the mobilisation of water from the ICF during conditions of thermal stress. The electrolyte concentrations in the plasma were unchanged 60 minutes after dehydration and this suggests that fluid movement between the ICF and ECF compartments was at a steady state. Immediately following dehydration, the mobilisation of ICF water to maintain the ECF was highly correlated with the change in plasma osmolality, and water movement from the ICF space followed the osmotic gradient. The authors also found a strong inverse correlation between free water loss and the concentration of sweat sodium over a wide range. Sodium and potassium concentrations in the sweat averaged 56.4 mmol/L and 9.6 mmol/L respectively, with the sweat being collected in a vinyl bag attached to the forearm. The average water loss was 1100-1400 mL/hr. The free water loss caused the increase in plasma osmolality, resulting in fluid mobilisation from the ICF space to maintain ECF volume. The authors concluded that at a given level of dehydration, the sodium concentration in the sweat determines the volume of fluid mobilised from

the ICF, and thereby determines the effective maintenance of circulating blood volume.

This conclusion emphasises the importance of producing a more dilute sweat in the heat adaptation process. It appears that the sweating rate, in hypohydrated subjects, is lower for any given core body temperature and that the potential for heat dissipation via evaporation is reduced, both resulting in elevated core body temperature. It has also been shown that hypohydration delays the onset of sweating during exercise heat stress (Sawka, 1985; Candas et al.,1986; Sawka, 1992). The physiological mechanisms mediating the reduction in sweat rate with hypohydration are not clearly defined. However, it has been postulated that the single and combined effects of plasma hypertonicity and hypovolemia result in the reduced sweating response.

In another paper, Nose et al. (1985) report a greater loss of osmotically active substances in the sweat, presumably in order to maintain plasma tonicity during thermal dehydration. They also reported that thermal dehydration causes the loss of fluid with an ionic concentration of 70-80 mmol/L (normal 140 mmol/L), so a hypotonic fluid is appropriate to compensate for depleted water and electrolytes from the body. The consumption of water to replace thermal dehydration results in voluntary dehydration, which is postulated by the present author as a defence mechanism against hypoosmolarity. In explanation, because thirst is not a good indicator of dehydration, workers will not sense that they are dehydrated and will fail to replace fluid loss, even when water is readily available. It is well known that people working in the heat will exhibit 'voluntary dehydration'; that is they maintain themselves about 2% of body weight below their ideal hydration status without any sense of thirst (Noakes, 1992).

THE EFFECT OF DRUGS ON BODY FLUID BALANCE

Living cells are permeable to water and therefore if the osmolality of the ECF compartment increases (i.e. there is a loss of hypotonic sweat), one of two things must occur: water or solute must move out of cells to restore equilibrium between the intracellular and extracellular spaces. Sodium is the principal extracellular cation, and the sodium content of the body determines the volume of the ECF. Homeostatic mechanisms exist which regulate the salt and water content of the body and under normal conditions maintain a healthy individual within normal limits. The accepted normal range of values for the concentration of electrolytes in plasma are as follows (Kokko & Tannen, 1990):

sodium	132-142 mmol/L
potassium	3.5-5.0 mmol/L
calcium	4.5-5.5 mmol/L
chloride	98-106 mmol/L
magnesium	1.5-2.0 mmol/L

Unlike sodium, which is located almost entirely in the extracellular compartment, potassium is primarily restricted to the intracellular space. The potassium in the extracellular compartment represents only about 2% of the total body potassium. The concentration of the major cations in the intracellular space are:

sodium	10 mmol/L
potassium	150 mmol/L
magnesium	40 mmol/L

When people are subjected to working in conditions of heat stress, the body temperature increases, and above the ambient temperature of 36 ^{o}C, the only means of heat loss is by evaporation of sweat from the skin. The fluid that is lost initially comes from the ECF and if not replaced may cause body fluid disorders and electrolyte disturbance. If the fluid is replaced with ion free water, then a dilution effect initially on the ECF, can occur. It would be reasonable to predict that if these imbalances are not corrected, then mental impairment which could compromise work safety, may result .

The term dehydration is misleading, since it implies the loss of water alone. This is rarely the case, dehydration is usually associated with losses of both water and salt (in disproportionate or proportionate quantities). Deficits of other ions such as potassium can also occur, and if significant, can cause disturbance to acid-base balance. Dehydration may be classified into three major groups:

1. Loss of water in excess of salt
2. Loss of salt in excess of water
3. Isotonic losses of water and sodium.

Sweating in response to thermal stress produces a fluid that is generally accepted as being hypotonic to the ECF. The loss of fluid can be as high as 3.7 litres per hour (Armstrong & Maresh, 1991). The fluid that is replaced in the body must therefore resemble the fluid lost in order to avoid fluid and/or electrolyte disturbances. To date, eccrine sweat has been an overlooked biological fluid in both clinical and basic studies. As a consequence fluid replacement protocols are based on scant and conflicting data.

The normal regulation of water intake is the sensation of thirst, with water excretion being controlled by the secretion and action of ADH. When free water losses are high, raising the plasma osmolality, ADH is secreted, causing the urine to become more concentrated so that water loss is minimised. At the same time the sensation of thirst is stimulated, causing water to be ingested, and the plasma osmolality returns to normal. However, the sensation of thirst is not a good indicator of hydration status (Nose et al.,1985; Maughan, 1991). A worker will not sense when he is hypohydrated and will fail to replace body water losses, even when drinking water is readily available. Research shows that a person working in the heat will exhibit voluntary dehydration, as previously mentioned they maintain themselves about 2% of body weight below their ideal hydration status without any sense of thirst. Since work performance declines significantly when water loss reachers more than 2% below ideal levels, workers must remember, or be reminded to replace fluid lost to evaporative sweat loss. Chronic dehydration has been associated with an increase in the incidence of several medical problems such as constipation, haemorrhoids, kidney stones and urinary infections, in addition to being an occupational hazard. The likelihood of the these conditions occurring can therefore be reduced by maintaining fluid balance when the loss from sweating is high. The composition and quantity of the fluid used to replace sweat loss therefore, is important.

A surprisingly large number of drugs cause electrolyte disorders. With a number of these agents this effect is an extension of the pharmacology for which the drug is normally used. Consequently, a high percentage of clinicians tend to have an

awareness of this type of disorder and institute sufficient measures to minimise their effects. For many other drugs however, the electrolyte disorder is a 'side effect' which often has an adverse reaction on the user. Unfortunately, these electrolyte disorders are often not well recognised in people who are working in conditions of heat stress and are subject to the added burden of fluid and electrolyte loss from sweating. It is beyond the scope of this work to detail the physiological and pharmacological principles of all drugs that may hinder the user to thermoregulate in the heat. However, a basic classification of disorders that are induced by drugs is presented because of the large number of workers in most workplaces who take some form of regular medication.

No drugs have been implicated in the suppression of thirst, although one study has indicated that the aging process is associated with diminished thirst. Phillips et al. (1984) showed that compared to young controls, elderly men (64-75 years old) showed virtually no thirst after 24 hours of water deprivation. In addition, virtually any drug with depressant effects on the central nervous system can decrease the ability to access free water. Although the age of the subjects in this study is beyond that of the normal working man there may be a lesser effect in people over fifty. To date, no data is available for people in younger age groups.

The ability to concentrate urine is dependant upon the appropriate release of ADH and the ability of the kidney collecting duct to respond to it. Glucocorticoids and opiate analgesics appear to elevate the osmotic threshold for the release of ADH, and as a consequence, a higher increase in serum osmolality is required to release ADH (Aubry et al.,1965; Miller, 1980). Ethanol is also known to suppress ADH release locally (Eggleton, 1942; Kleeman et al.,1955). It should be noted that any of the above acting alone would not cause hypernatremia, however an interplay of drug intake and prolonged sweating may well result in nett loss of water in excess of sodium.

Net sodium gain with resulting hypernatremia can occur with the rapid intake of a number of therapeutic hypertonic solutions containing sodium as the predominant cation (Feig & McCurdy, 1977). Sweat is hypotonic to plasma so has the effect of increasing its osmolality. If copious amounts of high sodium content drinks are consumed to replace sweat loss, the osmolar load they create may result in electrolyte disruption. The ability to excrete a sodium load or gain access to sufficient water may be compromised and take time. It is not unlikely that such a state could arise in the workplace if workers had unrestricted access to such fluids. Some commercially available hydration formulas contain high sodium concentrations, and should therefore be used with caution as the principal rehydration fluid.

Some drugs may stimulate thirst and cause overconsumption of water that in turn may dilute the ECF. The dose may exceed the ability of the body to excrete the water load. This is unlikely to occur in a normal environment. However, if sweat has a high sodium content, which has been shown in this study to occur, prolonged replacement of sweat loss with ion free water may dilute the ECF and cause hyponatremia. The use of drugs with anticholinergic properties such as antihistamines and tetracyclic antidepressants stimulate the thirst response, and may act in concert with sweating (with ion free water replacement) to decrease the sodium

concentration of the plasma to levels that may impair judgement. These drugs may also affect the control system of sweating (sweat innervation is cholinergic).

The intake of large quantities of alcohol is common in many work sites. Alcohol acts as a diuretic and can significantly reduce body fluids and place a worker at risk of hypohydration. The effects of alcohol and caffeine containing beverages on body fluids should not be overlooked. Induction training given to people commencing work in hot climates should include advice on the effects of alcohol, tea, coffee and caffeine containing 'soft' drinks on body fluids. Their use as a rehydration fluid should be warned against. Alcohol in particular would represent the biggest threat, since high consumption in single men's accommodation on mine sites is commonplace. A breakfast free of fluids (other than coffee), following heavy alcohol intake the night before, would be a poor start to a days work in the heat when substantial sweat loss is expected. Workers under such circumstances would quickly become hypohydrated and place themselves at risk of heat illness.

The effects of drugs on body fluid balance can be significant and place a person working in the heat at greater risk of heat illness. It is therefore important that workers be placed in tasks that minimise heat exposure when taking certain prescribed medication. Prevention of heat illness in these workers would be best achieved by a comprehensive medical examination by an occupational physician prior to their deployment to hot work environments. Alternatively, existing workers prescribed medication that may impair fluid or thermal balance should be temporarily placed in tasks that minimises the risk of heat illness. Regrettably this is not currently common practice.

HEAT STRESS AND HEAT DISORDERS

As stated previously, in a few hours of exercise or work in the heat, water loss or dehydration can reach proportions that impede heat dissipation and severely compromise cardiovascular function and work capacity. This occurs as hypovolemia decreases venous return to the heart producing lowered stroke volume and cardiac output. The result is less oxygen delivery to the working muscles. As dehydration progresses and plasma volume drops further, sweating is reduced and thermoregulation becomes progressively more difficult. If not corrected the decrease in circulating plasma volume may cause circulatory failure, with a consequent rise in core body temperature that may be lethal.

Fluid losses are distributed in varying proportions among the plasma, extracellular water, and intracellular water. The decrease in plasma volume that accompanies dehydration may be of particular importance in determining work capacity. Blood flow to muscles must be maintained at a high level to supply oxygen and substrates, but a high blood flow to the skin is also necessary to transport heat to the body surface where it can be dissipated (Maughan, 1991). When the ambient temperature is high and blood volume has been decreased by sweating during prolonged exercise, there may be difficulty in meeting the requirement for a high blood flow to both these tissues. In this situation, skin blood flow is likely to be compromised, allowing central venous pressure and muscle blood flow to be maintained but reducing heat loss and causing body temperature to rise (Maughan, 1991; Sawka, 1992).

If the normal signs of heat stress, such as thirst, tiredness and visual disturbances, are not heeded, a series of disabling complications can occur. The major forms of heat illness in increasing order of severity are:

Heat cramps

Although the precise cause of heat cramps or involuntary muscle spasms has not been elucidated, much of the research concentrates on electrolyte imbalances in the exercising muscle and/or dehydration. Armstrong and Maresh (1991) reported heat cramps in the skeletal muscles of the legs, arms and abdomen of individuals who had lost a large volume of sweat and had drunk a large volume of unsalted fluid. It is assumed that adequate replenishment of both water and electrolytes can prevent heat cramps. These authors also concluded that heat cramps are less likely in acclimatised individuals. The main symptom of this condition is painful involuntary contraction of muscle.

Heat exhaustion

Heat exhaustion is believed to be caused by ineffective adjustments in the circulatory system, compounded by a depletion of extracellular fluid (especially blood volume), owing to excessive sweating and inadequate fluid replacement. Blood usually pools in the dilated peripheral vessels. This combination of factors drastically reduces the venous return necessary to maintain adequate cardiac output. Armstrong and Maresh (1991) report that the incidence of heat exhaustion signs and symptoms is relatively high during the first eight days of heat acclimatisation. This condition is characterised by weakness, vertigo, headache, nausea and peripheral vascular collapse.

Heat stroke

Heat stroke is essentially a failure of the heat regulating mechanisms as indicated by excessively high body temperatures. Although sweating usually ceases upon failure of thermoregulation, continued intense exercise may be accompanied by some sweating, but heat gain by the body greatly outstrips heat loss. If untreated, the disability progresses until death ensues due to circulatory failure and eventual damage to the central nervous system due to hypoxia and hyperthermia. Armstrong and Maresh (1991) report that there is no currently recognised relationship between heat acclimatisation and the incidence of heat stroke which is characterised by hyperpyrexia, cessation of sweating.

Repeated heat stress initiates physiological thermoregulatory adjustments that result in improved exercise capacity and less discomfort on subsequent heat exposures. This heat acclimatisation brings about a favourable distribution of cardiac output and a greatly increased capacity for sweating. Exercise induced heat stress brings about adjustments in peripheral circulation and evaporative cooling similar to those observed with passive heat acclimatisation. Full heat acclimatisation cannot be achieved without actual exposure to heat stress. The training adaptation for thermoregulation is only seen if the person is fully hydrated during work.

In summary the prevention of heat related disorders seems to depend on:

1) The state of acclimatisation.
2) The state of physical training.
3) The anthropometrics of the individual.

The state of hydration and electrolyte balance before exposure to the heat, plus adequate hydration and the maintenance of electrolyte balance during heat exposure would seem to be imperative in preventing heat disorders. Although high intensity exercise in extreme heat can induce heat illness in almost any person, heat acclimatisation reduces the signs, symptoms and incidence of most forms of heat disorders.

Other considerations

Certain skin disorders may result in impaired human thermoregulation and exercise-heat intolerance (Pandolf, 1987). Sunburn is a reaction of the epidermal and dermal layers of the skin to UV radiation (UV-B, 280-320nm) with associated skin injury or phototoxicity (Glazer, 1981). The UV-B light at these wavelengths penetrates the stratum corneum, with 90% of the energy absorbed in the epidermis and 10% in the dermis (Holti, 1955). Most cells in the skin are thought to be damaged, including those of the dermal blood vessels and possibly the sweat glands (DeLeo, 1986). Although the UV-B damage skin is completely healed in approximately three weeks, evidence suggests that the effects on dermal vasculature and on the sweat glands may last longer (Mallory & Watts, 1987). The UV radiation associated with sunburn may impair eccrine sweat gland function (Montagna, 1962). Theoretically, this may be achieved by two possible mechanisms:

1) Inhibition of actual sweat production.
2) Prevention of the progress of sweat from the duct.

Pandolf et al. (1992) hypothesise that sunburn affects local sweating responses, such as sweat sensitivity and core body temperature threshold for sweat initiation, and final local sweating rate. The authors emphasise that during muscular exercise in the heat, human thermoregulation is primarily influenced by the individual's state of heat acclimatisation, level of aerobic fitness, hydration state and clothing worn. However, the authors also demonstrated that mild sunburn had the potential to alter local sweating responses indicating diminished sweat gland function, as opposed to a decrement in the sudomotor drive. It was concluded that local sweating responses, such as sweat sensitivity, and final local mean sweat rates were reduced 24 hours post-sunburn as compared to the pre-sunburn sweat losses under the same conditions. This suggests that mild sunburn has an effect, locally mediated, on both the responsiveness and capacity of the sweat gland to deliver sweat to the cutaneous surface. The precise physiological mechanisms for this is yet to be elucidated. In conjunction, the subjects displayed enhanced subjective distress after sunburn during exercise heat exposure, as measured by elevated perceptual responses to rated perceived exertion and thermal stress (Pandolf et al.,1992).

FLUID REPLACEMENT IN THE HEAT

Barr et al. (1991) reported that the need for sodium replacement would be unexpected for exercise of less than 6 hours duration in physically trained, acclimatised individuals. They recommend the replacement of 400-600 mL/hr fluid replacement during exercise at 55% VO_2 max in warm/humid conditions, in order to prevent hyponatremia. Gisolfi and Duchman (1992) stated there is no need for sodium replacement for sports events lasting less than 3 hours. However, ultra endurance events may indicate the need for sodium replacement in order to prevent hyponatremia. Convertino (1991) reported average sweat rates of 800-900 mL/hr for

subjects exposed to 42 ^{o}C and 93% RH. However, most well trained individuals have sweat losses considerably above this figure. Montain and Coyle (1992) reported that the replacement of sweat loss when exercising resulted in a reduced heart rate and core body temperature, with maintenance of stroke volume during the second hour of exercise. They concluded that fluid ingestion maintains a high skin blood flow, thus reducing hyperthermia by attenuating increases in serum osmolarity, sodium concentration, or some other substance that is influenced by dehydration. Millard-Stafford (1992) in a review article recommends that each individual calculate their own sweat rate based on net changes in body weight over time, and match their fluid dose accordingly, although she suggests that highly trained individuals may require at least 1000 mL/hr. The same author also stated that additional scientific data is needed to determine the ideal fluid dosage when competing in the heat. As a consequence of this gap in the literature there are several fluid replacement regimes recommended for people exercising in the heat. Whether or not these fluid dosages are appropriate for women, children and older adults in the heat is not well documented. Individual variation of sweat loss seems therefore to be the basis for the recommendation that fluid replacement should be individually calculated.
Gisolfi and Duchman (1992) recommend the consumption of 300-500 ml for events lasting an hour and 800 - 1000 mL/hr for events lasting more than 3 hours. These authors also stated that the amount of sweat lost will range between 800-1600 mL/hr for most athletes. The difference in the fluid lost and recommended replacement, is that there is no advantage in consuming fluids is excess of 65% of fluid lost. It should be pointed out that factors such as stomach fullness, and so an uncomfortable feeling, as well as gastric emptying, contribute to these recommendations. The rate at which the athletes worked was between 65-90% VO_2 max. The recommended work rate maximum for a worker over the duration of a 8 hour shift is 40% VO_2 max. It would be reasonable therefore to question the application of these fluid replacement guidelines to the workplace setting. One noteworthy finding, however, is that most athletes voluntarily replace only half their fluid loss at moderate exercise intensity (60% VO_2 max) , and at high intensity (>60% VO_2 max) less than half. The percentage voluntary replacement of lost fluid by workers would be of interest, given they work at much lower intensities.

Maughan (1991) found under controlled conditions that the sweating rate during 1 hour of exercise at a workload of 70% VO_2 max in 23 ^{o}C, ranged from 426-1665 mL/hr. However, the need for fluid replacement was greater in those individuals that sweated profusely. The relationship between VO_2 max of the subjects and fluid loss, however, was not given. No other explanation was given for the significant variation in fluid loss in these subjects. Maughan (1991) went on to say that there is no physiological requirement for sodium replacement during exercise of less than 3 hours of duration, but there may be an advantage in adding sodium to drinks to avoid the danger of hyponatremia for events exceeding 3 hours in duration. The same author recommended sodium replacement post-exercise up to 80 mmol/L to ensure that adequate rehydration be established.

It was reported that maximum hourly gastric emptying rates are between 1 and 1.5 litres (Montain & Coyle, 1988; Armstrong & Maresh, 1991; Sawka, 1992). Gastric emptying rate is influenced by a variety of factors including, but not limited to, fluid temperature, osmolality, sodium content, fat content, pH and electrolyte content.

Maximal gastric emptying rates are reportedly decreased during high intensity exercise (>75% VO_2 max), hypohydration and heat strain (Rehrer, 1991a; Sawka, 1992). Brouns (1991), and Millard-Stafford (1992), reported that it is impossible to give general guidelines with regards to fluid replacement due to the widely differing sweat rates of all individuals. Rehrer (1991a) concluded that fluid should be taken early in an exercise period before dehydration sets in, at a rate of approximately 1 L/hr. Sawka (1992) showed difficulty in the ability of human beings to balance fluid output with fluid intake during exercise-heat exposure. They also reported that thirst provides a poor index of body water requirements, and that ad libitum drinking results in inadequate fluid replacement or voluntary dehydration. Sawka (1992), in a review article, stated that in hypohydrated humans the volume of the rehydration fluid consumed is highly correlated with plasma hyperosmolality.

Candas et al. (1986) reported that significant hypovolemia was associated with sweating depression, and that sweat rate reductions were associated with hypohydration. However, these authors failed to support these observations in a study conducted on four unacclimatised male subjects (aged 20-23 years), exercising at a low workload for three hours in a climate chamber (34 ^{o}C; 10 ^{o}C dew-point) under various dehydration and rehydrative states. The only alteration noted with respect to the eccrine glands was increased sweat sensitivity induced by the ingestion of water during the exercise, as compared to the ingestion of a hypertonic (846 mOsmol/L derived from glucose and fructose), hypotonic (120 mOsmol/L) or isotonic (293 mOsmol/L derived from sucrose and electrolytes) solution, or no fluid consumption at all. This observation supports the hypothesis that plasma osmolarity may have some direct influence on sweating regulation, however only the condition of no fluid intake produced a significantly higher plasma osmolarity. It was also reported by Candas et al. (1986) that dehydration and consumption of a hypertonic solution during exercise induced heat stress was associated with significantly higher heart rates at a given workload, compared to the other rehydrative states. The authors believed that the hypertonic solution delayed water absorption, however, the recorded change in plasma volume for this condition over the exercise period was not significant and does not support their hypothesis. Candas et al. (1986) concluded that the consumption of water or a hypotonic solution as a rehydration drink restored all physiological parameters to pre-test levels. Hypertonic solutions increased heart rate and reduced sweat sensitivity during rehydration, while isotonic solutions resulted in an expanded blood volume but no accompanying reduction in heart rate was demonstrated.

Glucose drinks from <2.5% to 10% are suitable for adequate fluid and carbohydrate replacement for events lasting longer than 90 minutes. Although elevations in blood glucose resulted in an insulin release that inhibited lipolysis, solutions up to 10% glucose showed no significant change in plasma insulin levels during exercise (Davis et al.,1990; Rehrer, 1991b). Carbohydrate solutions of 12% consumed during prolonged intermittent exercise in the heat, have slower absorption rates and may therefore increase the risk of gastrointestinal distress and performance decrements (Davis et al.,1988). The type of carbohydrate, simple, maltodextrins or polymers does not affect the availability for their dilution in body fluids and they are absorbed at similar rates (Davis et al.,1990).

THE ECCRINE SWEAT GLAND

The eccrine sweat gland is a secretory as well as an excretory organ. Sweat is a dilute electrolyte solution containing mainly sodium (Na^+), chloride (Cl^-), potassium (K^+), bicarbonate ($HCO3^-$) and inorganic compounds such as lactate, urea, and ammonia (Sato et al.,1991). The primary function of sweating is the maintenance of body temperature during exposure to environments inducing heat stress, or to physical activity. The excretory function of the sweat gland is to rid the body of orally ingested ketoconazole and griseofulvin to the stratum corneum thus bypassing the slow diffusive pathway across the epidermal cell layer (Shah et al.,1974; Harris et al.,1983).

The gland is a simple tubular epithelium consisting of a secretory coil and duct. Embryologically, it evolves as a cord of epithelial cells forming from the epidermal layer of the skin on the palms and soles at 3 months, and on the rest of the body by 5 months. By the eighth month of gestation they resemble adult sweat glands (Ellis, 1967). The coiled portion of the sweat gland is for secretion and has a proximal duct. The distal portion is reasonably straight and connects with the epidermis of the skin. In the epidermis and stratum corneum the duct becomes coiled again before opening onto the skin surface (Sato et al.,1991). The size of a sweat gland can vary considerably between individuals. Sato and Sato (1983) have reported size differences as great as fivefold. The same authors stated that this correlates well with individual variation in sweat rate and possibly regional differences of secretion. The duct of the sweat gland is composed of 2 layers of cells which look similar at the light microscope level. However, at higher magnification there are distinct differences. The basal ductal cells are rich with mitochondria and the cell membrane has enzyme concentrations suggestive of the membrane being involved in Na^+ pumping for ductal Na^+ absorption. Luminal cells on the other hand have few mitochondria, with the membrane serving as an absorptive surface by housing Na^+ and Cl^- channels (Sato & Sato, 1988).

Control of sweating

When the core body temperature increases as a result of thermal and/or metabolic load, thermo-sensitive neurons in the pre-optic region and hypothalamus sense these changes and respond to maintain a constant core body temperature (Bouland, 1981). Local heating of the hypothalamus and pre-optic region of animals brain activates sweating, panting and vasodilation (Smiles et al.,1976; Adair, 1977) and cooling the pre-optic region initiates shivering. Bouland (1981) demonstrated that some pre-optic neurons can be classified as warm sensitive and cool sensitive. Heating the warm sensitive neurons alters their firing rate, as does an increase in afferent impulses from cutaneous and spinal thermoreceptors in response to an increased core body temperature. He proposed that the pre-optic neurons may be involved with thermal sweating. Nadel et al. (1974) observed that sweating may be controlled by a weighted sum of core body and skin temperatures, however they also stated that a rise in core body temperature seemed the dominating influence in sweat activation. They go on to state that the temperature set point for the initiation of sweating changes with acclimatisation in normal subjects, in different homeostatic states, and during exercise or heat exhaustion. It is well known that sweating is reduced or absent during heat

exhaustion despite a marked increase in core body temperature. Greenleaf (1979) attempted to determine whether changes in plasma electrolyte concentration may change the core body temperature set point. He reported that the infusion of hypertonic saline into the brain of experimental animals raised the body temperature by 1 ^{o}C. The same response occurred when other methods were used to produce a state of hypernatremia. Nielsen et al. (1988), reported that elevation of plasma sodium levels of humans when exercising at 40% VO_2 max slowed the onset of sweating and raised the plateau level of the core body temperature. Conversely, increasing the level of plasma calcium initiated an early onset of sweating and lowered the core body temperature plateau. A change in the temperature set point may play an important role in normal and abnormal physiological states, and may partially explain why hyperthermia occurs during heat stroke. Fortney et al. (1981) demonstrated that isotonic hypovolemia reduces the sensitivity of the sweating response, delays the onset of sweating, and increases the threshold for the onset of cutaneous vasodilation.

Innervation of sweat glands

Sweat glands are supplied by sympathetic postganglionic fibres. However, in contrast to the ordinary sympathetic innervation, acetylcholine is the principal terminal neurotransmitter. The work of Uno and Montagna (1975) demonstrated the presence of both adrenergic and cholinergic innervation to the periglandular tissue in both human and monkey sweat glands. This finding decided the debate of dual innervation to sweat glands. More recently, vasoactive intestinal peptide (VIP) has been noted in the periglandular nerves of the human eccrine gland (Lundberg et al.,1979). It appears that periglandular norepinephrine (NE) is complimentary to VIP and has functional similarities. Both VIP and NE are only 20-50% as effective as acetylcholine as a stimulant for sweat secretion (Sato, 1977; Sato & Sato, 1984). Sato et al. (1991) concluded that the biochemical significance of multiple innervation of the sweat gland may be to maximally increase the cyclic adenosine mono-phosphate (cAMP) accumulation in the secretary cells (cAMP acts as a transport messenger).

Mechanism of sweat gland function

Sodium concentration in the primary fluid (the fluid at the point of production) is isotonic to plasma irrespective of the sweat rate (Sato, 1982). The concentration in secondary sweat reaching the skin must therefore be dependent on the absorption of sodium by the cells lining the duct of the sweat gland. The capacity of ductal absorption is dependent on the time the fluid is in transit from the coil to the skin surface, and has been reported to be inversely related to sweat rate (Sato, 1982). This would suggest that the sodium concentration of sweat increases with an increasing sweat rate, due to decreased absorption. It has been observed however, that sweat sodium increases at low sweat rates both in vivo and in vitro (Sato, 1982). The mechanism of sodium ductal absorption is poorly understood although it has been reported that aldosterone can increase sodium ductal absorption (Sato & Dobson, 1971). Whether aldosterone is the principal regulator of ductal absorption of sodium is, however, still unclear.

It is well documented that acetylcholine increases the potassium permeability of exocrine cells (Bovell et al.,1989; Takemura et al.,1991; Sato et al.,1991). In salivary

and lacrimal acini this is due to the activation of calcium-activated and voltage-dependent potassium channels (Bovell et al.,1989). The permeability increase is divided into two components:

1) A transient phase, independent of extracellular calcium and attributed to the mobilisation of calcium from cytoplasmic stores.
2) A sustained calcium dependent phase, due to an influx of extracellular calcium.

Preliminary studies on the human sweat gland demonstrate that the transient phase is inhibited by the removal of external sodium (Wilson et al.,1990). Both phases are inhibited by the removal of external bicarbonate (Wilson et al.,1990; Takemura et al.,1991). Reddy and Quinton (1991) reported that potassium conductance across the basolateral membrane could be modulated by sodium transport status in the apical membrane.

The response of the human sweat gland to acetylcholine involves both the release of bound intracellular calcium and calcium influx (Sato et al.,1991). As already mentioned, acetylcholine is the primary inducer of sweat in human sweat glands, although the sudomotor control mechanisms in all species has an adrenergic component. Adrenaline, in contrast to acetylcholine, only evokes the calcium-dependent sustained phase, resulting in calcium influx (Bovell et al.,1989). It is suggested that potassium permeability of the secretory cells increases during activity (Bovell et al.,1989). It has been shown that a blockade of the potassium conductance channels in the basolateral membrane inhibits sweat secretion, and that a fall in cytoplasmic potassium accompanies sweat secretion (Bovell et al.,1989). Microanalytical studies reveal that a fall in the cytoplasmic potassium concentration does not occur in the active coiled duct of the human sweat gland, and therefore the increased potassium efflux is attributed almost exclusively to the secretory cells (Bovell et al.,1989; Wilson et al.,1990). Human sweat is always hypertonic compared to plasma in its potassium concentration, but the actual specific source of the potassium is unknown. Potassium may be secreted hypertonically into the primary sweat by the secretory coil, or into the lumen of the reabsorptive sweat duct. Reddy and Quinton (1991) reported no evidence of potassium transport systems across the apical membrane. Since potassium concentration of sweat increases with decreased rates of sweat secretion, greater water reabsorption across a potassium-impermeable membrane may increase potassium concentration in the lumen of the sweat duct. The authors argue that this is not the sole reason for potassium accumulation in the sweat, and that it is more likely that the primary sweat from the secretory coil is the source of higher potassium concentrations in the sweat. It is also reported that the concentration of intracellular potassium in the sweat duct cell is above equilibrium, due mainly to the activity of the sodium potassium pump in the basolateral membrane (Sato et al., 1991).

In summary, the sweat change that occurs going between the cytoplasm and the surface of the skin seems to occur in the following way:
The fluid for sweat is derived from the interstitial fluid and is believed to be isotonic and similar to plasma. If this is the case, the sodium concentration would be approximately 145 mmol/L and the potassium concentration approximately 4 mmol/L. Upon nervous stimulation via acetylcholine in response to an increased core body temperature, the secretory cells of the eccrine sweat gland apparently release

potassium into the surrounding isotonic fluid. The potassium efflux is due to the opening of calcium-dependent, voltage controlled channels in the cell membrane. These channels are 'opened' by an increase in intracellular calcium. This acetylcholine-stimulated calcium mobilisation can be divided into two phases:

1) Transient phase, which is dependent upon extracellular sodium and bicarbonate concentration, where intracellular calcium stores are mobilised.
2) Calcium-dependent sustained phase, which is dependent upon the extracellular bicarbonate concentration.

Sweat can only be produced if the potassium channels are opened to allow potassium efflux. At this point, the primary sweat would probably still be very close to isotonic, but with a slightly increased potassium concentration and reduced calcium concentration. This fluid would then move up the absorptive sweat duct where sodium and water are absorbed through the apical membrane of the eccrine duct cells. The absorption of sodium occurs by virtue of the electrochemical driving force created by the efflux of intracellular potassium at the basolateral membrane. Sodium influx into the cells continues until a new steady state is created, which is equal to the sodium efflux across the basolateral membrane by the sodium potassium pump. This suggests that initial sweat production may be lower in sodium concentration due to the initial large influx of sodium across the apical membrane continuing until a new intracellular steady state is created. The sweat concentration would be approximately 6-9 mmol/L of potassium and a hypotonic sodium concentration. The concentration of the sodium would depend on the sweat rate, since the faster the transit time of the fluid through the duct, the less sodium can be absorbed. In addition to the transit time, the size and density of sweat ducts may also partially determine the concentration of the sweat upon arrival on the skin surface. These factors could at least partially explain the individual variation of sweat rate that exists.

PHYSIOLOGICAL ACCLIMATISATION TO HEAT

Heat tolerance is defined as the level of physiological strain associated with exercise in the heat. High heat tolerance is characterised by a small increase in heart rate, core body temperature and rate of body temperature change, a high muscle blood flow and a high rate of sweat secretion and evaporation at a given exercise intensity. Heat tolerance may be achieved by heat acclimatisation, and further increased by physical training (Gavhed & Holmer, 1989; Pandolf et al.,1992).

The role of acclimatisation in the prevention of heat related disorders has been well documented. Sutton et al. (1977), Libert et al. (1988), Gavhed and Holmer (1989), Sato et al. (1990) and Armstrong and Maresh (1991) all reported that heat acclimatisation involves a complex of adaptations which include decreased heart rate, core body temperature, perceived exertion as well as increased plasma volume and altered sweat gland function. However, whether heat acclimatisation actually increases sweat rate remains unclear. Reported increase in sweating efficiency with heat acclimatisation is associated with reductions in both skin and internal temperatures (Libert et al.,1988). The fall in heart rate at rest and at work with heat acclimatisation is indicative of the diminution in physiological strain induced by the environmental stress (Libert et al.,1988; Gavhed & Holmer, 1989). This results in an improvement in the ability to perform muscular work (Libert et al.,1988; Armstrong et al.,1990; Kenney & Johnson, 1992). A reduction in the Na^+ and Cl^- levels in sweat

and urine with heat acclimatisation has been reported, but some authors have reported that losses are dependent on environmental conditions and diet (Brandenberger, 1989; Armstrong & Maresh, 1991). Brandenberger et al. (1989), and Kenney and Johnson (1992) also reported reduced urinary volume, increased urinary osmolality, reduced urinary sodium loss, increased plasma volume, increased plasma osmolality and increased plasma sodium and protein levels with heat acclimatisation. However, there is little agreement on the duration, intensity, and climatic conditions necessary to induce the physiological adaptations associated with repeated heat exposure. The following review of papers on acclimatisation clearly demonstrates this diversity.

In a review article by Armstrong and Maresh (1991), it was concluded that up to 14 days is required before full physiological heat acclimatisation is observed. Whether this is in conditions of continuous heat exposure or intermittent heat exposure, with or without physical activity is unclear. The exact environmental conditions for derivation of these results is found in Armstrong and Dziados (1986). The early heat acclimatisation adaptations primarily involved an improved control of cardiovascular function, including expanded blood volume, reduced heart rate, and autonomic nervous system habituation which redirects cardiac output to skin capillary beds and active muscle. These processes are reportedly completed after six days heat exposure. These are then replaced by longer lasting adaptations such as increased sweat rate (after day 8), reduction in sweat sodium concentration (after day 6) and decreased skin blood flow.

A previous study by Armstrong et al. (1990), conducted on 15 male subjects (mean age 25yrs; mean VO_2 max 51 mL/kg.min) who performed 90 minutes of treadmill exercise for 7 days in hot/dry conditions (40.1 ^{o}C; 23% RH), reported acclimatisation within 7 days (10.5 hours total heat exposure). This was indicated by statistically significant reductions occurring in final heart rate and rectal temperature after the 7 days. The mean sweat rate, sweat sensitivity and sweat sodium concentrations were not significantly different from day 1-day 7. It was concluded that sweat rate was not significantly increased until after 10 days of heat acclimatisation (15 hours total heat exposure).

Pandolf et al. (1988) showed an increased sweat rate 7 days after 120 mins of heat exposure to hot/dry (49 ^{o}C; 20% RH) conditions per day, while exercising at 45% VO_2 max (14 hours total heat exposure). Libert et al. (1988) however, reported that during the first three days of continuous heat exposure to hot/dry conditions, cardiac adaptive adjustments and sweating adjustments occur. The 6 male subjects in this study (mean age 24 years; mean VO_2 max 49 mL/kg.min) exercised at 35% VO_2 max for a total of 120 mins per day in a climate chamber (35 ^{o}C; 7% RH). The sweating rate of the subjects increased initially but declined after the first three days of continuous heat exposure, presumably in order to maximise circulatory stability.

Brandenberger et al. (1989) also reported an increased sweat rate in the first 3 days of continuous passive heat exposure to a hot/dry environment (35 ^{o}C; 7% RH) in young (20-29 year old) males without physical exercise being performed, which then declined without any concomitant increase in body temperature. After 5 days of continuous heat exposure (120 hours), significant increases were found in plasma volume, osmolality, plasma sodium and protein levels. Pandolf et al. (1988),

however, showed no changes in plasma osmolality or plasma sodium after 10 days of heat acclimatisation which involved exposure to hot/dry conditions (49 ^{o}C; 20% RH) for 120 mins per day while exercising at 45% VO_2 max (20 hours total heat exposure).

Maughan (1991) reported an increase in plasma volume with heat acclimatisation was associated with an increase in sweating rate. This occurred within the first few days of exercise in the heat (8-12 hours total heat exposure). The main adaptations were an increased sweat sensitivity, an increased sweat rate and an improved thermoregulatory response, which occurred slightly later than the cardiovascular adaptations, with little change in the first 4 days (after 16 hours total heat exposure). The subjects were exercising at 40% VO_2 max for 4 hours/day for 10 days in 45 ^{o}C (40 hours total heat exposure).

Armstrong and Maresh (1991) reported that the nature of fluid and electrolyte adaptations during heat acclimatisation are dependent upon the hydration status and dietary NaCl intake of the individual. This study showed that NaCl intakes below 3 g/day theoretically place athletes at increased risk of heat disorder. This experiment was performed on eight male subjects (mean age 23 years; mean VO_2 max 4 L/min) exercising at 60% VO_2 max in a hot environment (35 ^{o}C). The sweat sodium loss as ascertained by the whole body washdown procedure was 49.7 mmol/L for the normal sodium intake and 38.4 mmol/L for the low sodium intake. Potassium sweat concentrations did not change for either diet and were 3.7-4 mmol/L.

Barr et al. (1991) reported sweat sodium concentrations of 26-29 mmol/L for well trained (55-60 mL/kg.min VO_2 max), acclimatised individuals (mean age 28 years), exercising in warm/humid conditions (30 ^{o}C; 50% RH), using a whole body washdown technique. They also showed that sweat sodium concentrations were comparatively lower during exercise when the subjects were well hydrated, however, the sweat sodium concentrations increased proportionately with the level of dehydration.

Heat acclimatisation results in a reduction in sweat NaCl concentrations. This reduction in total salt loss results in a smaller reduction in plasma volume following exercise in heat acclimatised individuals (Armstrong & Maresh, 1991). Armstrong et al. (1990) reported no significant reductions in sweat sodium concentration following a 7 day heat acclimatisation procedure (10.5 hours total heat exposure). Sweat sodium concentration values, as determined by the whole body washdown technique, ranged between 31 and 45 mmol/L for young (25 years), well trained (mean VO_2 max 51 mL/kg.min) individuals.

Gisolfi and Duchman (1992) showed that heat acclimatised subjects lose considerably less salt at a given sweat rate than their unacclimatised counterparts, but the amount of sodium lost increases with increasing sweat rates.

In a review article, Sawka (1992) stated that over the course of heat acclimatisation, the sweat sodium concentration decreases by 59%, despite a sweat rate increase of 12%. Therefore, the heat acclimatised individual loses less solute from the plasma for

a given sweat rate. In other words, for a given amount of body water lost as sweat, the heat acclimatised individual retains more solute in the plasma which osmotically draws greater amounts of water from the intracellular space, compared to the unacclimatised individual. This imparts a greater advantage to the heat acclimatised individual, especially in hypohydrated states, since the plasma volume necessary to maintain circulatory and thermoregulatory stability is better defended. The same study showed that heat acclimatised individuals have a smaller plasma volume reduction for a given body water loss compared to an unacclimatised individual. Sawka (1992) also supports the theory that when hypohydration is induced by a more hypotonic sweat, as in heat acclimatised compared to unacclimatised individuals, the lower magnitude of hypovolemia reported is due to the greater redistribution of intracellular water.

Maughan (1991) reported a range of sweat sodium concentrations between 20 -80 mmol/L, showing that sweat composition undoubtedly varies between individuals, and in the same individual depending on the state of training and heat acclimatisation. The sweat rate increases with training and acclimatisation, and electrolyte content decreases.

Some authors have reported increased sodium concentration of the sweat with increasing sweat flows, while others have found no relationship between sweat sodium concentration and flow rate. The explanation given by those reporting the former is that a higher sweating rate decreases the time available for sodium to be reabsorbed in the duct of the sweat gland (Maughan, 1991; Meyer et al.,1992). The potassium content of the sweat remains unaffected by sweat rate and is measured to be between 4-8 mmol/L (Maughan, 1991; Meyer et al.,1992).

Heat acclimatisation results in increased plasma volume (Armstrong & Maresh, 1991; Kenney, 1988). This increase in plasma fluid is important to the exercising athlete in hot environments. Armstrong et al. (1990) reported blood volume expansion by day 8 in young, fit male subjects exposed to 90 mins of hot/dry conditions, (40.1 ^{o}C; 23% RH), per day while performing treadmill exercise. Total heat exposure was 12 hours.

Greenleaf (1979), and Kenney and Johnson (1992) showed that heat acclimatisation activates various mechanisms of the water intake mechanism (thirst). Interestingly, the drive for thirst is proportional to the blood osmolality, which is related to core body temperature. Fellmann (1992) reported that the combination of exercise and heat enhances hypervolemia. The magnitude of the plasma expansion (9-25%) depends on preceding physical training, ambient conditions, duration and intensity of exercise, body posture and genetic potential.

The adaptations of the sweat gland are distinctly different during dry and humid heat exposure according to Armstrong and Maresh (1991). Humidity played an important role in that the sweat rate remained essentially unchanged in dry heat, but increased markedly during hot, humid trials. This conclusion was revealed by reviewing previous heat acclimatisation studies. Unfortunately the environmental conditions, exercise intensity, length of heat exposure and the physical and morphological characteristics of the subjects involved in these studies were not reported. In the same paper it was also indicated that another important factor in sweat gland adaptation was the absolute rate of sweating. If hourly sweat rate does not exceed 400 - 600 mL/hr,

increases in whole body sweat rate may not occur. In contrast to these findings, Smolander et al. (1987) showed identical sweat rates in hot/dry and warm/humid environments for physically trained, unacclimatised individuals.

Seidman et al. (1991) reported sweat rates of 680-720 mL/hr in highly trained (mean VO_2 max 58 mL/kg.min) acclimatised 20 year olds exercising in warm/humid environments at an unknown work level. In agreement, Barr et al. (1991) showed mean sweat rates of 720 mL/hr in highly trained individuals (59 mL/kg.min VO_2 max) exercising at 55% VO_2 max in warm/humid conditions. Davis et al. (1988) reported sweat losses of 1.6 L/hr for well trained (63 mL/kg.min), acclimatised individuals exercising at the higher rate of 75% VO_2 max in 28 ^{o}C and 68% RH. If the sweat rate does increase following heat acclimatisation, the individual exercising in the heat would have to drink more water to maintain normal water balance. An extreme case was cited by Armstrong and Maresh (1991). They reported sweat rates of 3.7 L/hr in a world class marathon runner after 19 days of intense heat acclimatisation. However, the body would become dehydrated as the maximum gastric uptake of water is approximately 1-1.5 litres/hr. The fluid loss over the 140 minutes it took to run the race could not be replaced by drinking.

The onset of sweating is reportedly earlier in physically fit subjects, but there is no increase in overall sweat rate between unacclimatised and acclimatised subjects. This was found at exercise intensities of 60-65% VO_2 max at 30 ^{o}C and 35% RH in trained marathon runners by Armstrong and Maresh (1991). The same authors also stated that sweat rate increases were not apparent until day 10 of heat acclimatisation, after approximately 15 hours total dry heat exposure. In agreement with the finding of sweat onset, Pandolf et al. (1988) reported that sweating onset time was reduced significantly following heat acclimatisation.

Complicating the issue, Libert et al. (1988) showed that heat acclimatisation resulted in a rapid increase in sweat rate response, which was later modified leading to a sweat rate reduction. The sweat rate increase occurred within the first 3 days of continuous heat exposure (35 ^{o}C; 70% RH) in six male subjects (mean age 20-29yrs; mean VO_2 max 49.1 mL/kg.min) exercising at 35% VO_2 max. After the first 3 days (72 hours), sweat rate declined without any corresponding change in body temperature. This finding was supported in another study by Brandenberger et al. (1989).

Heat exposure without exercise (passive heat acclimatisation), induces some heat acclimatisation responses, notably an improved ability to dissipate heat. Most investigators agree that both physical training in a cool environment and a large inherent VO_2 max, improve physiological responses to exercise at high ambient temperatures. This partial heat acclimatisation state has been attributed to one or more of the following factors: earlier onset of sweating, improved body fluid dynamics, high cardiovascular stability and high VO_2 max. The rate at which heat acclimatisation occurs being related to cardiorespiratory fitness. In one study (Armstrong & Maresh 1991) subjects who had a VO_2 max of 65 mL/kg.min or greater, achieved a plateau of heart rate and rectal temperature within four days of heat exposure. In contrast, subjects who had a VO_2 max of 50 and 40 mL/kg.min required an average of 6 and 8 days respectively, to reach the same plateau.

It is essential for people to exercise in the heat if maximal heat acclimatisation effects are desired. Because the maintenance of an elevated core body temperature appears to be the stimulus for the induction of optimal heat acclimatisation, strenuous interval training or continuous exercise at 50% VO_2 max is required according to Armstrong and Maresh (1991). Other authors concluded that core body temperature must increase during exercise training to stimulate sweating for physical training in order to improve exercise heat tolerance. These findings support the contention that VO_2 max per se may not be as important in determining exercise heat tolerance as the thermal adaptations associated with attaining the fitness level (Pandolf et al.,1988). Smolander et al. (1987) showed a greater sweat rate in physically trained unacclimatised individuals compared to physically untrained individuals, and concluded that the higher sweat rates are coupled to greater aerobic fitness. This is also supported by Brouns (1991) who reported improved quantitative sweat secretion and sensitivity of sweat glands to a thermal load after physical training. It was also stated that 'trained sweat glands' will respond with an earlier onset of sweat secretion. Brouns (1987) also showed that well trained individuals have developed the ability to minimise the excretion of electrolytes by the sweat glands (mean 32.7 mL/L ascertained by whole body washdown technique).

The electrolyte composition of sweat differs between passively induced (sauna) and exercise induced sweating. Gavhed and Holmer (1989) showed that the effects of physical training on the cardiovascular system are similar to that achieved by heat acclimatisation, such as increases in plasma volume and stroke volume. This study was conducted on unacclimatised individuals (mean age 33 yrs; VO_2 max 47 mL/kg.min) exercising in 36 ^{o}C and 52% RH at 75W on a cycle ergometer. It was concluded that endurance type physical training enhances sweating and improves cardiovascular function. Also, the duration of the experiment for each individual was best correlated with VO_2 max.

Millard-Stafford (1992), and Convertino (1991) demonstrated that highly trained individuals have a higher sweat rate than their untrained counterparts. One example confirming this was reported by Gisolfi and Duchman (1992), where they state, 'highly trained endurance athletes can sustain metabolic rates of 1000W for several hours without becoming hyperthermic because of their capacity to produce more sweat'.

Smolander et al. (1987) showed significantly higher sweat rates (475 $g/m^{-2}.hr$ compared to 270 $g/m^{-2}.hr$) for well-trained individuals (mean VO_2 max 59 mL/kg.min), compared to less well-trained counterparts (mean VO_2 max 40 mL/kg.min), in both hot/dry and warm/humid environments.

In a review article Convertino (1991) stated that the magnitude of hypervolemia induced by exercise training is related to increased sweat rate and evaporative cooling during exercise. Exercise training can induce changes in thermoregulatory mechanisms, such as increased recruitment and sensitivity of sweat glands. Fellmann (1992) showed that hypervolemia seen with exercise training was beneficial, since trained individuals sweat more than their untrained counterparts. However, it was

also observed that plasma volume expansion had no effect on the control of sweating at any anatomical site.

It has been well documented that when people work in a hot environment for the first time, their work performance is reduced, their heart rate and core body temperature increases greatly, and they are more prone to heat disorders, such as heat syncope and heat exhaustion (Smolander et al.,1987). The same authors reported that during unacclimatised physical exercise in the heat, the reduction of stroke volume was the important mechanism limiting physical performance. In a hot environment at heavy workloads, the lowered stroke volume prevents any thermally induced rise in cardiac output, and increments in skin blood flow occur through the redistribution of the blood flow from visceral organs and inactive muscles. These authors concluded that unacclimatised individuals cannot reach a circulatory and thermal steady state during heavy work in the heat, and the work periods tend to be short. Heat stress resulted in more pronounced sweating often resulting in dehydration. These same authors also speculated reduced sweat sodium concentrations among well trained individuals exercising in the heat.

Greenleaf (1979) demonstrated that with continued sodium depletion, the rate of water loss is reduced via progressive decreases in sweating and urinary flow. Whether this decrease in water flow resulted in increased concentrations of sweat and urinary sodium was not stated.

In a study using patas monkeys, Sato et al. (1990) provided experimental evidence that increased sweat gland function with heat acclimatisation was associated with significant morphological changes in the sweat gland itself. Heat acclimatisation increased the size and secretory capacity of eccrine sweat glands, and these larger glands produced more sweat. The sensitivity of the sweat gland to nervous stimulation was also increased following heat acclimatisation. The monkeys of this study were acclimatised to warm/dry conditions (33 ^{o}C; 13% RH) for 9 months prior to the commencement of the experiment.

In hot workplaces, the metabolic demands are usually relatively low (30% VO_2 max), however, the work periods can last for several hours and work overalls are often worn which can cause significant extra heat strain because of their insulative properties. Smolander et al. (1987) conducted a study to ascertain the different thermal and circulatory responses of unacclimatised individuals with differing physical fitness levels in a warm/humid and a hot/dry environment. The trained group had a mean VO_2 max of 59.1 mL/kg.min, the untrained 40.6 mL/kg.min. The mean age of all subjects was 32 years. The environmental conditions were 40 ^{o}C and 20% RH for the hot/dry and 30 ^{o}C and 80% RH for the warm/humid. These environments were matched to give the same heat stress index value in terms of WBGT. The subjects walked on a treadmill at 30% VO_2 max for 3.5 hrs in both environments. It was found that the physically trained individuals had higher evaporation and sweat rates, and that sweat rate did not differ between environments, however, the evaporation rate was significantly lower in the warm/humid environment. It was concluded that during acute heat exposure, impaired work performance was due mainly to circulatory instability caused by the inability of the peripheral circulation to increase heat dissipation. Although the environments were matched for WBGT, the warm/humid

environment exerted the highest thermal strain as indicated by the slightly higher heart rates and incidence of heat syncope.

A recent paper by Kraning and Gonzalez (1991) reported that intermittent (as opposed to continuous) work, increased the thermal strain and reduced the endurance time in unacclimatised male individuals of unknown physical training status in a warm/humid environment. It was concluded that intermittent work patterns with postural changes, cause shifts in cutaneous blood and heat flow and changes in sweat evaporation rates which serve to increase thermal strain.

In summary, the majority of authors support the view that sweat rate increases with acclimatisation. The time required to gain all physiological benefits can be reduced by working in the heat as opposed to passive heat exposure. However, the intensity of exercise necessary to gain maximal overall acclimatisation varies considerably, as does the time for each individual adaptation to occur. The method used to measure acclimatisation is not always the same for the reviewed studies and may at least partially explain the diverse findings.

SUMMARY OF THE LITERATURE REVIEW

The loss of fluid from sweating results in hypohydration if the loss is not replaced. The hypohydrated worker will then incur a thermal penalty. The increase in the core body temperature will impair mental and physical performance to varying extents, depending on the degree of hypohydration. If the hypovolemia is not corrected then heat illness will ultimately result. However, even if the dehydration is not sufficient to cause heat illness, the mental and physical deficits may be sufficient to compromise the individual's, and their workmates', safety in the workplace. The euhydration of workers when exposed to conditions of heat stress has become one of the most important health and safety issues we presently have in Western Australia. Many thousands of workers in the North West of the state live and work in very harsh hot environments where facilities are often limited. Unfortunately, poor guidelines and instructions are provided for fluid replacement in the workplace. In addition, workers arriving from cooler climates are not given the opportunity to acclimatise before exposure to extreme heat, rendering them prone to heat disorders. One of the reasons for this situation is that little research has been conducted to better understand the incidence of heat illness in occupations located in hot environments. On the other hand, the importance of fluid balance and acclimatisation to optimise physical and mental performance in sport is evident by the number of papers published and directed towards this small elite population.