

Challenges to Temperature Regulation When Working in Hot Environments

Nigel A.S. TAYLOR

Human Performance Laboratories, School of Health Sciences, University of Wollongong,
Wollongong, NSW 2522, Australia

Received November 29, 2005 and accepted April 10, 2006

Abstract: The focus of this review is upon acute exposure to hot environments and the accompanying physiological changes. The target audience includes physiologists, physicians and occupational health and safety practitioners. Using the principles of thermodynamics, the avenues for human heat exchange are explored, leading to an evaluation of some methods used to assess thermally-stressful environments. In particular, there is a critique of the wet-bulb globe temperature (WBGT) index, and an overview of an alternative means by which such assessments may be undertaken (the heat stress index). These principles and methods are combined to illustrate how one may evaluate the risk of heat illness. Three general areas of research are briefly reviewed: the physiological impact of wearing thermal protective clothing, heat adaptation (acclimation) and whole-body pre-cooling. These topics are considered as potential pre-exposure techniques that may be used to reduce the threat of hyperthermia, or to enhance work performance in the heat.

Key words: Clothing, Fire, Heat adaptation, Hyperthermia, Pre-cooling, Thermoregulation

Introduction

Humans can tolerate a vast range of thermal environments using both physiological and behavioural strategies. However, from a clinical perspective, body-core temperature (T_c) must be held within a very narrow range, and it is normally regulated at approximately $36.7 \pm 0.3^\circ\text{C}^{(38)}$. If T_c varies by more than 2°C either side of 37°C , then one can assume that thermal balance has been lost, or thermoregulatory failure has occurred. In this state, the regulation of body temperature has been transiently compromised, resulting in either hypothermia ($<35^\circ\text{C}$) or hyperthermia ($>39^\circ\text{C}$), with the possibility of death accompanying a T_c reduction of about 10°C , or an elevation of only $5^\circ\text{C}^{(59, 63)}$. Nevertheless, humans are extremely resilient, and the T_c extremes of human survival, following accidental hypothermia and hyperthermia, are $14.4^\circ\text{C}^{(23, 45)}$ and $47^\circ\text{C}^{(58)}$. In this paper, the focus is upon acute and chronic physiological changes (strain) accompanying

human exposure to hot environments (stress), and the factors that modify these responses. Figure 1 provides a generalised perspective of the interaction of these stress and strain phenomena.

An Overview of Thermodynamics

The total amount of energy within a closed system remains constant (*first Law of Thermodynamics*). However, this energy may be converted from one form to another. When working or exercising, humans convert stored chemical potential energy (carbohydrates and lipids) into kinetic and thermal energy (heat¹). Since we are only about 20% efficient, approximately 80% of this converted chemical energy will not contribute to useful external work, but will appear in the body as heat. Consequently, a 70-kg person performing 200 Watts (W) of external work (*e.g.* cycling, running, working) would consume about 2.5 l of oxygen each minute, and would experience metabolic energy conversion at the rate of approximately $1,000 \text{ J.s}^{-1}$, with nearly 800 J.s^{-1} being converted into thermal energy. Unless all of this heat can

¹The temperature of an object quantifies the average kinetic energy of the molecules in that object.

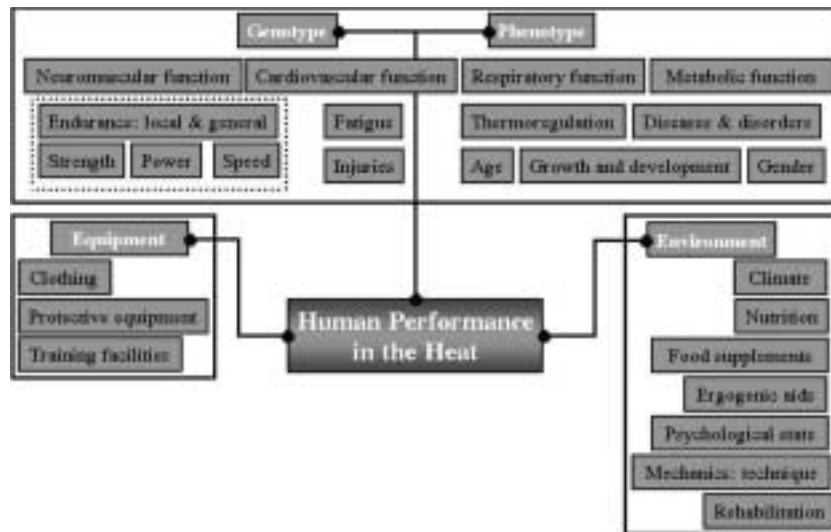


Fig. 1. A perspective on the interaction of physical and physiological phenomena on human performance in the heat.

The characteristics within the dashed box are the relevant components of physical fitness.

be dissipated to a cooler environment, then heat storage at the rate of 800 J.s^{-1} will cause the average tissue temperature of the body to rise approximately 1°C in about 5 min. While such a rapid rise can occur in some states, such a change in body temperature is not generally observed. For instance, if a person is immersed in temperate water, heat loss can easily keep up with internal (endogenous) heat production. However, when faced with this internal (endogenous) heat load in hot water (*e.g.* 40°C ; the adiabatic state), thermal homeostasis will be rapidly compromised. This paper focusses upon the problems of regulating body temperature when work or exercise are performed under conditions where air and skin temperatures are equivalent.

The thermodynamics of this problem are dictated by the avenues through which thermal energy is exchanged between the body and its physical environment, and also by the interactions of the thermal environment and physiological adaptation of these heat-loss avenues. The principal avenues for heat flux are illustrated in the heat balance equation.

$$S = M - (\pm W) \pm E \pm R \pm C \pm K \quad [\text{W.m}^{-2}] \dots\dots\dots \text{Equation 1}$$

where:

S = heat storage (+ for storage; – for loss) $[\text{W.m}^{-2}]$

M = endogenous heat production (metabolism) $[\text{W.m}^{-2}]$

W = work leaving (+) or entering (–) the system $[\text{W.m}^{-2}]$

E = heat exchange via evaporation (–) $[\text{W.m}^{-2}.\text{kPa}^{-1}]$

: heat can also be gained via the condensation of water vapour on the skin (*e.g.* steam bath)

R = heat exchange via radiation (– for loss; + for gain)

$[\text{W.m}^{-2}]$

C = heat exchange via convection (– for loss; + for gain)

$[\text{W.m}^{-2}]$

K = heat exchange via conduction (– for loss; + for gain)

$[\text{W.m}^{-2}]$

During work or exercise in the heat, the avenues for non-evaporative (dry) heat dissipation are impeded (R , C , K), and can even be reversed, leading to heat influx. For instance, under a full solar load, the body experiences radiative heat gains from the sun and the nearby hot surfaces. Similarly, natural convective losses cease when air temperature approximates skin temperature ($31\text{--}33^\circ\text{C}$). Under these conditions, the body becomes heavily, if not totally, reliant upon evaporative cooling for heat dissipation. The capacity of the body to continue its rate of endogenous heat production, without sustaining a progressive elevation in tissue temperature, is now dictated by the compensability of the thermal environment.

Thermal compensability defines the interaction of the body and the environment, such that it defines the conditions under which the body is most likely to enter a state of dysthermia. For example, in hot environments, where the primary avenue for heat dissipation is the evaporation of sweat, then thermal compensability is dictated by the ratio of the required evaporative heat loss (E_{req}) to the maximal evaporative cooling that the environment, including clothing, will permit (E_{max}). If E_{req} is greater than E_{max} , then the environmental conditions are uncompensable.

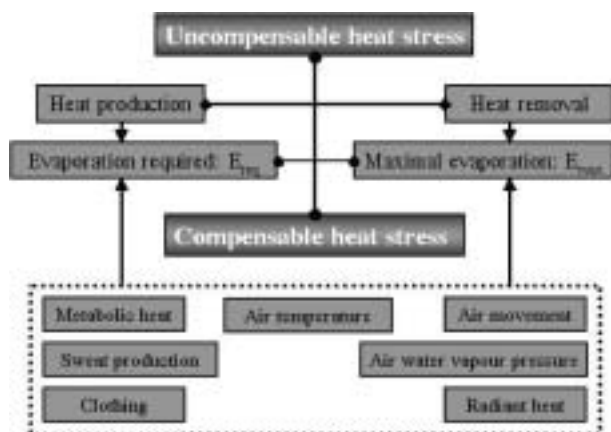


Fig. 2. Thermal compensability in hot environments: the relationship between the required evaporative heat loss (E_{req}) and maximal evaporative cooling the environment will permit (E_{max}). The factors enclosed in the lower box act upon both E_{req} and E_{max} to modulate thermal compensability.

This ratio was first suggested by Belding and Hatch⁹) for use as a Heat Stress Index (HSI) to relate thermal stress to physiological strain, and is illustrated in Fig. 2, with the derivations of the two variables being summarised in equations 2 and 3.

$$E_{\text{req}} = H - E_{\text{resp}} \pm R \pm C \quad [\text{W}] \dots\dots\dots \text{Equation 2}$$

where:

E_{req} = required evaporative cooling [W]

H = metabolic energy transformation, or the nett result of resting and exercising metabolism, and external work ($M - (\pm W)$) [W]

E_{resp} = evaporation accompanying ventilation [W]

$R \pm C$ = heat exchanges via radiation and convection [W]

$$E_{\text{max}} = 6.45 \cdot A_D \cdot i_m / I_{\text{TOT}} \cdot 2.2 \cdot (P_{\text{sk}} - (RH_a \cdot P_a)) \quad [\text{W}] \dots\dots\dots \text{Equation 3}$$

where:

E_{max} = maximal attainable evaporative cooling for a given environment and clothing configuration [W]

A_D = body surface area (Du Bois equation) [m^2]

i_m = moisture permeability index (0.45 if unknown) [dimensionless]

I_{TOT} = total insulation², including the trapped boundary layer air and clothing insulation [$\text{m}^2 \cdot \text{K} \cdot \text{W}^{-1}$]

RH_a = relative humidity of the air [%]

P_a = water vapour pressure of the air [kPa]

P_{sk} = water vapour pressure at the skin surface [kPa]
6.45 and 2.2 = constants

The above analyses provide us with a first-principles means by which to evaluate the potential for thermal environments to induce physiological strain. However, there are a variety of less complex methods routinely used to assess the risk of dysthermia within the workplace. These techniques may be categorised within either of two classes of thermal indices: effective temperature (sensation) scales and rational scales¹⁰. The next section provides a generalised critique of some of these methods.

Identifying Potentially Hazardous Conditions

The hazards of excessive and repeated heat exposures are well established²⁸), but our ability to provided a universally-valid means through which to assess the risk of hyperthermia has proven to be elusive. This problem was perhaps first identified more than 30 yr ago¹⁰), it still exists today²⁵), and it is due to the intricate interactions of a wide variety of physical and physiological phenomena that determine the probability of hyperthermia^{10,26}). As with many pathological states, these phenomena may be classified as agents of, or host factors associated with hyperthermia²⁶), and are summarised in Fig. 3.

An early thermal stress index was developed Houghten and Yagloglou³⁴): the effective temperature. The critical feature of this scale was that it aimed at defining thermal comfort limits for people within air-conditioned spaces, by identifying combinations of dry-bulb temperature, air motion and relative humidity that would elicit equivalent thermal comfort. If one assumes that thermal discomfort initiates behavioural responses^{15, 31}), and that the signals driving autonomic thermoregulation may be of a different origin^{14, 31, 39, 64}), then the link between the effective temperature scale and assessing physiological risk is perhaps insubstantial. Consider also that these experiments were performed with subjects wearing standard office clothing, and that the resultant scale was designed for use in environments close to the thermal comfort zone. Thus, extrapolation to thermally-stressful environments is also tenuous, particularly when heavy physical work is to be performed, or when people are wearing protective clothing.

Nevertheless, a wide variety of effective indices have arisen directly from this scale and, due to their simplicity, these are the most widely used thermal indices¹⁰). Of these, the most frequently used index for industrial, military and sporting applications is the wet-bulb globe temperature index (WBGT), developed by Yagloglou³ and Minard⁸²) to reduce

²1 clo = 0.155 $\text{m}^2 \cdot \text{K} \cdot \text{W}^{-1}$

³A co-author of the effective temperature scale (Houghten and Yagloglou (1923)³⁴).

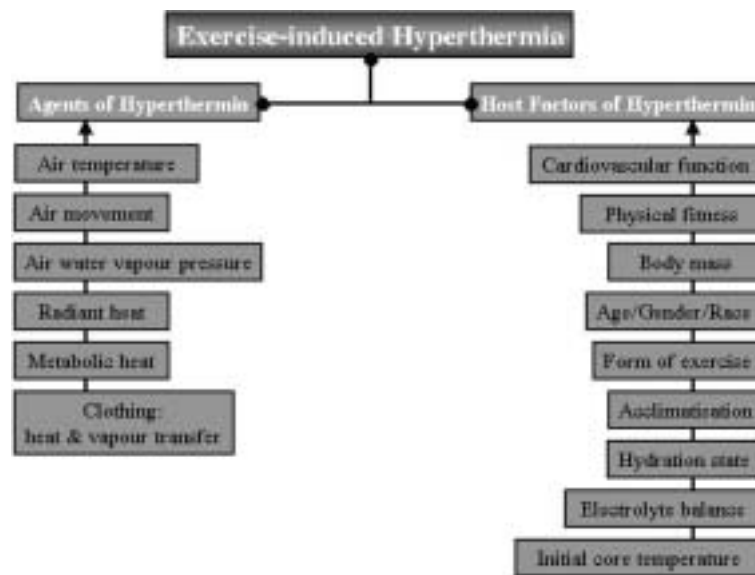


Fig. 3. Hyperthermia during work: the interaction of agents and host factors that modify the risk of hyperthermia (after Goldman, 2001)²⁶.

The agents are largely dictated by the conditions of work, while the host factors are generally independent of the working environment, varying among individuals, and are most easily modified to reduce the risk of hyperthermia.

the incidence of heat illness during military training. Indeed, general use of the WBGT-index was recommended by the Occupational Safety and Health Administration⁵⁴), and subsequently adopted by the International Standards Organisation for quantifying thermal stress³⁷), the National Institute for Occupational Safety and Health⁵²), and the American College of Sports Medicine²).

$$\text{WBGT (outdoors)} = 0.7 T_{\text{nwb}} + 0.2 T_g + 0.1 T_a \text{ [}^\circ\text{C]} \quad \text{.....Equation 4}$$

$$\text{WBGT (indoors)} = 0.7 T_{\text{nwb}} + 0.3 T_g \text{ [}^\circ\text{C]} \quad \text{.....Equation 5}$$

$$\text{WBGT (indoors: Oxford)} = 0.85 T_{\text{nwb}} + 0.15 T_g \text{ [}^\circ\text{C]} \quad \text{.....Equation 6}$$

where:

T_{nwb} = natural wet bulb temperature⁴ [°C]

T_g = black globe temperature [°C]

T_a = air temperature [°C]

A number of researchers have evaluated the physiological efficacy of using the WBGT-index^{6, 46, 49, 60, 61}), with the most eloquent studies coming from Wenzel's group^{35, 75, 76}).

Notwithstanding the almost ubiquitous adoption of the WBGT-index, these studies have identified several significant

limitations of this method. First, the index tends to over-emphasise the effects of dry bulb temperature towards the top end of the scale¹⁰). Second, it does not adequately consider air velocity under hot-humid conditions¹⁰), and is insensitive to this affect once air velocity exceeds 1.5 m.s^{-1} ⁶), yet this can have a significant impact upon heat dissipation. Third, it lacks the capacity to accommodate different rates of metabolic heat production^{10, 75, 76}), or variations in skin temperature or skin wettedness⁶). Since hyperthermia can be induced simply by exercise-induced heat production, then metabolic heat production is a critical consideration. Furthermore, Lind^{42, 43}) and Wenzel⁷⁵) demonstrated that the physiological influence of air humidity, at a fixed air temperature, was elevated when metabolic rate was increased. Fourth, Ilmarinen³⁵) and Wenzel *et al.*⁷⁶) both found that body mass loss (gross sweating) was not independent of climatic conditions, and it invariably diverged from the changes in T_c and cardiac frequency (heart rate). That is, physiological responses varied within and among climatic conditions, such that conditions that elicited equivalent mass losses did not simultaneously evoke predictable changes in T_c or cardiac frequency. Fifth, the usefulness of the WBGT-index for clothed workers has been found to range from inferior⁴⁶) to wholly inappropriate when encapsulating ensembles are used²⁵).

One can generally attribute these limitations to the fact

⁴The wick is cooled by natural convection, thus using the appropriate air movement rather than forced convection.

the WBGT-index is not a rational scale. That is, it is not based upon heat balance, and the thermodynamics of these heat exchanges, but solely upon quantifying the thermal environment; its greatest strength (simplicity) has thus become its greatest limitation. Consequently, investigators have found that different combinations of air temperature, globe temperature and humidity can result in an identical WBGT, but with markedly different physiological strain^{35, 75, 76}. In general, while one can reliably assume that conditions with a WBGT of 25°C will be less stressful than those with a WBGT of 35°C, we can also generally expect that physiological strain will be greater for hot-dry than for hot-humid conditions, even when both states have an equivalent WBGT⁷⁵.

It is therefore the opinion of the current author that rational heat indices provide a superior means by which to identify potentially hazardous environments. Such scales attempt to integrate the quantification of heat exchange with the resultant physiological strain. The first rational index (operative temperature) was that of Winslow *et al.*⁷⁷, with Belding and Hatch⁹ subsequently developing the Heat Strain Index ($E_{\text{req}}/E_{\text{max}}$ ratio), from which several further modifications have arisen. While these indices also have limitations⁵, the principles upon which they are based are both sound and balanced.

The inter-relationships presented in Fig. 2, and equations 2 and 3, illustrate both the importance of work rate in determining the thermal compensability of a given set of working conditions, and how readily both E_{req} and E_{max} may be affected by changes within the thermal environment. Let us now take our example of an exercising person (70 kg, 170 cm tall) several steps further, using the following environmental conditions: temperate (25°C, 75% relative humidity), hot-dry (35°C, 40% relative humidity) and hot-humid (35°C, 75% relative humidity). Table 1 contains a summary of the predicted thermal exchange requirements for this person, for each of these environmental states. From these analyses, three key points emerge. First, for each of the environments, the combination of metabolic rate and thermal load dictate that E_{req} is always greater than E_{max} ; they are deemed to be uncompensable conditions. Second,

Table 1. Predicted heat balance during work in three thermal environments: temperate (25°C, 75% relative humidity), hot-dry (35°C, 40% relative humidity) and hot-humid (35°C, 75% relative humidity).

Variables	Temperate	Hot-dry	Hot-humid
Water vapour pressure: air (kPa)	2.38	1.27	4.22
Water vapour pressure gradient (kPa)	3.25	3.37	1.41
Radiative + convective heat loss (W)	42.1	0	0
Required evaporative cooling (E_{req} : W)	727.9	770	770
Maximal evaporative cooling (E_{max} : W)	462.5	480.5	200.2
Ratio: $E_{\text{req}}/E_{\text{max}}$ (%)	157	160	385

Constants: total heat production (905 W), wind velocity (2.78 m.s⁻¹), initial core (37°C), skin (35°C) and mean body temperatures (36.3°C), skin water vapour pressure (5.62 kPa), total insulation (0.47 m².K.W⁻¹ (3 clo)), clothing moisture permeability index (0.4), uncovered skin surface area (30% or 0.54 m²), respiratory evaporative heat loss (135 W).

under the temperate and hot-dry conditions, it is the metabolic rate that drives E_{req} beyond the capacity of the environment to facilitate the evaporation of sweat. Third, it is the elevation in the water vapour pressure⁶ of the hot-humid state that results in a greater than two-fold elevation in the $E_{\text{req}}/E_{\text{max}}$ ratio, for a fixed metabolic heat production.

The Risk of Exertional Heat Illness

The risk of exertional heat illness⁷ is heightened with increments in T_{c} ⁸¹. While the World Health Organisation has recommended an upper limit for T_{c} in workers (38°C, thus implicitly limiting metabolic heat production to 325 watt or less⁷⁸), other criteria have also been suggested. For example, work:rest ratios and the cessation of work have been recommended on the basis of cardiac frequency limits¹⁰, the convergence of skin and core temperatures⁵⁵, and several heat stress indices^{10, 43}. However, from an occupational health perspective, the primary reason for evaluating the thermal environment is to determine the maximal likely physiological responses that may be elicited by those conditions. Thus, it becomes necessary to interpret these data with respect to the probability of adverse health outcomes. This is a difficult topic due to the lack of empirical evidence, and is therefore beyond the scope of this paper. Nevertheless, these interpretations will be propelled by the opposing needs to maintain worker health and industrial productivity. In the military and sporting situations, interpretation can become very skewed, with the outcome frequently left to the discretion of the individual, often with health being compromised.

To illustrate the process of evaluating the risk of exertional heat illness, a sporting example has been selected: the summer

⁵Limitations: (i) assumes skin temperature is 35°C regardless of metabolic rate; and (ii) assumes that all derived values that are equivalent will have the same physiological impact, regardless of whether the $E_{\text{req}}/E_{\text{max}}$ ratio is 50/100 or 300/600 (Belding, 1970)¹⁰.

⁶Since it is the water vapour pressure gradient between the skin surface and the air that dictates the evaporation of sweat, then it is more appropriate to use this term than to refer to relative humidity.

⁷Probability of heat stroke in humid environments: 38.2°C 1:500 chance; 38.0°C 1:1,000 chance; 37.8°C 1:10,000 chance; 37.6°C 1:500,000 chance.

Games of the 28th Olympiad (Athens). On the basis of the WBGT-index, and the recommendations of the American College of Sports Medicine²⁾, the risk of heat illness for the summer Olympic Games over the past 20 yr has been high to very high on four occasions, with only Seoul (moderate) and Sydney (low) conducted under favourable environmental conditions. From analyses of daily air temperature and relative humidity data for August, collected over six years, one could obtain a reasonable prediction of the worst-case conditions prior to the Athens Games.

Using these data, it was predicted that the mean maximal air temperature would be approximately 35°C and relative humidity would approach 55%. Such conditions would result in an ambient water vapour pressure of approximately 2.8 kPa. In addition, one could expect >13 h of sunlight, and sea-water temperatures of approximately 26°C.

Using the principles defined in equations 1–3, the heat balance components, as well as E_{req} and E_{max} were computed for rest and heavy endurance exercise (marathon running). The data derived from this modelling are summarised in Fig. 4, which also includes corresponding calculations for the Sydney Games. A typical elite marathoner would generate more than 1,570 W of heat. In Athens, respiratory evaporation would be expected to account for about 250 W of heat removal, while convective cooling would dissipate <20 W, and this would be facilitated only because the athlete was running (5.47 m.s⁻¹). The E_{req} would therefore need to be 1,300 W, and this would require a sustained sweat rate of 1.9 l.h⁻¹. Such a steady-state sweat rate would be almost impossible in an unacclimatised person.

At the Sydney Olympics, the ambient water vapour pressure was approximately 1.5 kPa, with a projected skin-air vapour pressure gradient of 3.0 kPa. In Athens, at rest, and assuming an elevation in cutaneous water vapour pressure with air temperature, this gradient could be reduced to about 2.2 kPa. This means that the evaporative power of the air would be reduced by approximately 28%. Thus, the E_{req} during marathon running in Athens, which is equivalent to that derived for Sydney since it is heavily dependent upon metabolic heat production, will now be greater than three times E_{max} , and this is due simply to the change in the environmental conditions (Fig. 4).

This combination of these environmental circumstances conspired to make the Games of the 28th Olympiad among the most physiologically stressful encountered over the past 20 yr. Under such conditions, the cumulative effects of metabolic and environmental thermal loads represented uncompensable heat stress, predisposing athletes to hyperthermia. Under these conditions, exercise- and heat-

induced augmentation of sweat gland function alone would not provide complete athlete preparation, forcing one to seek other strategies to facilitate performance optimisation.

The Physiological Impact of Thermal Protective Clothing

We shall now turn our attention to a very specific thermal and applied problem, where personal (thermal) protective ensembles are worn to minimise the radiant, convective and conductive heat loading typically encountered within environmental extremes, and during emergency operations. The example chosen to illustrate this topic will be the firefighter (readers with a specific interest in total encapsulation are directed to Goldman²⁵⁾). Since the personal protective ensemble of the firefighter is designed with impermeable and semi-permeable fabric layers that trap air, they create a microclimate between the skin and the clothing. This, in combination with the mass of these ensembles and other protective equipment, places a significant physiological burden upon the wearer, both in the form of increased metabolic rate and reduced heat dissipation⁵³⁾. Certainly, such ensembles minimise the penetration of external heat, but they also reduce the escape of metabolically-produced heat. Our focus in this section is upon the latter problem, which is frequently inadequately addressed by either manufacturers or purchasing officers.

Relative to the surrounding air, the trapped (microclimate) air is warmer, it contains more water vapour, and its movement across the skin surface is limited. Thus, clothing will markedly affect heat and water vapour transfer. Knowing this, it becomes critical to understand two physical properties of clothing. The first factor is the thermal insulation provided by the ensemble. This relates to the characteristics of the garment that allow it to trap a layer of air, which is an extremely good insulator. The thicker the garment, the more air that is trapped, and the greater is its insulating capacity. For instance, the personal protective ensembles of firefighters have a total insulation of about 0.47 m².K.W⁻¹ (3 clo). Second, the vapour (moisture) permeability of the garment is important. This is the ability of the fabric to allow water vapour to pass through, and so facilitate evaporation at the skin surface. Whilst thermal insulation and moisture permeability are individually very important factors, it is their ratio that dictates the performance of a garment within any thermal environment: permeability index / clothing insulation²⁵⁾.

Using first-principles thermodynamics, this scenario was also modelled for a person wearing a personal protective

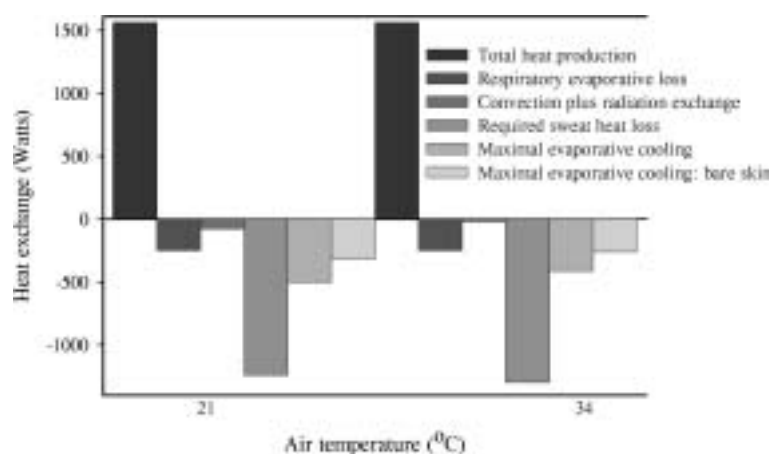


Fig. 4. Predicted thermal exchanges during marathon running: a comparison of heat exchanges during the summer Olympics of Sydney (2000: 21°C) and Athens (2004: 34°C).

ensemble with an inherent insulation of $0.47 \text{ m}^2 \cdot \text{K} \cdot \text{W}^{-1}$ (Fig. 5). The resultant data are shown as a three-dimensional surface (total heat exchange) for combinations of external work (rest to 300 Watts) and air temperature (15–50°C). The zero total heat exchange axis is the reference plane, where heat gain equals heat loss. It can readily be seen that, within most conditions encountered by firefighters, heat will be stored.

We shall now explore the physiological consequence of such heat storage, using data collected from both laboratory and field trials to provide the reader with both useful data, and some insight into a specific and applied thermal problem^{3, 20, 73}. These studies involved 35 combinations of environmental conditions and clothing ensembles, and were conducted using 21 subjects.

First, we evaluated the thermal protective properties of firefighters' ensembles under realistic (field) conditions including tower-climbing, a Hot Fire Cell exposure, and a Flashover simulation⁷³. We observed an average T_c of 38.2°C, with the greatest rate of increase being $1.7^\circ\text{C} \cdot \text{h}^{-1}$. During one flashover simulation, the temperature of air trapped under the outer flame protective cover ranged from 31.7°C to 145.1°C, averaging 63.7°C over 34 min. The highest upper-body clothing and local skin temperatures were: 91.1°C (inner tunic), 59.4°C (outer shirt), 53.5°C (inner shirt) and 41.7°C (chest). The inner tunic to chest thermal gradient (49.4°C) can be almost entirely attributed to the volume of trapped air between these sites. The corresponding lower-body temperatures were: 102.7°C (outer pant), 86.9°C (inner trouser) and 46.7°C (leg). Skin temperatures between 39–41°C are at the threshold for transient pain, the skin burn

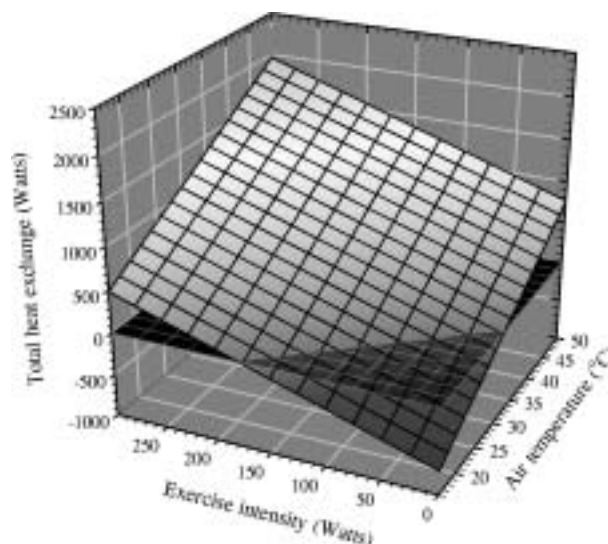


Fig. 5. The heat exchange surface for combinations of external work and air temperature.

The zero heat exchange axis is the reference plane, where heat gain equals heat loss. Points located above this axis indicate heat storage.

threshold is between 41–43°C, and local skin temperatures $>45^\circ\text{C}$ are invariably accompanied by local tissue damage. Thus, a second-degree burn would be anticipated from a contact exposure of $>50^\circ\text{C}$ for $>4 \text{ min}^{50}$. Accordingly, despite the use of personal protective ensembles, skin temperatures during these flashover simulations approached, and exceeded levels associated with skin burns. Indeed, each of the subjects in one series of flashover simulations experienced minor skin burns at sites where the breathing apparatus harness compressed air trapped within the tunic, reducing local

insulation.

While such case-study experiments are limited in their ability to contribute to clothing evaluation, they do provide a means by which a more realistic assessment of heat penetration may be undertaken. Such data provide insight into the magnitude of the thermal load transmitted through personal protective clothing, and the thermal micro-environment within each ensemble, during extreme and more realistic heat exposures.

In the second project³⁾, we evaluated the physiological impact of wearing a fire helmet (laboratory trials). Helmet testing is regulated by various national and international standards that primarily deal with helmet design and construction issues, but do not specifically address the physiological impact of the helmet. In some instances, the end product can represent a compromise between the competing needs to control heat flow into, and out of the helmet. However, the fire helmet must represent the culmination of work designed to provide the firefighter with both a safe and functional item of personal protective equipment. We therefore studied thermal strain in subjects wearing a standard thermal protective ensemble, whilst also wearing each of six different fire helmets. Estimates of a helmet's capacity to insulate the skin from an external heat source, in conjunction with trapped air, were obtained during manikin trials, where helmets were exposed to external heat sources. These tests resulted in external helmet temperatures peaking at 141.5°C. Records of inner helmet surface temperatures revealed marked deviations among the helmets, with variations of nearly 30°C between the coolest and hottest helmets.

The major difference among the helmets during laboratory trials, where helmets were evaluated in exercising subjects, was that, on all measures except for thermal discomfort, one helmet was universally associated with the least physiological (cardiovascular load, core temperature, mass change) and psychophysiological strain (effort sense). This helmet also allowed for significant heat penetration. Thus, the compromise between preventing the influx of external heat and facilitating heat to escape through its solid components, has resulted in a physiologically superior helmet.

The final project focussed upon the cardiovascular consequences of wearing thermal protective clothing²⁰⁾. For this project, seven males completed two trials of semi-recumbent, intermittent cycling (39.6°C, 45% relative humidity) wearing either thermal protective clothing or shorts (control trial). Core and skin temperatures, cardiac frequency, stroke volume, cardiac output, arterial pressure, forearm

blood flow, plasma volume changes and local sweat rates were measured. We hypothesised that, during mild exercise-induced hyperthermia in uncompensable heat, an elevation in skin blood flow would not act to facilitate heat dissipation when a thermal protective ensemble was worn, but instead would precipitate significantly greater cardiovascular strain. That is, when the heat loading is high enough, or the exposure duration long enough, cardiovascular function would be compromised, and no longer able to serve both skin and muscle blood flow demands⁴⁰⁾. In this situation, blood pressure would decline, and cutaneous vasoconstriction would ensue.

In the clothed trials, subjects experienced significantly shorter times to volitional fatigue (52.5 versus 58.9 min), which occurred at lower peak work rates (204.3 versus 277.4 W), and with higher core (37.9 versus 37.5°C) and mean skin temperatures (37.3 versus 36.9°C). There was a significant interaction between time and clothing for cardiac frequency, such that, over time, the clothing effect became more powerful. Clothing had a significant main effect on cardiac output, but not stroke volume, indicating the higher cardiac output was driven by changes in cardiac frequency. Despite a greater sweat production when clothed (923.0 versus 547.1 g.m⁻².h⁻¹; $P < 0.05$), forearm blood flow and plasma volume changes remained equivalent between the two trials.

Thus, while thermal protective clothing reduced exercise tolerance, and increased both thermal and cardiovascular strain, there was no apparent affect on the exercise-, posture- or temperature-specific cardiovascular responses observed at the point of volitional fatigue. Furthermore, neither cardiac output nor forearm blood flow were compromised during the clothing trial. In fact, these were both equivalently elevated. We had predicted that clothing would elevate cardiovascular strain to the point where blood pressure regulation would have been compromised, resulting in a reduced skin blood flow. The fact that neither of these changes occurred indicates that these variables did not limit either sub-maximal or maximal exercise in the clothed state, at least under the current experimental conditions. It was therefore concluded that, during moderately-heavy exercise under hot-dry conditions, the strain on the unclothed body was already high, such that the additional stress imparted by the clothing ensemble represented a negligible, further impact upon cardiovascular stability.

While the precise mechanisms that lead to fatigue-related decreases in human performance, and the premature termination of exercise are debated, we do know that T_c is intimately linked with these outcomes during extended-duration exercise and work. Indeed, we know that increases

in T_{e} are directly dependent upon increments in exercise intensity, air temperature, ambient water vapour pressure, progressive dehydration and the use of clothing. Conversely, it is well established that heat adaptation and whole-body pre-cooling serve as potential pre-exposure protective mechanisms to either reduce the threat of hyperthermia, or to enhance physical performance in the heat. We shall now turn our attention to each of these topics.

Heat Adaptation

During prolonged exercise or work in the heat, human thermal homeostasis is first challenged, and may eventually be lost, as one moves from a compensable state through to uncompensable heat stress. This transition is dictated by the combined effects of air temperature, water vapour pressure, exercise intensity, clothing (and its permeability to water vapour), body composition, hydration status, long-term endurance fitness, and the state of heat adaptation¹⁷⁾. During the first week of an unaccustomed heat exposure, work performance is most affected, and the threat of heat illness is greatest. As we have seen above, when air temperature approaches skin temperature, and when solar loads are high, the possibility for dry heat loss is negated, forcing an almost total reliance upon evaporative cooling at the skin surface. Given a sufficiently long exposure time, the body will undergo a three-phase adaptation to better tolerate such conditions. From a physiological strain perspective, such adaptations are equivalent to lowering the air temperature²⁶⁾. The most common means through which heat tolerance is improved is via heat adaptation, and its associated elevation in sweat secretion.

Natural acclimatisation is universally recognised to be the most effective means by which to increase heat tolerance^{19, 72)}, particularly within climates that experience wide seasonal swings in air temperature^{36, 68, 71)}. However, its practical limitations minimise its use for many occupational and sporting groups, resulting in the development of a wide range of heat adaptation (acclimation) techniques. We can group these methods within three general categories^{70, 72)}.

- (i) *Passive heat acclimation*: External heat is applied to the resting body to elevate and hold a thermal load necessary to induce adaptation (e.g. water baths, saunas and climate chambers).
- (ii) *Exercise-induced heat adaptation*: Endurance exercise elevates muscle and deep tissue temperatures and, if this thermal load is applied regularly, heat adaptation will ensue⁸⁾. This method may be used in

three different ways, each of which can modify the resultant thermal strain:

- *Exercise under cool and temperate states*: However, exercise in the absence of a T_{e} elevation will not elicit heat adaptation³³⁾.
- *Exercise with a significant solar load*.
- *Exercise with sweat clothing*: There is little empirical evidence to indicate that this procedure is any more beneficial than endurance training.

- (iii) *Combined exercise and heat stress acclimation*: Conventional heat acclimation regimens involve moderate-to-heavy intensity exercise (e.g. walking, running, cycling, bench stepping) within a temperature- and humidity-controlled chamber. Such methods may be grouped into three general categories, that differ according to how the exercise forcing function is applied:

- *Constant work-rate methods*: This is the most common heat-acclimation method, and is the typical model used when acclimating people for tasks that are performed at fixed work rates.
- *Self-regulated exercise methods*: The person dictates the work rate during heat exposure.
- *Controlled-hyperthermia (isothermal) methods*: The work rate is adjusted to maintain a constant thermal strain, so the work rate is progressively elevated as acclimation progresses²²⁾.

From the vast body of research evidence on human heat adaptation, the following generalisations appear to be justified. First, passive acclimation is not as effective as methods incorporating exercise stress^{68, 79)}. Second, exercise-induced heat adaptation without a significant heat load is inferior to the more traditional heat acclimation methods^{62, 69)}, it is an inadequate substitute for heat acclimation^{5, 24, 44)}, and the elevation of cutaneous tissue temperatures appears to be a necessary stimulus for more complete heat adaptation⁶²⁾. Nevertheless, exercise under temperate conditions can induce heat adaptation, but this depends upon the capacity to elevate and hold body temperatures for an extended time, and it appears to provide thermal protection for only relatively short-term heat exposures⁷⁹⁾. Third, humid-heat acclimation produces a greater sweating adaptation than does dry-heat acclimation^{30, 69)}, while adaptation to repeated dry exposures do not provide optimal protection for subsequent humid-heat stress⁴⁾. Fourth, the type of exercise forcing function used during exercise-heat acclimation will dictate the nature

of the adaptation produced⁷²⁾. It appears that the controlled-hyperthermia model will induce a more complete and sustained heat adaptation than either the constant or the self-regulated work rate techniques^{56, 57, 70)}.

Notwithstanding differences in heat adaptation methods, one typically observes an enhancement of eccrine sweat gland (sudomotor) function^{1, 18, 41, 51, 56, 65)}, which serves to boost our most effective means of heat dissipation within hot environments. Specifically, there is an increased steady state-sweat rate (two-fold elevation), heightened sweat gland sensitivity to increments in T_{e} , a reduced temperature threshold for sweating onset, more effective reabsorption of sodium and chloride within the sweat duct, and better conservation of the extracellular electrolyte content and fluid volume.

The substrate for this elevated sweat secretion is provided via an expansion of the plasma volume, which occurs in association with an elevated, and a superior maintenance of the osmotic potential of the blood^{29, 67)}. An expanded plasma volume is better able to withstand fluid loss via sweating. Thus, when working at a given intensity, stroke volume is larger and cardiac frequency lower following heat adaptation^{16, 48)}, permitting superior regulation of blood pressure, an elevation in skin blood flow, and a lowering of the vasodilatory threshold²²⁾.

These physiological adaptations facilitate a more rapid transfer of heat from the body core to the periphery for dissipation. People report being less stressed, and are better able to tolerate work and heat stress. Thus, one can move from a working state that may be physiologically uncompensable, into a state in which compensation is physiologically attainable (*i.e.* this is equivalent to lowering air temperature).

Recent work from our laboratory has challenged two long-held doctrines pertaining to human heat adaptation. First, it has generally been assumed that, as part of the sweat adaptation process, there is a redistribution of sweat secretion towards the limbs. Using the controlled-hyperthermia technique, we tested this possibility⁵⁶⁾. Eleven non-adapted males were acclimated over three weeks (16 exposures), cycling 90 min per day, six days a week (40°C, 60% relative humidity), in which work rate was modified to achieve and maintain a target T_{e} (38.5°C). These conditions were uncompensable. Local sudomotor adaptation (forehead, chest, scapula, forearm, thigh) and onset thresholds were studied during constant work rate, heat stress tests (39.8°C, 59.2% relative humidity) conducted on days 1, 8 and 22 of acclimation. Whole-body sweat rate increased significantly: $0.87 \pm 0.06 \text{ l.h}^{-1}$ (day 1); $1.09 \pm 0.08 \text{ l.h}^{-1}$ (day 8); and 1.16

$\pm 0.11 \text{ l.h}^{-1}$ (day 22). However, not all skin regions exhibited equivalent relative sweat rate elevations from day 1 to 22. The relative increase in forearm sweat rate ($117 \pm 31\%$) exceeded that at the forehead ($47 \pm 18\%$; $P < 0.05$) and thigh ($42 \pm 16\%$; $P < 0.05$), while the chest sweat rate elevation ($106 \pm 29\%$) also exceeded that of the thigh ($P < 0.05$). Thus, our data did not support the hypothesis of a generalised and preferential trunk-to-limb sweat redistribution following heat acclimation.

The second tenet is that the plasma volume expansion that typically accompanies heat adaptation, will subside as adaptation proceeds^{7, 80)}. Data from these early, and some more recent experiments, have been interpreted to indicate that the plasma volume expansion is restricted to the vascular compartment, and occurs only in the early adaptation phase, but is not sustained. Using methods described above, we measured intra- and extravascular body-fluid compartments in 12 resting males before (day 1; control), during (day 8) and after (day 22) a three-week, exercise-heat acclimation⁵⁷⁾. On days 8 and 22, the plasma volume expanded and was maintained relative to control values (day 1: 44.0 ± 1.8 ; day 8: 48.8 ± 1.7 ($P < 0.05$); day 22: $48.8 \pm 2.0 \text{ ml.kg}^{-1}$). The extracellular fluid compartment was equivalently expanded on both days 8 and 22. Therefore, the plasma and interstitial fluid compartments exhibited similar relative expansions on days 8 ($15.0 \pm 2.2\%$ versus $14.7 \pm 4.1\%$; $P > 0.05$) and 22 ($14.4 \pm 3.6\%$ versus $6.4 \pm 2.2\%$; $P = 0.10$). We interpreted these data to show that the acclimation-induced plasma volume expansion could be maintained following prolonged heat acclimation using small, but progressive increments in exercise intensity. In addition, this expansion was not selective, but represented a general expansion of the entire extracellular compartment.

Pre-cooling

It has been known for many years that lowering body heat content (pre-cooling) prior to heat exposure can enhance subsequent heat tolerance in resting subjects⁷⁴⁾. In addition, pre-cooling may delay the onset of fatigue by increasing time to exhaustion in temperate conditions, when working at either fixed⁶⁶⁾ or self-selected rates³²⁾. This benefit has subsequently been extended to include hot environments^{11, 13, 27)}.

Recently, we have undertaken a series of pre-cooling experiments, using the whole-body immersion method to pre-cool subjects prior to exercise in the heat^{12, 13, 47)}. Subjects sat semi-reclined, with water at the level of the axilla. The initial water temperature was 28–29°C, and was gradually reduced to 23–24°C, with immersions terminated at the first

sign of shivering. This protocol was designed to extract heat without eliciting cold-induced heat production (thermogenesis). Following pre-cooling, subjects cycled for 35 min at 60% of peak oxygen uptake (air temperature 35°C and 50% relative humidity): physiologically uncompensable. This protocol was repeated under control (thermoneutral) conditions, with immersion in warmer water (34–35°C, 45 min).

We found pre-cooling to have limited impact on muscle metabolism, with no differences observed between the two conditions for muscle glycogen, triglyceride, adenosine triphosphate, creatine phosphate, creatine or lactate concentrations at rest, or following exercise. These data indicated that improved endurance during exercise in the heat may not be attributable to altered muscle metabolism¹². A more likely possibility was that reduced thermoregulatory and cardiovascular strain were responsible for the improved endurance.

We subsequently tested the possibility that reduced tissue temperatures may explain this improved performance using three whole-body immersion treatments¹³: pre-cooling (as above), thermoneutral (control: as above) and pre-heating (39°C). Pre-cooling reduced muscle temperature by 6.3°C while pre-heating increased it 3.4°C, relative to the control trial. Despite this offset, exercise in the heat caused muscle temperature to climb exponentially towards a common asymptote within each trial, with pre-cooling offering no thermal advantage beyond about 40 min. Following pre-cooling, exercising T_c initially increased at 0.09°C.min⁻¹, being significantly faster than either the control (0.05°C.min⁻¹) or pre-heated conditions (0.03°C.min⁻¹). Pre-cooling lowered the sweat threshold and also resulted in a reduced cardiac frequency across the exercise-heat exposure. The principal observations from this project were that pre-cooling reduced thermoregulatory strain during exercise in the heat, as demonstrated by: (1) lower core and muscle temperatures, (2) augmented conductive heat loss, (3) a reduced sweat threshold, and (4) a lower exercise cardiac frequency. This last change was consistent with greater cardiovascular stability. However, pre-heating had minimal impact upon muscle temperature during exercise. Indeed, despite a muscle temperature difference of approximately 10°C at the end of immersion (pre-cooling versus pre-heating), muscle temperature steadily climbed towards a uniform final value, that appeared to be dictated by the metabolic load, and was only influenced by the pre-exercise treatments early during the exercise-heat exposure. Accordingly, our observations do not support the hypothesis that pre-cooling delays fatigue development in the heat, via

a reduction in muscle temperature during the latter phase of an exercise-heat exposure. Instead, we suggest that pre-cooling could perhaps favourably modify cardiovascular function which, in turn, may improve physical performance whilst working in the heat.

To test this hypothesis, we investigated the cardiovascular and thermoregulatory responses of whole-body pre-cooling during an identical, uncompensable exercise-heat exposure⁴⁷. Nine males participated in two, 35-min cycling (60% peak power) trials in the heat (35°C, 50% relative humidity), preceded by either pre-cooling (water immersion: as above), or rest in an air-conditioned laboratory (control). Pre-cooling significantly reduced skin and core temperatures. Forearm blood flow was attenuated following pre-cooling ($P<0.05$), with the final exercising blood flow being <50% of that observed for the control trial. Pre-cooling also delayed the vasodilatory threshold, elevating the mean body temperature threshold by 0.59°C ($P<0.05$). The stroke volume was enlarged at rest, and throughout the first 20 min of exercise (pre-cooling; $P<0.05$). Cardiac output was significantly higher during the pre-cooled trial, in which it increased immediately, and remained constant throughout exercise. These observations are consistent with superior central cardiovascular stability, possibly due to reduced competition for the available blood volume. Such a strain reduction may help account for the observation that, during uncompensable exercise in hot-humid conditions, physical performance can be improved following whole-body pre-cooling.

It is the considered view of our laboratory that the optimal means by which to pre-cool a person, prior to work in the heat, is via cool-water immersion. The water temperature should be slightly cooler than thermoneutral (28–29°C), and should be allowed to gradually fall by 4–5°C over 50–60 min. This method extracts heat without incurring a powerful metabolic (thermogenic) response.

Conclusion

While humans can tolerate a wide range of thermal environments, optimal physiological function is dependent upon the maintenance of thermal homeostasis. Both the metabolic heat production and the capacity of the thermal environment to support evaporative cooling will dictate the extent to which humans can maintain homeostasis. When either heat production or evaporation adversely affect thermal homeostasis, the operational conditions are said to be uncompensable. This is most dramatically illustrated when thermal protective garments are worn during exercise-heat exposures. However, heat adaptation and pre-cooling may

be used to extend the range of work rates and climatic conditions that define this uncompensable state, although both methods have significant practical limitations for workers. In addition, heat adaptation is not necessarily beneficial for workers wearing encapsulating garments, where elevated sweat, but not evaporation rates are associated with greater thermal discomfort. In such situations, whole-body pre-cooling and auxiliary cool may be employed to lower T_{re} or to supplement heat loss.

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