Chapter 2
Human Physiology in the Heat

Luke N. Belval and Ollie Jay

Abstract Safety and performance during exercise and physical activity in the heat are limited by the human body's physiological ability to balance heat gain and heat loss. Circumstances where heat gain from internal or external sources outweighs the ability to dissipate it can lead to dangerous increases in body temperature. Humans possess an ability to adapt to exercise in warm environments and minimize the deleterious effects through heat acclimatization. In situations where human physiology cannot overcome thermal challenges, exertional heat illnesses can manifest. These exertional heat illnesses can range from relatively benign to potentially fatal when left untreated. Technologies, techniques, and strategies to mitigate the consequences of exercise in warm environments should consider the existing physiological mechanisms to successfully promote health and maximize performance.

Introduction

From an evolutionary perspective, humans' ability to adapt to the thermal environment has allowed our species to thrive. The mechanisms that allowed for persistence hunting, the practice of hunting animals much larger and faster than humans by out-enduring them, have greatly shaped our physiology [1]. While this physiology allows us to succeed where other species may fail, thermal environments still exist that humans cannot overcome. Whether it is an internal or external limit, the human body can be restricted by the amount of heat it is able to dissipate. By understanding these limits, we can identify strategies and technologies to further human capabilities for exercising in hot environments.

L. N. Belval (✉)
Korey Stringer Institute, Department of Kinesiology, University of Connecticut, Storrs, CT, USA
e-mail: luke.belval@uconn.edu

O. Jay
University of Sydney, Lidcombe, NSW, Australia

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This chapter will cover the biophysical and physiological mechanisms the human body employs for heat gain and dissipation. This examination of heat balance is fundamental to the understanding of the thermoregulatory challenges faced by athletes, laborers, and war fighters. We will also examine how the body adapts to thermal environments over time through heat acclimatization, a powerful tool in prevention and performance enhancement. Finally, we will discuss the dysfunctions of the thermoregulatory system and their manifestations as exertional heat illnesses.

Human Heat Balance

From a fundamental perspective, critical elevations in human body temperature (hyperthermia) arise from a sustained inability to balance the amount of heat generated from internal metabolic processes with a sufficient amount of heat dissipation from the skin surface to the surrounding environment.

In its simplest form, the human heat balance equation (Eq. 2.1) states that in order to prevent the storage of heat energy inside the body (i.e., $S = 0$) of zero, the rate of metabolic heat production (H_{prod}) must be offset by an equal rate of combined heat loss (H_{loss}), which is almost exclusively derived from heat transfer pathways at the skin surface:

$$H_{prod} = H_{loss} \pm S \tag{2.1}$$

To obtain a better understanding of the various physiological and biophysical mechanisms that lead to hyperthermia, H_{prod} and H_{loss} can be broken down into the following principal components.

Metabolic Heat Production

By definition, H_{prod} is the difference between metabolic energy expenditure (M) and the amount of this energy that is converted into mechanical work (W). As a rule, humans are very inefficient at this conversion, typically resulting in a high ratio of M to W (>3:1). Cycling is the most mechanically efficient activity with 30% of M used to create W [2] and the remaining ~70% liberated as heat energy inside the body that must subsequently be transferred to the skin surface and dissipated to prevent an increase in S. At the other end of the efficiency spectrum is running on flat ground, which creates approximately zero net external work as the propulsion and breaking forces of gait yield equivalent positive and negative work [3]. As such, all metabolic energy during running on a flat surface is released as heat (i.e., $H_{prod} = M$). The elevation in M during exercise is mainly determined by the rate at which oxygen is consumed (VO_2) with every 1 L of VO_2 per minute yielding

approximately 21 kJ of energy per minute. It follows that activities requiring a greater VO_2 result in a higher H_{prod}. For example, military tasks such as intermittent marching for 3 h with standard combat gear and weapon (total load, ~25 kg) and digging soft sandy ground to a depth of 1 m at a self-regulated pace result in a VO_2 of 1.7–1.8 L·min^{-1} [4], which is equivalent to a M of 36–38 kJ·min^{-1}. In a sports-related context, elite marathon runners sustain VO_2 levels of ~3.5 L·min^{-1} and M of ~74 kJ·min^{-1} [5].

Combined Heat Loss

Heat transfer at the skin surface to the surrounding environment can be split into two primary subcomponents: dry heat transfer (H_{dry}) and evaporative heat loss (H_{evap}). As such, the simplest form of the human heat balance equation can now be reexpressed as:

$$H_{prod} = \left(H_{dry} + H_{evap}\right) \pm S \tag{2.2}$$

While H_{evap} almost exclusively arises from evaporation of sweat from the skin surface, H_{dry} occurs by a combination of three different pathways – conduction (K), convection (C), and radiation (R):

$$H_{prod} = \left(C \pm R \pm K\right) + H_{evap} \pm S \tag{2.3}$$

Dry Heat Transfer: K is the transfer of heat from direct contact with a solid surface and under most circumstances is considered negligible from a whole-body heat balance perspective [6]. C is the transfer of heat promoted by the movement of a fluid, usually air, and is proportional to (i) the difference in temperature between the air and the skin and (ii) the rate at which air passes across the skin [7]. Skin temperature is typically ~35°C in a fully vasodilated state; therefore when air temperature exceeds this value, convective heat loss becomes heat gain, contributing to an increase in S. R is the transfer of electromagnetic energy from a relatively warm body to a cooler one. In outdoor environments, the sun is usually the greatest source of radiant energy. R is proportional to the temperature difference between mean radiant temperature, derived using a black globe thermometer and air velocity, and mean skin temperature. R is also determined by the effective radiative area of the body and is altered by posture and the orientation of the person relative to the radiation source. R often serves as an environmental heat gain when skin temperature is lower than mean radiant temperature, which in the summertime can be more than 10°C greater than ambient air temperature (measured in the shade).

Evaporative Heat Loss: The rate of evaporation from the skin surface is determined by the absolute water vapor pressure difference between the skin (primarily dictated by eccrine sweating) and air. For the purposes of whole-body heat balance,

negative evaporative heat loss (i.e., condensation) is negligible. The evaporation of sweat is also promoted by an increased rate of air flow across the skin, which can arise from a combination of self-generated (from physical movement) and ambient air flow. Humans have a finite capacity for evaporative heat loss, with the maximum rate of evaporation (E_{max}) of a person determined by the fraction of body surface area that they can physiologically cover with sweat. This relative value is called "skin wettedness" (ω) [8] and ranges from a minimum value of 0.06 at rest with no thermoregulatory sweating to a maximum value with maximal sweating of ~0.75–0.85 for an unacclimated person and 1.00 for a fully heat acclimated person [6]. As ω rises toward these maximal values, the volume of sweat that evaporates relative to what is produced (i.e., sweating efficiency) decreases due to a limited environmental humidity gradient [8]. For every gram of sweat that does evaporate from the skin, 2.426 kJ of latent heat is liberated from the body [9]. The same sweat rate in a humid environment will therefore result in a lower evaporative heat loss because a greater proportion of sweat drips off the body before evaporating. For example, a whole-body sweat rate of 15 g·min^{-1} in an arid (dry) climate yielding a sweating efficiency of 90% will result in a H_{evap} of 32.8 kJ·min^{-1}, whereas the same sweat rate in a tropical (humid) environment with a sweating efficiency of 50% will result in a H_{evap} of 18.2 kJ·min^{-1}.

For all heat loss components (dry and evaporative), the absolute amount of heat transfer for a particular person is determined by body surface area. That is, a person with a larger surface area, which is predominantly determined by height and weight, will have a greater absolute heat loss. Clothing also heavily influences dry and evaporative heat losses. In a cold environment, ensembles with large amounts of insulation limit convective and radiative heat loss, whereas in an environment with a high radiant heat load, these properties protect the wearer from excessive environmental heat gain and potential burn injuries. Clothing with a high evaporative resistance (e.g., nuclear, biological, chemical suits or American football pads) greatly reduces E_{max} under a fixed set of environmental conditions by slowing the rate at which water vapor passes through the clothing and can substantially add physiological heat strain, particularly when such clothing is worn during physical activity.

Body Heat Storage

If at any time H_{prod} is not matched by an equal amount of H_{loss} (i.e., the sum of H_{dry} and H_{evap}), a change in body heat storage (S) will occur. An accumulation of heat energy inside the body (i.e., a positive S value), as is often observed during exercise/physical activity in a hot environment especially when clothing with a high evaporative resistance is worn, results in a rise in internal body temperature. For a given person, a greater heat storage leads to a greater rise in internal body temperature; however, between people of different body sizes, a smaller individual will get hotter for a given S value [10]. For example, for a person weighing 65 kg, an S value of +320 kJ would cause a 1.4 °C rise in mean body temperature, whereas the same S

value for 95 kg person would only lead to a rise in mean body temperature of 0.95°C. The average specific heat capacity of the body (C_p) is typically assumed to be 3.47 kJ·kg^{-1}·°C^{-1}; however, because the C_p of fat is lower (2.97 kJ·kg^{-1}·°C^{-1}) than the muscle (3.64 kJ·kg^{-1}·°C^{-1}), these elevations in mean body temperature for a fixed S should be altered by the amount of body fat. While large differences in body fat (~20%) do seem to result in slightly higher rises in core temperature (~0.2°C), adipose tissue does not seem to interfere with the capacity to dissipate heat [11]. Indeed, while fat does serve as an insulator during cold exposure, a pronounced peripheral vasodilation of the skin during exercise and heat exposure renders these insulating properties ineffectual, and sweating capacity does not seem to be altered by the presence of body fat. This primarily can be attributed to the superficial nature of skin blood vessels relative to adipose tissue. Of note, however, is that extremely large individuals with a body surface area of >2.5 m^2 may exhibit reductions in ω_{max} and, therefore, reduction in E_{max} secondary to a lower sweat gland density [12].

Compensable and Uncompensable Heat Stress

During exercise/physical activity, H_{loss} is augmented to balance elevations in H_{prod}, primarily through increases in eccrine sweating and thus H_{evap}. The amount of evaporative heat loss required (E_{req}) to attain an S of 0 (i.e., heat balance) is determined by:

$$E_{req} = H_{prod} - \left(C \pm R \pm K\right) \tag{2.4}$$

If E_{req} is possible, the imbalance between H_{prod} and H_{loss} is transient, and core temperature will rise but then reach an elevated plateau (compensable heat stress; Fig. 2.1a–b). However, if E_{req} is greater than E_{max}, either because H_{prod} is very high or the prevailing climate and/or clothing worn does not permit a sufficiently high rate of evaporation, (i) H_{loss} fails to match H_{prod}, (ii) S continually occurs, and (iii) internal body temperature rises uncontrollably (i.e., uncompensable heat stress; Fig. 2.1c–d). This latter condition can prove to be potentially dangerous as eventually critically high levels of core temperature can be reached and the risk of heat-related illnesses rapidly develops.

Heat Acclimatization

Humans possess a profound ability to adapt to hot environments, greater than any other environment they encounter. In some circumstances, situations that create uncompensable heat stress can become compensable through repeated exposure. These physiological changes, labeled heat acclimatization, greatly increase the

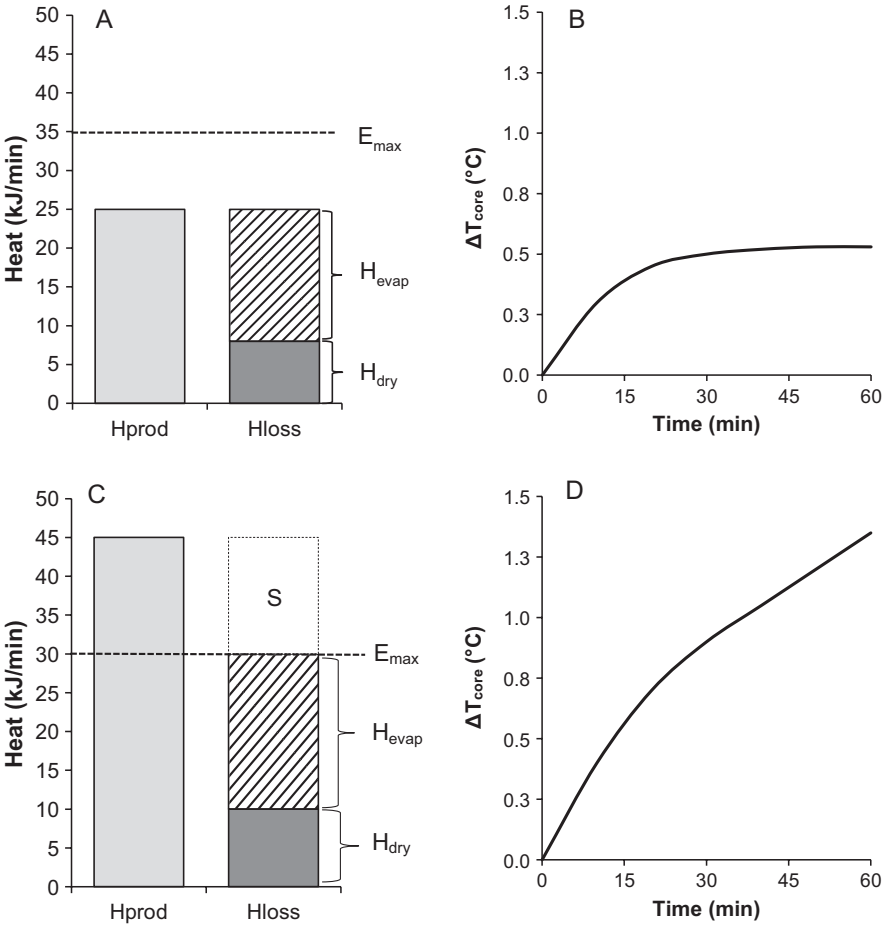

Fig. 2.1 (**a**–**d**) Examples of human heat balance status (left) and concomitant changes in core body temperature (right) during compensable (top) and uncompensable (bottom) heat stress scenarios

capacity for exercise in the heat [13]. The magnitude of heat acclimatization is so great that a lack of heat acclimatization is largely considered to be a risk factor for exertional heat illnesses.

The adaptations associated with heat acclimatization occur primarily in the cardiovascular and thermoregulatory systems through 10–14 days of repeated exercise heat stress [14]. It is also possible to induce these processes from simulated hot environments or clothing and equipment that inhibits H_{loss}; however, in these artificial situations, the changes are labeled heat acclimation [15].

Cardiovascular Changes

During exercise in the heat, the body must maintain adequate perfusion to the exercising muscles, the visceral organs, the brain, and the skin vasculature. Over the course prolonged exercise, where sweat fluid losses decrease the total plasma volume available, cardiovascular drift occurs as the body is not able to provide adequate perfusion to all of the above locations [16]. Cardiovascular drift occurs through a compensatory increase in heart rate to maintain cardiac output despite a decreasing stroke volume.

The cardiovascular adaptations that occur during heat acclimatization directly combat cardiovascular drift. Aldosterone and arginine vasopressin-induced fluid retention lead to an increase in total body water of 2–3 L (5–7%) [17]. Net increases in total intravascular protein facilitate fluid movement from the interstitial to the intravascular space, resulting in plasma volume expansion of 4–15% [17]. Therefore, to counteract the changes typically seen in cardiovascular drift, heart rate decreases via decreased myocardial autonomic tone and stroke volume increases to maintain a constant cardiac output at a given workload.

Thermoregulatory Changes

While the cardiovascular changes that occur through heat acclimatization allow for increased work output, they do not directly support an increased ability to maintain heat balance during exercise heat stress. Heat acclimatization decreases resting internal body temperature, theoretically creating a greater capacity for S during exercise [18]. In addition, skin blood flow during exercise increases, supporting greater H_{dry} [17].

Chiefly, the changes that occur in the thermoregulatory system's effector organs, eccrine sweat glands, support a further H_{prod} and diminished H_{loss}. In total, acclimatized individuals have been shown to have H_{evap} increase by 11% [19]. Sweat glands not only begin excreting at lower internal body temperatures in heat acclimatized individuals (decreased sweat onset), they will also excrete more (increased sweat rate) at a faster rate (increase sweat sensitivity) [17].

While these changes in sweat onset, rate, and sensitivity increase the capability for the H_{evap}, it also increases the fluid losses from sweat and increases the rate of dehydration. To a certain extent, the relationship between thirst and fluid needs also improves to diminish this effect [17]. To prevent concurrent large losses of sweat electrolytes due to greater sweat excretion, eccrine sweat glands increase electrolyte absorption along with increased electrolyte reabsorption in the kidney [18].

Benefits of Heat Acclimatization Beyond Heat Stress

The ability to perform a given amount of exercise with less physiological strain is a hallmark sign of the successful completion heat acclimatization process. Furthermore, the nature of the adaptations induced by heat acclimatization lead to changes that benefit exercise performance in both warm and cold environments [13]. Improvements in VO_2 and cycling power at a fixed heart rate are commonly observed following heat acclimatization [17].

Heat Acclimatization Protocols

Many strategies exist to induce heat acclimatization, ranging from short-term high intensity programs [20] to passive programs that utilize a sauna or hot water immersion post-exercise [21]. The most commonly studied protocols involve 90–120 min of treadmill walking in a warm environment for 10–14 days [18].What remains consistent is that the thermoregulatory and cardiovascular system must be stressed in order for adaptations to occur [22]. Furthermore, heat acclimatization is not permanent; some intermittent exposure to exercise heat stress is required to maintain heat acclimatization status [19]. From an athletic standpoint, American football has developed some of the most comprehensive policies for heat acclimatization, wherein exercise duration, intensity, and equipment worn are all gradually phased in over the first 2 weeks of practice [23]. Similar concepts can be utilized across the physical activity spectrum to reduce heat stress and improve performance.

Exertional Heat Illnesses

As a manifestation of dysfunctions in the thermoregulatory system, exertional heat illnesses constrain the ability to perform physical activity or labor. Exertional heat stroke may be the only fatal heat illness; however, even minor conditions can limit the ability to successfully compete, operate, or work in warm environments. While exertional heat illnesses cover a wide spectrum of severities, they do not exist in a continuum. It is a commonly propagated myth that a more severe heat illness is predicated on the previous presences of less severe one (Table 2.1).

Table 2.1 Exertional heat illness prevention strategies and physiological processes they affect

Strategy	Physiological process
Increase frequency and duration of rest breaks	Increase H_{loss} Decrease rate of H_{prod}
Gradually increase exercise intensity and equipment	Increase H_{evap} through heat acclimatization
Identify supplements and drugs that affect thermoregulation, and limit their use	Decrease H_{prod} from thermogenic substances
Encourage adequate hydration	Maintain adequate skin perfusion for H_{dry}, and maximize H_{evap}
Limit exercise with febrile illness	Increase capacity for S
Improve physical fitness	Decrease relative H_{prod}
Modify activities based on environmental conditions	Decrease environmental H_{gain}

Abbreviations: H_{dry} dry heat transfer, H_{evap} evaporative heat loss, H_{gain} combined heat gain, H_{loss} combined heat loss, H_{prod} metabolic heat production, S body heat storage

Minor Heat Illnesses

The minor heat illnesses, heat edema and miliaria rubra, are not directly limiting to exercise in the heat but rather are caused by heat exposure. Heat edema is the inflammation of the extremities caused by pooling of fluid in the interstitial space [24]. As it is a relatively benign one of the only treatments is to remove the heat exposure.

Miliaria rubra, typically called a "heat rash" or "prickly heat," is the presence of small erythematous papules caused by clogged sweat glands [24]. This condition also corrects itself to a certain extent when the heat exposure is removed, but proper hygiene can help limit the recurrence. Since the sweat glands become clogged in miliaria rubra, H_{evap} may be diminished leading to an increased risk of more severe exertional heat illnesses.

Heat Cramps or Exercise-Induced Muscle Cramps

The current understanding of the etiology of muscular cramps indicates that heat exposure is not a primary cause of cramps during exercise. Rather, it appears that most cramps occur as a result of neural fatigue, which may be exacerbated by either the increased physiological demands of exercise in the heat or sweat electrolyte losses [25]. This type of muscular cramp typically appears in a localized, visible fashion and can be very painful. Most instance of cramping respond well to stretching and rest. Individuals who repeatedly cramp during exercise in warm environments should monitor their hydration and sodium intake as cramping may be indicative of a fluid or electrolyte imbalance [26].

Heat Syncope

Postural hypotension from either prolonged standing or a sudden of cessation of exercise in warm environments defines heat syncope. The condition is often exacerbated by dehydration, which further contributes to poor venous return [24]. Syncope that is not a collapse associated with a more serious exertional heat illness is typically rapidly reversed by placing the patient in Trendelenburg's position. This supine position with the feet elevated 15–30 degrees above the head coupled with fluid replacement allows for a normalization of central blood pressure and facilitates a rapid recovery [27].

Heat Exhaustion

Heat exhaustion is primarily a diagnosis by exclusion. Broadly defined as the inability to continue exercise in the heat, the fundamental cause of collapse from heat exhaustion is cardiovascular insufficiency [28]. The individual with heat exhaustion will exhibit an elevated body temperature, but will not have persistent central nervous system dysfunction. Care for heat exhaustion includes removing the patient from exercise and any heat. It may also be beneficial to provide some cooling [27]. In cases where patients do not rapidly improve, healthcare providers should evaluate for exertional heat stroke.

Exertional Heat Stroke

Exertional heat stroke is the most severe of the exertional heat illnesses and is a medical emergency. Contrasted to classical heat stroke, which normally occurs in individuals with compromised thermoregulatory systems during heat waves, exertional heat stroke is caused by exercise-induced hyperthermia. Most cases occur in a warm environment; however, cases have been reported in cooler conditions where sufficient exercise stress dangerously elevates internal body temperature.

The current understanding of exertional heat stroke pathophysiology is that hyperthermia induces leakage of endotoxin from the gastrointestinal tract into the systemic circulation, causing acute kidney and liver failure, rhabdomyolysis, and disseminated intravascular coagulation [29, 30]. Heat-shock proteins, chaperone molecules within the body, demonstrate a limited ability to minimize the damage for short durations of extreme hyperthermia [31].

The risk factors for exertional heat stroke can be dichotomized as either intrinsic, internal to the individual, or extrinsic, a factor imposed on the individual. Intrinsic risk factors can further be delineated as temporary or permanent. Poor fitness, febrile illness, and sleep deprivation are all risk factors that may preclude individuals from safely exercising in the heat until they are corrected [27, 32]. Meanwhile indi-

viduals with certain genetic factors (e.g., malignant hyperthermia), taking drugs or supplements that affect thermoregulation, or who are overweight should be closely monitored during periods of intense exercise [33, 34].

Extrinsic risk factors include both organizational and environmental components. From an organization standpoint, the risk of exertional heat stroke can be minimized by changing activities to parts of the day, providing more frequent and longer rest breaks and enacting heat acclimatization protocols [23, 27]. In most cases, the environmental risk factors for exertional heat stroke cannot be modified but rather monitored. Conditions with high ambient temperatures, high humidity, and direct sunlight exposure increase the likelihood of heat gain from the environment. Abnormally extreme conditions may require activity modification to allow for participants to safely complete their tasks [35].

The two pathognomonic criteria of exertional heat stroke are an internal body temperature greater than 40.5 °C and end organ dysfunction, typically manifesting as neuropsychiatric dysfunction [27]. A wide variety of conditions from concussion to hypoglycemia also display neuropsychiatric dysfunction, making an accurate assessment of body temperature crucial for exertional heat stroke diagnosis. Furthermore, some exertional heat stroke patients have a lucid interval where central nervous system dysfunction is not immediately obvious. The only field expedient measure of body temperature that has been validated for exercise-induced hyperthermia and exertional heat stroke diagnosis is a rectal temperature [36–39]. Other temperature modalities may falsely indicate normothermia, delaying appropriate treatment.

Survival from exertional heat stroke requires that the extreme state of hyperthermia is reversed before irreversible organ damage occurs. Inappropriate, ineffective, or absent treatment is deadly for the exertional heat stroke patient [40]. As stated above, the body has a limited ability to tolerate extreme hyperthermia; optimal prognoses from exertional heat stroke occur when the body temperature is reduced shortly after collapse [41]. The most effective treatment for exertional heat stroke has been found to be cold-water immersion [42]. When cold-water immersion is initiated shortly after the patient collapses, survival is likely [43]. In more remote situations, tarp-assisted cooling may be used as an adjunct for cold-water immersion [44, 45]. Ice packs in the axillary and groin or misting water with fans are not effective cooling modalities for an exertional heat stroke patient [46].

Conclusion

In summary, human physiology dictates the limits of safety and performance during exercise in the heat. Situations where excessive heat gain or limited heat loss create uncompensable heat stress can lead to performance decrements or exertional heat illnesses. Overall, the strategies to mitigate heat stress for athletes, laborers, and war fighters should focus on these physiological limitations while utilizing the body's own adaptations to maximize safety and optimize performance in the heat (Table 2.1).

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